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CARCINOGENESIS ABSTRACTS

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CARCINOGENESIS ABSTRACTS

VOLUME 16, ISSUE 5

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CARCINOGENESIS ABSTRACTS makes available abstracts, annotations or citations of significant carcinogenesis articles collected from the current major biomedical sources of world literature. This service is provided by the National Cancer Institute through a contract with the Franklin Research Center for preparation of the publication, under Contract No. NOI-CP-75885 with the National Cancer Institute, U.S. Department of Health, Education and Welfare. Published and distributed by the Franklin Institute Press^{SM}.

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ABBREVIATIONS

URNAL names are abbreviated according to the *List of Journals Indexed in Index Medicus, Abbreviation sting*. If the journal is not listed in this, abbreviations are derived from the *International List of Periodical Title ord Abbreviations.*

NGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. e source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

BBREVIATIONS used in abstracts:

	angstrom(s)	mOsm	milliosmolar
CTH	adrenocorticotropic hormone	max	maximum
DP	adenosine diphosphate	mEq	milliequivalent(s)
MP	adenosine monophosphate	min	minute(s)
TP	adenosine triphosphate	ml	milliliter(s)
prox	approximately	µl	microliter(s)
	average	mm	millimeter(s)
CG	bacillus Calmette-Guerin	mo	month(s)
d	twice daily	mol wt	molecular weight
	degree(s) centigrade	N	normal concentration
l	calorie(s)	NAD	nicotinamide adenine dinucleotide
cal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
	cubic centimeter(s)		
	curie(s)	NADP	nicotinamide adenine dinucleotidephosphate
nCi	millicurie(s)		
Ci	microcurie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
	centimeter(s)		
NS	central nervous system	NCI	National Cancer Institute
m	counts per minute	NIH	National Institutes of Health
NA	deoxyribonucleic acid	PAS	periodic acid-Schiff
$_{50}$	median effective dose	po	orally
TA	ethylenediamine tetraacetic acid	ppb	parts per billion
	gram(s)	ppm	parts per million
g	kilogram(s)	qid	four times daily
ng	milligram(s)	qod	every other day
g	microgram(s)	QO_2	oxygen quotient
	hemoglobin	R	roentgen
	hour(s)	RBC	red blood cells (erythrocytes)
	intra-arterial	RNA	ribonucleic acid
	intradermal	rpm	revolutions per minute
A	Immunoglobulin A	sc	subcutaneous
B	Immunoglobulin B	sec	second(s)
G	Immunoglobulin G	SGOT	serum glutamic-oxaloacetic transaminase
M	Immunoglobulin M	SGPT	serum glutamic-pyruvic transaminase
S	increased life span	soln	solution
	intramuscular	TCD	tissue culture dose
	intraperitoneal	TCD_{50}	median tissue culture dose
	International Unit(s)	tid	three times daily
	intravenous	UV	ultraviolet
n	Michaelis constant	WBC	white blood cells (leukocytes)
	lethal dose	wk	week(s)
$_{50}$	median lethal dose	wt	weight
	molar	X	times
M	micromolar	yr	year(s)

8-2401 **Carcinogenic Potential of Cancer Chemotherapeutic Agents in Man.** (Eng) Adamson, R. I. (Lab. Chemical Pharmacology, Div. Cancer Treatment, NCI, Bethesda, MD, 20014); Sieber, S. M. *Cancer Bull* 29(6): 79-183; 1977.

Case studies have indicated that many alkylating agents, anitumor antibiotics, and antitumor antimetabolites used in the reatment of both malignant and nonmalignant conditions an be carcinogenic. It is suggested that a Second Tumor Registry be established to assess the carcinogenic potential of these agents. This registry would contain information on hemotherapy and other treatment, the patient, and the size of the population receiving similar treatment. (7 refs)

'8-2402 **Dangers of Oversimplification in the Tier Approach (Meeting Abstract).** (Eng) Russell, W. L. Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830). *Autat Res* 53(1): 104; 1978. (no refs)

'8-2403 **In Vivo DNA Damage/Alkaline Elution Assay for Predicting Carcinogenic Potential (Meeting Abstract).** (Eng) Petzold, G. (Upjohn Co., Kalamazoo, MI, 9001); Harbach, P.; Bedell, M.; Swenberg, J. A. *Mutat Res* 3(1): 110-111; 1978. (no refs)

'8-2404 **Evidence Against the Reversion of Mutation in the *Haemophilus Influenzae* Phage HP1c1 by Preinfection Treatment of Host Cells or Phage with MNNG.** Eng) Boling, M. E. (Biology Div., Oak Ridge Lab., Oak Ridge, TN, 37830); Kimball, R. F. *Mutat Res* 50(1): 37-42; 978.

The mutagenic effects of N-methyl-N′-nitro-N-nitrosoguaniline (MNNG) on the temperature-sensitive *Haemophilus influenzae* phage HP1c1 were studied. Phage DNA and uninfected cells were treated with MNNG (50 or 100 μg/ml) at) C and then combined. Cells treated with MNNG after ransfection served as a positive control. No mutations were nduced when the bacteria and/or phage DNA were treated with MNNG before transfection. In contrast, when phage DNA was treated inside the host cell, a high frequency of reversion was observed. Thus, there is no evidence that treatment of bacteria with MNNG induces alterations in the bacteria that can produce mutations in either untreated DNA or DNA treated separately before transfection. It is likely that the initial lesions in the DNA are eliminated during transformation either by repair or by a failure to be incorporated into the host genome. (17 refs)

78-2405 **Approaches to Determining the Mutagenic Properties of Chemicals: Risk to Future Generations.** (Eng) DHEW Working Group Subcommittee Environmental Mutagenesis (No affiliation given). *Environ Pathol Toxicol* 1(2): 301-352; 1977.

There are three objectives for mutagenicity testing of chemicals: (1) to determine whether a chemical has the potential to cause heritable genetic changes in humans; (2) to obtain data from animal model systems for which safe exposure levels for humans may be extrapolated; and (3) to provide information on other toxicological manifestations, such as carcinogenicity. Factors to be considered in designing a test strategy include the use and exposure category of the test substances, stage of development of a product, existing and anticipated levels of human exposure, legislative aspects, regulatory status, and available resources for testing. Procedures necessary to define a chemical as a human mutagen include tests that detect chromosome and point mutations. In evaluating chromosomal effects, either the heritable translocation test or the sex chromosome loss test in mice can provide sufficient evidence. For evaluating point mutations, the only mammalian test available that can provide sufficient evidence in itself is the specific locus test in mice. As an alternative, positive results in 2/3 following categories should be considered evidence of mutagenicity: point mutations in eukaryotic microorganisms or in mammalian cells in cultures and recessive lethal mutations in insects. The current mutagenicity testing experience of various classes of chemicals is illustrated in an extensive table. (90 refs)

78-2406 **Does Carcinogenic Potency Correlate with Mutagenic Potency in the Ames Assay?** (Eng) Ames, B. N. (Dept. Biochemistry, Univ. California, Berkeley, CA, 94720); Hooper, K. *Nature* 274(5666): 19-20; 1978.

Reply is made to criticism of current research in which the relation between the potency of a chemical in an animal cancer test and in the *Salmonella typhimurium* mutagenicity assay was explored. Given the millionfold range in carcinogenic and mutagenic potencies, correspondence between the two can be useful without needing to be very precise. Several literature reports indicate that there is a good correlation for most chemicals examined so far. In addition, other short-term tests seem to show responses similar to that of *Salmonella*. It is true that varying the standard test parameters could change the potency somewhat and that there is a problem of false negatives/positives, but some carcinogens will never be detected in the *Salmonella* test because they cause cancer by nonmutagenic mechanisms or for other reasons. However, many short-term tests, including the *Salmonella* assay, have successfully predicted carcinogenicity for several environmental chemicals with widespread human exposure (ie, tris, 1,2-dichloroethane, hair dye chemicals). It is expected that a battery of short-term tests will soon be useful in estimating carcinogenic potency as well. (14 refs)

78-2407 **Use of Purified Lesion-Recognizing Enzymes to Monitor DNA Repair in Vivo.** (Eng) Paterson, M. C. (Biology and Health Physics Div., Chalk River Nuclear Lab., Atomic Energy of Canada Limited, Chalk River, Ontario, Canada). *Adv Radiat Biol* 7: 1-53; 1978.

This review deals with a recent methodological advance, exploitation of the unique properties of purified lesion-recognizing enzymes as the basis of assays that monitor, with a high degree of sensitivity and specificity, the repair of DNA lesions in vivo. The enzymes include UV endonucleases, apurinic site endonucleases and N-glycosidases, and γ-endonucleases. The assays are described in detail, their assets and liabilities are weighed, and new findings emerging from their use are presented. (178 refs)

78-2408 **Environmental Carcinogenesis (Meeting Abstract).** (Eng) Falk, H. L. (Natl. Inst. Environmental Sciences, Research Triangle Park, NC). *Clin Chem* 24(6): 971-972; 1978. (no refs)

78-2409 **Experimental Identification of Chemical Carcinogens, Risk Evaluation, and Animal-to-Human Correlations.** (Eng) Saffiotti, U. (Experimental Pathology Branch, NCI, NIH, Bethesda, MD, 20014). *Environ Health Perspect* 22: 107-113; 1978.

Experimental methods for the identification of chemical carcinogens include animal bioassay methods, animal models for cancer induction at major organ sites, models for the study of the effects of carcinogens in cells and tissues in culture, and methods for the study of molecular events. The following steps are necessary for the evaluation of risks, benefits, and technological alternatives for public policy on environmental carcinogens: (1) risk evaluation, which should consider sources, adequacy, quality and limits of the evidence, quantitative dose-response extrapolation within the same biological system, and species and model conversion factors; (2) benefit evaluation; (3) analysis of technological alternatives to identify the estimated least hazardous technological alternative and to determine its feasibility; (4) comparative judgement and decision; and (5) open public documentation. The problem of animal-to-human correlations is centered on bridging the gap between whole-animal experiments and human pathology. A laboratory approach has been developed, with particular emphasis on respiratory tract carcinogenesis studies, which includes development and study of whole animal models for carcinogenesis, analysis of animal tissue responses to carcinogens in vivo and in vitro and development of in vitro culture methods to study human target tissues and correlation of the results with human pathology in vivo. (64 refs)

78-2410 **Mechanisms of Chemical Carcinogenesis.** (Eng) Weisburger, E. K. (Carcinogen Metabolism and Toxicology Branch, NCI, Bethesda, MD, 20014). *Annu Rev Pharmacol Toxicol* 18: 395-415; 1978.

The mechanisms of action of chemical carcinogens, their metabolic activation, and the influence of other xenobiotics on carcinogens are reviewed. Most chemical carcinogens are initially converted through mixed function oxidases to activated forms. Further reaction leads to entities, often transient, that are electrophiles capable of reacting with cellular nucleophiles. Other environmental factors may enhance or inhibit the action of carcinogens. (211 refs)

78-2411 **Metals as Mutagens.** (Eng) Flessel, C. P. (Biochemistry Group, Air and Industrial Hygiene Lab., State California, Dept. Health, 2151 Berkeley Way, Berkeley, CA). *Adv Exp Med Biol* 91: 117-128; 1977.

Evidence that compounds of arsenic, chromium, copper, iron, manganese, molybdenum, platinum, and selenium are mutagenic in microorganisms is presented. In addition, compounds of aluminum, antimony, arsenic, cadmium, copper, lead, mercury, nickel, and tellurium induce chromosome damage in higher organisms. Of these metals, arsenic, cadmium, chromium, iron, lead, manganese, and nickel are reportedly carcinogenic in animals or humans. In contrast, selenium and platinum have been described as cancer-protecting or carcinostatic agents, respectively. (44 refs.)

78-2412 **Occupational Exposure to Inorganic Arsenic.** (Eng) Brinkerhoff, G. (Office Compliance Programs, Occupational Safety and Health Administration, Room N-3112, Third St. and Constitution Ave., N. W., Washington, DC, 20210). *Fed Regist* 43(88): 19584-19631; 1978.

The Occupational Safety and Health Administration's ruling on inorganic arsenic exposure in the workplace is presented, along with the evidence on which the ruling is based. Occupational exposure to inorganic As is limited to 10 $\mu g/m^3$ based on an 8-hr time-weighted av. The evidence indicates that inorganic As exposure causes lung cancer, with the risk increasing with exposure. Pentavalent As should be regulated as a human carcinogen. (29 refs)

78-2413 **Talc in Drugs (Letter to Editor).** (Eng) Nicar, M. J. (Health Science Center at Dallas, Southwestern Medical Sch., Univ. Texas, Dallas, TX, 75235); Norman, D. A. *N Engl J Med* 299(10): 556-557; 1978.

Although several pharmaceutical drugs contain talc, which is chemically similar to asbestos, no experimental data have shown that ingestion of talc or asbestos causes cancer. The carcinogenicity of asbestos seems limited to inhaled asbestos. Therefore, there should be more immediate concern for pharmaceutical employees who might inhale talc rather than for patients who take these drugs. (6 refs.)

78-2414 **The Biological Effects of Talc in the Experimental Animal: A Literature Review.** (Eng) Lord, G. H. (Johnson and Johnson Res. Foundation, New Brunswick, NJ). *Food Cosmet Toxicol* 16(1): 51-57; 1978.

Literature on the exposure of experimental animals to talc is reviewed. Although the talcs used have not been characterized, it has been shown that the mineral, in different degrees of purity, is fibrogenic and that the fibrotic response is a function of dose. However, certain levels of exposure are tolerable. No neoplastic response has been observed. (41 refs)

78-2415 **Toxicology of Vinyl Chloride.** (Fre) Heuse, A. (Laboratoire de Medecine du Travail et Hygiene du Milieu, Universite Libre de Bruxelles, Brussels, Belgium). *Brux Med* 58(1): 13-34; 1978.

A review of the literature on vinyl chloride (VC) traces the history of the toxicity of the compound. Also reviewed are the toxic effects of polyvinyl chloride (PVC), relative risk of industrial exposure to VC and PVC, and potential danger of exposure of the general population to PVC. (196 refs)

78-2416 **Dechlorination Mechanisms of Chlorinated Olefins.** (Eng) Van Dyke, R. A. (Mayo Clinic, Rochester, MN, 55901). *Environ Health Perspect* 21: 121-124; 1977.

The dechlorination of chlorinated hydrocarbons were investigated in detail. The reaction occurs optimally in the presence of O_2 and it is catalyzed by Cytochrome P-450. The oxidative dechlorination of hydrocarbons begins by an attack on the C atom and the halogen is then released from the oxidized C. Chlorinated olefins are not easily dechlorinated enzymatically. Since the chlorines migrate readily across the double bond, cyclic chloronium ions must occur as intermediates. It is not known if an epoxide is a necessary intermediate for chloronium ion formation. (22 refs)

78-2417 **Oncogenic Transformation of Rodent Cell Lines by Chemical Carcinogens.** (Eng) Heidelberger, C. (Los Angeles County-Univ. Southern California Cancer Center, Los Angeles, CA, 90031). *In: Human Risk Assessment. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H., Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. 4(Book C), 583 pp.; 1513-1520; 1977.

Experimental studies of the chemical transformation of mouse prostate fibroblasts and mouse embryo C3H/10T1/2 cells are reviewed. If these lines can be transformed by representative compounds from various classes of chemical carcinogens, they will be of value as prescreens for environmental carcinogens. (78 refs.)

78-2418 **Evaluation of the Health Effects of Benzene Inhalation.** (Eng) Haley, T. J. (Dept. Health, Education and Welfare, Food and Drug Admin., Natl. Center Toxicological Res., Jefferson, AR, 72079). *Clin Toxicol* 11(5): 531-548; 1977.

Animal and human data on the acute and chronic toxicity, absorption, distribution, metabolism, and excretion of benzene are reviewed. Benzene exposure produces hypoplasia of the bone marrow, which leads to aplastic anemia; chronic exposure, either occupationally or domestically, can cause leukemia. (100 refs)

78-2419 **The Metabolism of Polycyclic Hydrocarbons and Its Relationship to Cancer.** (Eng) DePierre, J. W. (Dept. Biochemistry, Arrhenius Lab., Univ. Stockholm, Fack, S-106 91, Stockholm, Sweden); Ernster, L. *Biochim Biophys Acta* 473(3/4): 149-186; 1978.

The metabolism of polycyclic hydrocarbons in rat liver and lung and in various human tissues is reviewed in relation to the carcinogenic properties of these compounds. The major pathways for the metabolism of polycyclic hydrocarbons in mammalian tissues involve the aryl hydrocarbon monooxygenase system, epoxide hydrase, and glutathione S-epoxide transferase. There is much evidence to support the hypothesis that epoxide intermediates formed by aryl hydrocarbon monooxygenase activity react nonenzymatically with DNA and are the immediate carcinogens. Although the exact mechanism of polycyclic hydrocarbon carcinogenesis is not known, correlations between aryl hydrocarbon monooxygenase and cancer have been found. The inducibility of aryl hydrocarbon monooxygenase in human peripheral lymphocytes was shown in one study to be controlled genetically by two alleles, A and B; cells containing AA had low inducibility, AB medium inducibility, and BB high inducibility. A study of 50 smokers with bronchial cancer revealed that 4%

had AA, 66% AB, and 30% BB compared with normal US incidences of 45% for AA, 46% for AB, and 9.4% for BB. It is also becoming clearer that smoking induces aryl hydrocarbon monooxygenase activity in lung cells. In one study, smokers (10-40 cigarettes/day for at least 2 yr) were found to have aryl hydrocarbon monooxygenase levels in their alveolar macrophages that were almost 12 times as high as those of nonsmokers. (426 refs.)

78-2420 **Problems in the Assessment of Human Risk of Carcinogenesis from Chemicals.** (Eng) Weinhouse, S. (Fels Res. Inst., Temple Univ. Sch. Medicine, Philadelphia, PA, 19140). *In: Human Risk Assessment. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4(Book C), 583 pp.; 1307-1309; 1977.

Problems involved in assessing the human risk of carcinogenesis from chemical carcinogens include the extrapolation of animal incidences at high doses to incidences at low doses, extrapolation of animal data (frequently in animals with specific genetic makeup) to humans (diversified genetic makeup), determination of a threshold dose and a latent period, and the significance of negative results of carcinogenesis studies. (4 refs.)

78-2421 **The Value of Long-term Testing for the Implementation of Primary Prevention.** (Eng) Tomatis, L. (Unit Chemical Carcinogenesis, International Agency Res. Cancer, Lyon Cedex 2, France). *In: Human Risk Assessment. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4 (Book C), 583 pp.; 1339-1357; 1977.

The role of experimental animal data in predicting the risk of chemical carcinogenesis in humans is reviewed. Three conditions are necessary for the implementation of primary cancer prevention: (1) cancer is caused by exogenous factors; (2) the factors can be identified; and (3) they can be eliminated once identified. Several examples in which carcinogens were identified in animals before they caused tumors in humans are presented; had the results of the animal studies been taken seriously, cancer prevention could have been implemented sooner in man. The results of long-term animal experiments have made possible the International Agency for Research on Cancer's program for evaluating the carcinogenic risk of chemicals to man. Of the 296 chemicals evaluated so far, 21 are probable human carcinogens. The value of sc testing of a chemical for carcinogenesis in mice has been questioned, but evidence suggests that this is both an appropriate index of carcinogenicity and method of administration. Legislation relating to the prevention of occupational cancer in 14 industrialized countries is reviewed briefly. (47 refs.)

78-2422 **Neoplastic Skin Lesions in Salamanders from a Sewage Lagoon Containing Perylene.** (Eng) Windsor, J. G. (Dept. Chemical Engineering, Massachusetts Inst. Technology, Cambridge, MA, 02139); Laflamme, R. E.; Hites, R. A.; Rose, F. L. *Science* 198(4323): 1280-1281; 1977.

Several sources are proposed for the presence of 300 ppm perylene in a Texas sewage lagoon that may have caused a high incidence of neoplastic skin lesions in tiger salamanders. The most probable one is the diagenesis of terrestrial pigments, but others include a fuel spill from a nearby air force base, dumping of asphalt, use of diesel fuel as a mosquitocide, and jet exhaust. (8 refs)

78-2423 **Mutagenic Studies with Acrylonitrile (Three Letters to Editor).** (Eng) Venitt, S. (Inst. Cancer Res., Royal Cancer Hosp., Chester Beatty Res. Inst., Pollards Wood Res. Station, Chalfont St. Giles, Buckinghamshire, England); Milvy, P. *Mutat Res* 57(1): 107-113; 1978.

A recent article on the mutagenicity of acrylonitrile in several strains of *Salmonella typhimurium* reported positive results, but recalculation of the data indicated that the results were positive but tenuous. In a rebuttal, flaws in the recalculation are pointed out, and it is indicated that the original findings were not disproved. In a reply to the rebuttal, it is stated that a test chemical can be considered mutagenic only if it induces a reproducible, dose-related, and statistically significant increase in mutation frequency over control values. (4 refs)

78-2424 **Endometrial Cancer in Turner's Syndrome Following Long-Term Estrogen Treatment.** (Dan) Nielsen, J. (Cytogenetisk laboratorium, Psykiatrisk hospital i Arhus, Arhus, Denmark); Sorensen, K. *Ugeskr. Laeg.* 140(4): 171-172; 1978.

Nine cases of endometrial carcinoma associated with Turner's syndrome were found in the literature. All the patients had been treated with stilbestrol or diethylstilbestrol for 4-20 yr. However, there is no clear relationship between estrogen treatment and endometrial carcinoma in women with Turner's syndrome. (21 refs.)

78-2425 **Exogenous Estrogen and Endometrial Cancer: A Statistical Problem.** (Eng) Barron, B. A. (Dept. Obstetrics and Gynecology, Columbia Univ., Coll. Physicians and Surgeons, New York, NY). *Contemp Obstet Gynecol* 11(21): 135-142; 1978.

The results of five studies in which estrogen use was found to be associated with endometrial cancer are reviewed. The risk of this cancer in users of estrogen preparations is 3.0-7.9 times greater than the risk among nonusers. The increased incidence appears to be associated with both the duration of use and dosage of the estrogen preparation. In another study, however, it was concluded that the apparent association between endometrial cancer and estrogens was the result of the differential rates of screening between users and nonusers. Three studies had concordant results: for at least a subset of women, the use of exogenous estrogens in the peri- and postmenopausal period may increase their risk of developing endometrial cancer. However, the lesions found in users may be different from those encountered in nonusers. The risk factors and natural history of endometrial cancer are also reviewed. (7 refs)

78-2426 **Estrogen Therapy and Endometrial Carcinoma (Letter to Editor).** (Eng) Cohen, M. H. (Veterans Admin. Hosp., 50 Irving St., NW, Washington, DC, 20422). *Arch Intern Med* 138(4): 526-527; 1978.

Studies indicate that women taking estrogen preparations are at a greater risk than nonusers for the development of endometrial cancer. They also indicate that estrogen therapy for breast cancer may result in endometrial cancer. It is suggested that estrogens are overused and that as low a dose as possible should be given for as short a time as necessary. Regular follow-up of the patient's gynecologic status should be made. (refs)

78-2427 **Do Oral Contraceptives Cause Hepatic Lesions? Appraising the Evidence.** (Eng) Reagan, J. W. (Dept. Pathology, Case Western Reserve Univ., Cleveland, OH). *Contemp Obstet Gynecol* 11(4): 107-112; 1978.

A review of the literature on the association between oral contraceptive use and the development of benign hepatic lesions suggests a relationship, but the actual risk involved has not been established. The risk appears to be related to the duration of therapy and, possibly, to the type of steroid used. The pathology of the different reported lesions is described. Hepatoblastomas and hepatocellular carcinomas are observed in patients exposed to oral contraceptives, but the number of cases is too small to allow any definite conclusions. (18 refs)

78-2428 **The Effects of Estrogens and Oral Contraceptives on Hepatic Function.** (Fre) Leclere, J. (Service d'Endocrinologie et Medecine E, C.H.U. de Nancy, Hopital de Brabois, F 54500 Vandoeuvre-les-Nancy, France). *Ann Endocrinol (Paris)* 38(6): 443-446; 1977.

Since 1973, about 150 reports of hepatic tumors, 24 of which were malignant, have been published in the literature. Usually, the patient has been a young woman using oral contraceptives. The well-established effect of estrogens on hepatic function is reviewed, and the possible carcinogenic role of the contraceptives in the rising incidence of hepatic tumors is discussed. (8 refs)

78-2429 **Hepatic Alterations due to Oral Contraceptives.** (Ger) Roschlau, G. (Pathologisches Institut des Bereichs Medizin, Humboldt-Universitat zu Berlin, Schumannstrasse 20/21, DDR-104 Berlin, E. Germany). *Dtsch Gesundheitsw* 32(48): 2271-2274; 1977.

Hepatic changes observed after the long-term use of oral contraceptives include multiple hyperplasia and malignant hepatocellular carcinoma. Steroids may have an indirect carcinogenic effect due to the inhibition of glutathione-S-transferase. (19 refs)

78-2430 **Transplacental Toxicity of Diethylstilbestrol: A Special Problem in Safety Evaluation.** (Eng) McLachlan, J. A. (Environmental Toxicology Branch, Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC); Dixon, R. L. *In: New Concepts in Safety Evaluation.* Mehlman, M. A.; Shapiro, R. E.; Blumenthal, H., eds. (New York: John Wiley & Sons): Advances in Modern Toxicology, Vol. 1, Part 1, 455 pp. 423-448; 1976.

The transplacental carcinogenicity of diethylstilbestrol (DES; 4,4′-dihydroxy-α,β-diethylstilbene) is reviewed. As early as 1959, an association between neonatal exposure to DES and genital tract abnormalities in female offspring was suggested. Twelve years later, the association of prenatal DES exposure and vaginal clear cell adenocarcinoma in daughters of women who took the preparations was reported. Squamous cell dysplasia and carcinoma in situ of the cervix and vagina have also resulted in prenatally exposed patients. The risk of genital tract tumor development in daughters of treated women is unknown. Although subfertility has been detected in laboratory animals exposed to DES, no adequate studies of humans have been performed. Tumor development in male offspring has not been studied adequately, but at least one report has linked prenatal DES exposure with an increase in prostatic, testicular, and bladder cancer in 10 to 19-yr-old male offspring. Another study found epididymal cysts and prostatic inflammation in utero-exposed men. Early gestational DES exposure appears to be necessary for later development of abnormalities. The results of experiments with DES in laboratory animals are also reviewed. (123 refs.)

78-2431 **Diet and Nutrition.** (Eng) Newberne, P. M. (Dept. Nutritional Pathology, Massachusetts

Inst. Technology, E18-611, Cambridge, MA, 02139). *Bull NY Acad Med* 54(4): 385-396; 1978.

A review of the association between various dietary and nutritional factors and cancer incidence is presented. Particular attention is focused on the potential role of diet in gastric, colonic, and mammary cancer. (44 refs)

78-2432 **Fiber, Intestinal Sterols, and Colon Cancer.** (Eng) Huang, C. T. (Section Nutrition and Gastroenterology, Dept. Pediatrics, Baylor Coll. Medicine, Houston, TX, 77030); Gopalakrishna, G. S.; Nichols, B. L. *Am J Clin Nutr* 31(3): 516-526; 1978.

The effect of dietary fiber on intestinal function and composition of lipids in the feces and its relationship to colon cancer are considered. Dietary fiber is composed of four major components, cellulose, hemicellulose, lignin, and pectin. Hemicellulose and pectin may increase fecal bile acid excretion in humans, a change that may be accompanied by a decrease in serum cholesterol. Fibers such as rolled oats, alfalfa, guar gum, and Bengal gram have hypocholesterolemic properties. Alfalfa, wheat straw, and some other fibers bound considerable amounts of bile acids in vitro, but wheat bran, oat hulls, and all the synthetic fibers tested bound only negligible amounts. Vegetarians in the US have lower plasma lipids and different plasma lipoprotein patterns than comparable populations on a mixed diet. They also have smaller daily fractional turnover rates of cholic acid and deoxycholic acid pool size. The current dietary fiber intake in the US is approx 20% of what it was a century ago. Diets depleted of fiber may prolong the contact between stool content and mucosa and they may favor the growth of bacterial flora capable of forming carcinogens from the degradation of bile acids. It is suggested that fiber may reduce colon cancer incidence by decreasing intestinal transit time; altering bile acid metabolism and thereby reducing potential carcinogen formation; diluting potential carcinogens as a result of its bulking effect and ability to bind water, sterols, bile acids, and fat; and, through its effect on intestinal flora, by decreasing the bacterial degradation of bile acids and neutral sterols. (90 refs)

78-2433 **Chemical Carcinogens in Plant Food Products (Literature Review).** (Rus) Neiman, I. M. (Inst. Nutrition, Moscow, USSR). *Vopr Pitan* (1): 55-60; 1978.

The carcinogenicity of plant food products is reviewed briefly. The chemical carcinogens contained in these food products can be divided into (1) exogenous carcinogens resulting from contamination due to environmental pollutants, pesticides, or the products of heat treatment and fumigation and (2) endogenous carcinogens synthesized in the plants in vivo. (10 refs)

78-2434 **Mechanism of Action of Mycotoxins.** (Fre) Moule, Y. (Institut de Recherches Scientifiques sur le Cancer, 94800 Villejuif, France). *Ann Nutr Aliment* 31(4/5/6): 803-810; 1977.

Hypotheses concerning the mechanism of the physiological effects induced by mycotoxins, particularly the aflatoxins and the toxin from *Penicillium roqueforti* (PR toxin), are reviewed. Although the long-term effects of the PR toxin are unknown, a lot of data reviewed have accumulated on the hepatocarcinogenic activity of aflatoxin B_1. It is hypothesized that both the carcinogenic and mutagenic effect of aflatoxin B_1 are the result of the mycotoxin's interference with DNA repair. Treatment of cultures of *Escherichia coli* with active metabolites of aflatoxin B_1 derepresses the system of inaccurate DNA repair (SOS system). In bacteria, the SOS system induces the synthesis of repair enzymes that leads to mutagenesis. The carcinogenic effect of aflatoxin could be explained by the tumor produced by successive generations of cells arising from the single cell with a specific mutation. (34 refs)

78-2435 **Mycotoxin Tolerances in Foodstuffs.** (Eng) Krogh, P. (Univ. Inst. Pathological Anatomy, DK-2100 Copenhagen O, Denmark). *Pure Appl Chem* 49(11): 1719-1721; 1977.

Although many mycotoxins produce toxic effects in humans and animals, the only one mentioned specifically in legislation is aflatoxin. Standards for various countries, which are reviewed briefly, cover all foods, those likely to contain the toxin, or trade commodities only. Tolerances for human food range from 0 to 50 ppb; for feeds, the levels are higher. (7 refs.)

78-2436 **Short-Term Tests and Mycotoxins.** (Eng) Malaveille, C. (International Agency Res. Cancer, 150, Cours Albert-Thomas, 69008 Lyon, France); Bartsch, H. *Ann Nutr Aliment* 31(4/5/6): 871-880; 1977.

The value of short-term tests in assessing the possible carcinogenic risk of chemicals and in investigating the mode of biological action of chemicals in mammals and in humans is reviewed, with special attention being focused on results obtained with mycotoxins. Studies have suggested that there is an empirical relationship between carcinogenesis and mutagenesis; this led to the development of many short-term tests for mutagenicity. Bioassays in which malignant transformation is used as the end-point most closely estimate the onset in vivo of neoplastic processes by chemicals; the malignancy of the cell is confirmed by injection of the treated cells into an appropriate host. However, since none of the available tests use the appearance of tumors in mammals as end-points, more than one mutagenicity assay system must be used to ascertain the carcinogenicity and mutagenicity of a chemical. The use of short-term tests with aflatoxins has revealed a good correlation between carcinogenicity and results in rec-assays, mutagenicity assays with *Salmonella typhimurium*, and induction of DNA repair synthesis. For other mycotoxins, such as patulin and luteoskyrin, the data are contradictory. In some instances, short-term tests have allowed determination of the mechanism of action of various carcinogens such as aflatoxin B_1, which forms a covalently bound adduct with hepatic nucleic acids and proteins. (36 refs)

78-2437 **The Effect of Alcohol Consumption on Risk of Cancer of the Head and Neck.** (Eng) Rothman, K. J. (677 Huntington Ave., No. 802, Boston, MA 02115). *In: Head and Neck Cancer. State of the Art Conference, February 16, 17 and 18, 1976.* (St. Louis, MO: Laryngoscope): Vol. 88, No. 1, Part 2, Suppl. 8, pp. 51-55; 1978.

Alcoholics are at increased risk for the development of oral cancers. It is estimated that alcohol plays an etiological role in > 50% of the oropharyngeal cancers in the US. The sites most affected are those that come into direct and prolonged contact with the alcohol, the floor of the mouth and the tongue. The risk from joint exposure to alcohol and tobacco is about 2.5 times the risk expected if the effects of the two substances were additive; a synergistic effect is thus apparent. Although education of the public on the increased risk of oral cancers from alcohol and tobacco use is important, screening programs for at-risk patients would have a more beneficial effect. Research should focus on whether carcinogens in tobacco smoke dissolve in the alcohol and are thus held in more prolonged contact with the oral mucosa or whether certain congeners in the alcohol exert a special effect. (12 refs.)

78-2438 **Assumption of Linearity in Dose-Effect Relationships.** (Eng) Pochin, E. E. (Natl. Radiological Protection Board, Harwell, Didcot, Oxfordshire OX11 OR9, England). *Environ Health Perspect* 22: 103-105; 1978.

Radiation protection standards have developed in three phases. At first, it seemed likely that no harm was caused unless there was a high radiation dose, so that protection from low doses was not considered. It then became clear that even the lowest doses might involve some risk, and the estimate of the possible risk at low dose was made by linear extrapolation from known high dose exposue in human populations developing various types of malignancy. Arguments from microdosimetric and other models have made it increasingly likely that linear and quadratic (ie, dose-squared) terms may determine the dose-effect relationship from low to moderately high dose levels. One of the critical questions is whether the slope of the dose-effect relationship is zero at the dose corresponding to that constantly received from natural sources. A linear hypothesis has also been used to estimate the likely frequency of harm from low doses of chemical substances which have defined harmful effects at high dose. The validity of this procedure depends on the way in which chemical pollutants or their metabolic products become distributed through body tissues and cause the relevant harmful effect on cells. The major difference between the two situations (radiation and chemical carcinogenesis) is the physically predictable nature of the radiation dose to particular tissues or cell structures compared to the less certain knowledge of the distribution and metabolism of chemical pollutants in the body. (12 refs)

78-2439 **Radiation.** (Eng) Upton, A. C. (NCI, Bethesda, MD, 20014). *Bull NY Acad Med* 54(4): 428-434; 1978.

A review of some of the current information on radiation-induced carcinogenesis is presented. It has been suggested that the relation between dose and cancer incidence is linear and that no dose is safe. Extrapolations from dose curves are difficult to assess because of the differing sensitivities of cells to radiation at different doses. The policy question in radiation carcinogenesis is how big is the risk for a given dose. No radiation exposure should be without some benefit, and whatever the exposure, it should be as low as possible. (4 refs)

78-2440 **Irradiated Diets Examined.** (Eng) Cooper, P. (No affiliation given). *Food Cosmet Toxicol* 16(1): 71-73; 1978.

Literature data on the effects of feeding irradiated foods to experimental animals are reviewed. Most of the studies showed no adverse effects of irradiated diets, but one showed that irradiated wheat increased the mutagenic index of nourished and malnourished rats and decreased the number of germ cells in the latter. In another study, polypoid cells were noted in children fed irradiated wheat 4 wk after the start of feeding; none were noted in children fed unirradiated grain. Irradiation has been shown to both increase and have no effect on aflatoxin production. (no refs)

78-2441 **Hiroshima at Home.** (Eng) Commoner, B. (Center Biology Natural Systems, Washington Univ., St. Louis, MO). *Hosp Pract* 13(4): 58, 63; 1978.

A preliminary study of the records of servicemen exposed to radiation from H-bomb tests in Nevada in 1957 indicates an increased incidence of leukemia in these subjects. Studies of workers connected with the uranium mining industry, nuclear submarines, and nuclear installations have found an increased cancer death rate among those exposed to radiation. (no refs)

78-2442 **Virus-associated Malignant Diseases in Animals and Man.** (Eng) Jose, D. G. (Peter MacCallum Clinic, 481 Lt. Lonsdale St., Melbourne, Victoria 3000, Australia). *Aust NZ J Med* 8(2): 195-214; 1978.

Current knowledge of the oncogenicity of DNA and RNA viruses is reviewed. Of the DNA viruses, herpes simplex virus type 2 has been implicated in cervical carcinoma and Epstein-Barr virus in Burkitt's lymphoma and nasopharyngeal carcinoma. Of the RNA viruses, the C-type viruses are associated with leukemias and sarcomas in many species. Molecular hybridization experiments suggest that animal RNA viruses are transmitted horizontally among humans. (253 refs)

78-2443 **Virus and Human Carcinogenesis.** (Ger) Birkmayer, G. D. (Institut fur Zellbiologie, Universitat Munchen, Goethestrasse 33, D-8000 Munich 2, W. Germany). *Wien Med Wochenschr* 127(23): 703-706; 1977.

Although there is no definitive evidence of a viral etiology of human tumors, oncogenic viruses have been isolated from certain human tumors and virus production has been observed in human leukemia cells. In addition, viral reverse transcriptase was found in human leukemia, lymphoma, osteosarcoma, melanoma, glioma, and breast carcinoma. (8 refs.)

78-2444 **Experimental Models and Conceptual Approaches to Studies of Lymphomas and Leukemia: Etiology, Biology, and Control.** (Eng) Ihle, J. N. (Basic Res. Program, NCI Frederick Cancer Res. Center, P.O. Box B, Frederick, MD, 21501). *Semin Hematol* 15(2): 95-115; 1978.

A review of the leukemias and lymphomas of thymus-derived lymphocytes is presented with emphasis on the murine lymphomas. The various topics include viruses and leukemia, spontaneous and environmentally induced leukemias, and leukemias as diseases of differentiation. It is suggested that leukemias with both viral and nonviral etiologies exist and that the former may be amenable to classical immunologic control. (116 refs)

78-2445 **Symposium on Liver Carcinoma. Hepatocellular Carcinoma and Hepatitis B.** (Eng) Zuckerman, A. J. (London Sch. Hygiene and Tropical Medicine, London WC1E 7HT, England). *Trans R Soc Trop Med Hyg* 71(6): 459-461; 1977.

Progress in establishing the progression of hepatitis B to hepatocellular carcinoma is reported. Although the viral surface and core antigens have not been detected in tumor cells, the surface antigen, subtype *ad*, was present in supernatant fluids from the cultured tumor cells of a patient with primary liver cancer, and circulating surface antigen was found in his blood. Liver DNA and RNA from patients with persistent hepatitis B infection and hepatocellular carcinoma significantly accelerated the reassociation rate of radioactive DNA from the 42-nanometer virus particles containing the core antigen. The viral DNA base sequences were alkali-stable, which suggested their attachment to host chromosomes. (13 refs.)

78-2446 **Assembly of Type C Oncornaviruses: A Model.** (Eng) Bolognesi, D. P. (Dept. Surgery, Duke Univ. Medical Center, Durham, NC, 27710); Montelaro, R. C.; Frank, H.; Schafer, W. *Science* 199(4325): 183-186; 1978.

Based on their fine structure, the molecular arrangement of the virion structural components in the particle, and data on the biosynthesis of these components, a model for the assembly of avian and murine C-type oncornavirus is proposed. The model includes the following events. Complexes consisting of the virus envelope components migrate from their sites of synthesis in the cytoplasm to the cell surface and are inserted in the membrane at the site of virus budding. Uncleaved precursor molecules to the internal virus polypeptides are then transported to the budding site, where one end of the precursor molecule is joined to the virus envelope complex and the other end associates with the virus RNA. As viral maturation progresses, proteolytic enzymes cleave the precursor molecules at the appropriate sites, generating the individual structural elements of the virus. The resultant virion components than associate with themselves and/or with other molecules to form the substructures found in the mature virus. The recently established precursor-product relationship between mouse mammary tumor virus A-type particles and mature B-type particles suggests that this class of oncornaviruses may assemble by mechanisms similar to those proposed for C-type particles. (37 refs)

78-2447 **Inhibition of Chemical Carcinogens.** (Eng) Wattenberg, L. W. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, MN); Lam, L. K.; Borchert, P. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 19 pp.; 1978. (no refs)

78-2448 **Erythropoietin and the Control of Red Cell Production.** (Eng) Graber, S. E. (Dept. Medicine, Veterans Admin. Hosp., Nashville, TN, 37203); Krantz, S. B. *Annu Rev Med* 29: 51-66; 1978.

A review of current knowledge on erythropoietin (Ep) is presented. Studies of Ep in cancer patients have indicated that despite the presence of anemia in many patients, there is no increase in Ep levels. The discovery of Ep has also led to a better classification of polycythemia. Polycythemia vera appears to be a clonal disease in which a cell gives rise to erythroid cells in the presence of small amounts of Ep and in which a normal cell line under Ep control is repressed. (109 refs)

78-2449 **The Respiratory Tract and the Environment.** (Eng) Brain, J. D. (Dept. Physiology, Harvard Sch. Public Health, Boston, MA, 02115). *Environ Health Perspect* 20: 113-126; 1977.

The respiratory tract is the most common route of entry of toxic or infectious particles in the working or general environment. Various diseases resulting from inhaled substances and respiratory mechanisms that act to prevent damage are outlined. Neoplastic responses have been observed following inhalation of cigarette smoke, asbestos, polycyclic aromatic hydrocarbons, radioisotopes, chromates, and compounds involved in nickel refining. (32 refs)

78-2450 **Peculiarities in the Occurrence of Adrenal Cortical Carcinoma.** (Eng) Miller, R. W. (Clinical Epidemiology Branch, NCI, A-521 Landow Building, Bethesda, MD, 20014). *Am J Dis Child* 132(3): 235-236; 1978.

A review of patients who developed adrenal cortical carcinoma indicated a substantial incidence of second primary neoplasms, especially those in the brain. The dissimilar neoplasms that occur in family cancer syndromes also develop excessively in these patients as second primary tumors, suggesting a genetic influence. The carcinoma also occurs in patients with congenital hemihypertrophy; and there may be an association between this tumor and others that tend to occur in patients with hemihypertrophy. (15 refs)

78-2451 **Retrospective Analysis of Control Animal Data--The Rat.** (Eng) Haley, T. J. (Natl. Center Toxicological Res., U.S. Dept. Health, Education, and Welfare, Food and Drug Admin., Jefferson, AR, 72079). *Clin Toxicol* 12(2): 249-263; 1978.

Retrospective data are presented on the type, incidence, and latency period of spontaneous tumors in different rat strains. The data are intended as a guide to the selection of control strains in projected studies of carcinogenesis and chronic toxicology. The effect that aging control animals can have on the statistical analysis of experimental data is noted, together with the possible role of virus activation in the development of malignant tumors. (71 refs)

78-2452 **The Role of Epidemiology and Geographic Pathology in Environmental Carcinogenesis.** (Eng) Higginson, J. (International Agency Res. Cancer, Lyon, France). *Bull Am Coll Surg* 63(4): 14-19; 1978.

Although studies have indicated that 70%-90% of all human cancers are related to environmental factors, these factors cover such a wide range (industrial, personal, viral, and general environmental exposure) that total prevention is currently unrealistic. The association of environmental factors with cancer of the esophagus, liver, colon, and rectum is outlined, and the role of physicians in identifying risk factors is reviewed. (13 refs)

78-2453 **Cancer Statistics, 1978.** (Eng) Silverberg, E. (Dept. Epidemiology and Statistics, American Cancer Society, New York, NY) *CA* 28(1): 17-32; 1978.

Figures and tables of estimates of cancer incidence and mortality, based on newly available comprehensive data from the National Cancer Institute's Third National Cancer Survey, are presented. Data are given for all sites and for each major site, but they do not cover carcinoma in situ of the cervix and nonmelanoma skin cancer. According to 1978 estimates of cancer incidence, lung (22%), prostate (16%), and colon and rectum (14%) will be the most common sites in men, breast (26%), colon and rectum (15%), and uterus (33%), in women. Three of the major sites of cancer deaths will be lung (33%), colon and rectum (12%), and prostate (10%) in men, and breast (19%), colon and rectum (15%), and lung (12%) in women. In 1975, cancer was the second leading cause of death (after heart disease) in men and women of all ages (199,443 and 166,250 deaths, respectively). During 1969-1971, the av annual incidence rate (per 100,000) of cancer in children < 15 yr old was 12.5% for all sites, highest for leukemia (4.2), brain and CNS (2.6), lymphoma (1.3), and kidney (0.8). In 1978, the estimated number of new cancer cases in men and women, respectively, will be 352,000 and 348,000 for all sites, 89,400 and 83,200 in the digestive organs, 88,900 and 25,000 in the respiratory system, and 61,800 and 69,200 in the genital organs. The respective highest number of deaths in 1978 will be due to cancer of the digestive organs (55,500 and 50,000), respiratory system (74,300 and 22,850), and genital organs (21,600 and 22,500), with all sites totaling 213,500 and 176,500. Among the remaining statistics are estimated new cancer cases and deaths by state (1978), age-adjusted cancer death rates for 44 countries (1972-1973), and 5-yr survival rates for major sites by stage and sex (1965-1969). (no refs)

78-2454 **Genesis and Spread of Intraepithelial Cervical Neoplasia.** (Ger) Bajardi, F. (Zytologisches Laboratorium, Landeskrankenhaus Graz, Auenbruggerplatz 26, A-8036 Graz, Austria). *Gynaekol Rundsch* 17(1): 7-13; 1977.

Carcinoma in situ and early invasive carcinoma of the uterine cervix are practically always accompanied by epithelial dysplasia. The time required for the dysplasia to progress to carcinoma in situ is estimated at 2-3 yr, and 10-12 yr are required for carcinoma in situ to progress to invasive carcinoma. Intraepithelial neoplasia is the result of transformation of epithelium into another type of epithelium via regeneration (indirect metaplasia). The spread of malignant epithelium is inhibited. Therefore, carcinoma in situ is never able to invade the entire endocervix or ectocervix, in spite of the long latent period involved (10-12 yr). (22 refs)

78-2455 **Eighty Percent of Cancer Is Related to the Environment.** (Eng) Schneiderman, M. A. (NCI, Landow Bldg., Room C403, Bethesda, MD, 20014). *Laryngoscope* 88(4): 559-574; 1978.

A review of epidemiologic data on cancer incidence suggests that as much as 80% of all cancers are related to environmental factors. These factors include personal variables such as cigarette smoking, alcohol consumption, sexual habits, and diet and impersonal variables such as industrial and occupational pollutants, water and air contaminants, pesticides, flame retardants, diet additives, and drugs. Elimination or reduction of these environmental factors would bring about a significant decline in cancer mortality. (18 refs)

78-2456 **An Epidemiologic and Clinical Study of Nasopharyngeal Carcinoma.** (Eng) Ho, J. H. (Medical and Health Dept., Inst. Radiology and Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong). *Int J Radiat Oncol Biol Phys* 4(3/4): 183-198; 1978.

An etiologic hypothesis for nasopharyngeal carcinoma is postulated from current epidemiologic and experimental data. The three factors thought to be involved are Epstein-Barr virus, a genetically determined susceptibility, and an environmental factor that may vary from one population group to another. In the case of the southern Chinese, especially the boat people living along the south China coast, who have the highest cancer risk, the most probable environmental factor is salted fish in the diet coupled with vitamin C deficiency in early life. When added to the diet of WA albino rats, salted fish induced nasal and paranasal carcinomas. Clinical studies showed a good correlation between prognosis and a grouped Tumor Node Metastases (TNM) stage classification. When patients were matched according to the system, no significant difference in radiotherapy results was observed for the two sexes. As a group, Stage I or II patients, but not Stage III patients, <40 yr old had significantly better 5-yr survival rates than patients aged 40 or over. Prophylactic cervical node irradiation did not improve the survival or tumor control prospects of Stage I patients. Therefore, irradiation of the neck should be withheld until nodal metastases become clinically evident. (44 refs)

78-2457 **Genetic Risk Factors for Malignant Tumors in Humans.** (Ger) Rudiger, H. W. (I. Medizinische Universitatsklinik, Martinistrasse 52, 2000 Hamburg 20, W. Germany). *Dtsch Med Wochenschr* 103(2): 77-84; 1978.

Studies of hereditary malignant tumors, precanceroses, and hereditary diseases associated with increased cancer risk are reviewed. Retinoblastoma, Wilms' tumor, neuroblastoma, pheochromocytoma, malignant melanoma, and Sipple's syndrome can be either hereditary or sporadic. (103 refs)

78-2458 **Asbestos Fiber Dust, an Actual Hazard.** (Ger) Weicksel, P. (Facharzt fur Lungen und Bronchial Heilkunde, Kaiserstrasse 26, D-8700 Wurzburg, W. Germany). *Med Klin* 73(10): 351-356; 1978.

Studies of the hazards of inhaled fine asbestos fibers are reviewed. The carcinogenic effect of asbestos fibers is related to their diameter, length, density, and elasticity, and not to their chemical composition or molecular structure. Two-thirds of all pleural mesotheliomas are believed to be due to industrial noxae such as asbestos. An abnormally high incidence of pleural plaques was found in people living near an asbestos mine in Finland; they were never exposed to asbestos occupationally. Asbestosis was diagnosed roentgenologically in an asbestos weaver 5 yr after he retired, and pleural mesothelioma was detected at autopsy. Asbestosis was found in a bricklayer who had been working with asbestos-containing refractory materials for 30 yr. Investigation of 304 patients with chronic bronchitis (198 smokers, 106 nonsmokers), 88 of whom were exposed occupationally to dusts (including asbestos), revealed that both cigarette smoke and asbestos dust enhance obstructive reactions in the bronchial system or are a possible cause of these reactions. Smoking and asbestos dust had only a moderate additive effect. (20 refs)

78-2459 **Epidemiology of Breast Cancer.** (Ger) Maass, H. (Gyn.-geb. Abt. II der Frauenklinik, Zentralkrankenhaus, Kliniken der Freien Hansestadt Bremen, St.-Jurgen-Strasse, 2800 Bremen, W. Germany). *Roentgenblaetter* 31(7): 401-404; 1978.

Epidemiologic data show that breast cancer in women is 8 to 10 times more frequent in Western industrial countries than in the Far East and in some developing countries. In all groups, late primiparas are at increased risk. Other risk factors are a family history of breast carcinoma, nulliparity, proliferating mastopathy, and a prior breast tumor. (no refs)

78-2460 **Benign and Malignant Liver Neoplasms. Some New Etiologic Associations.** (Eng) Rudolph, R. H. (Mason Clinic, P.O. Box 900, Seattle, WA, 98111). *Postgrad Med* 63(3): 56-60, 62, 65; 1978.

Recent studies of benign and malignant hepatic neoplasms have suggested associations between these tumors and oral contraceptives, androgens, hepatitis virus, and vinyl chloride. It is suggested that hepatic arteriography is the best method for diagnosing hepatic adenomas in women using oral contraceptives. (20 refs)

78-2461 **Environmental Aspects of Injury and Disease: Liver and Bile Ducts.** (Eng) Reynolds, E. S. (Dept. Pathology, Univ. Texas Medical Branch, Galveston, TX, 77550). *Environ Health Perspect* 20: 1-13; 1977.

Mechanisms by which environmental agents interact with the liver and cause liver and biliary damage are surveyed. Known hepatotoxins (vinyl chloride, CCl_4), potential hepatotoxins, cohepatotoxins, mixed-function-oxidase inducers, and potential hepatotoxins that could be encountered in various environments are listed. (27 refs)

78-2462 **Epidemiological Studies of Oral Cancer.** (Eng) Pindborg, J. J. (Dept. Oral Pathology, Royal Dental Coll., Copenhagen, Denmark). *Int Dent J* 27(2): 172-178; 1977.

A review of epidemiologic data indicates that oral cancer is characterized by marked geographic differences. In Norway, lip cancer is 20 times more frequent among men than women, and its occurs with a double frequency in rural areas compared with cities. Cancer of the tongue occurs with the same frequency in men and women. Oral cancer incidence rates increase sharply with age, especially for men. In a series of seven Western countries, Canada has the highest incidence of cancer of the lip, but within Canada there are marked differences. Regional differences in lip cancer incidence within Czechoslovakia have been explained by more UV radiation, more spicy food, and pipe-smoking with a short-stemmed pipe. There is a high relative frequency of oral cancer in most countries of Southeast Asia. In Singapore, both male and female Indians and Pakistanis have much higher incidence rates than the other ethnic groups (Chinese, Malays, Europeans). This difference is likely due to the chewing of tobacco together with betel nut and also the occurrence of oral submucous fibrosis. Most of the studies dealing with the etiology of oral cancer have implicated tobacco usage. (16 refs)

78-2463 **The Interactions of Smoking, Environment and Heredity and Their Implications for Disease Etiology. A Report of Epidemiological Studies on the Swedish Twin Registries.** (Eng) Cederlof, R. (Dept. Environmental Hygiene, Karolinska Inst., Stockholm, Sweden); Friberg, L.; Lundman, T. *Acta Med Scand [Suppl]* 612: 105-128; 1977.

Using data compiled by the Swedish Twin Registries, the interactions of smoking, environment, and heredity on disease etiology were reviewed. Morbidity studies revealed associations between smoking and lung cancer and other respiratory diseases. (159 refs)

78-2464 **Re: Smoking and Cancer of the Uterine Cervix: Hypothesis (Letter to Editor).** (Eng) Schoenberg, B. S. (Section on Epidemiology, Natl. Inst. Neurological and Communicative Disorders and Stroke, Bethesda, MD, 20014). *Am J Epidemiol* 107(4): 353-354; 1978.

In a previous study, a high correlation was found between the age-adjusted incidence rates for cancers of the uterine cervix and lung, and a relationship between smoking and cervical cancer was suggested. Literature dealing with multiple primary cancers that also support this association are cited. Each of the cancer cites linked to cervical cancer was strongly correlated with cigarette smoking. Heavy cigarette consumption may be associated with the subgroups of women at high risk of cervical cancer or it may play a more direct role in the etiology of this cancer. (10 refs)

78-2465 **Characterization of Polycyclic Aromatic Hydrocarbons in Sewage Sludge by Gas Chromatography.** (Ger) Grimmer, G. (Biochemisches Institut fur Umweltcarcinogene, Sieker Landstrasse 19, D-2070 Ahrensburg, W. Germany); Bohnke, H.; Borwitzky, H. *Fresenius Z Anal Chem* 289(2): 91-95; 1978.

Eighteen polycyclic aromatic hydrocarbons were determined in sewage sludge samples by gas chromatography combined with mass spectrometry. They included fluoranthene, pyrene, benzo(a)fluorene, benzo(b/c)fluorene, benz(a)anthracene, chrysene, benzo(a)pyrene, perylene, dibenz(a,j)anthracene, and anthanthrene. (6 refs)

78-2466 **Absolute Stereochemistry of *cis*-1,2-, *trans*-1,2-, and *cis*-3,4-Dihydrodiol Metabolites of Phenanthrene.** (Eng) Koreeda, M. (Dept. Chemistry, Johns Hopkins Univ., Baltimore, MD, 21218); Akhtar, M. N.; Boyd, D. R.; Neill, J. D.; Gibson, D. T.; Jerina, D. M. *J Org Chem* 43(6): 1023-1027; 1978.

The absolute stereochemistry of the *cis*-1,2-, *cis*-3,4-, and *trans*-1,2-dihydrodiols of phenanthrene was determined by the exciton chirality circular dichroism method and chemical degradation. The results represent the first determination of the absolute stereochemistry of a bay region dihydrodiol; ie, *cis*-3-hydroxy-4-benzoyloxy-1,2,3,4-tetrahydrophenanthrene. (78 refs)

78-2467 **Glutaminase and Glutamine Synthetase in 20-Methylcholanthrene Treated Mice.** (Eng) Bhattacharya, M. (Indian Inst. Experimental Medicine, Calcutta 700032, India); Maity, P.; Chaudhuri, L.; Banerjee, S.; Shrivastava, G. C. *Indian J Exp Biol* 15(1): 52-53; 1977.

Levels of glutamine synthetase (GS) and glutaminase were studied in the liver and skin of male Swiss mice treated with 20-methylcholanthrene. A single brush stroke of the carcinogen (0.5% in benzene) was applied thrice weekly for an av of 14 wk to the shaved dorsal skin. After the initiation of treatment, hepatic GS activity increased up to 30 days and then slowly decreased with the appearance of hyperplasia. It regained normal levels by 90 days. In animals treated with benzene only, GS activity remained constant after an initial 30-day increase. Glutaminase activity in the skin increased for 90 days, but the activity in benzene-treated animals returned to normal after peaking at 45 days. Glutaminase activity in the liver also increased with tumor progression. It appears that the initial rise in GS activity meets the requirement of glutamine for tumor growth; the remaining enzyme activity was adequate to maintain normal plasma glutamine levels. The rises in glutaminase activity confirm an increased demand for glutamine by the tumor-host system. (16 refs)

78-2468 **Fractionation of 3M KCl Extracts from the Methylcholanthrene Induced Fibrosarcoma, MCA-F (Meeting Abstract).** (Eng) Pellis, N. R. (Univ. Texas Health Science Center, Houston, TX, 77030); Yamagishi, H.; Wiseman, F. C.; LeGrue, S.; Kahan, B. D. *Fed Proc* 37(6): 1562; 1978. (no refs)

78-2469 **Studies on the Specificity of In Vitro-induced Lymphocytotoxicity to Methylcholanthrene-induced Fibrosarcoma Cell Lines.** (Eng) Warnatz, H. (Inst. Policlinic Clinical Immunology, Univ. Erlangen, Univ. Erlangen, D 8520 Erlangen, W. Germany); Koops, A. C.; Meissner, K.; Hiemeyer, K. *Cell Immunol* 35(2): 436-447; 1978.

To study the specificity of cytotoxic reactions to methylcholanthrene-induced fibrosarcoma (MCAF) cells, cytotoxic effector lymphocytes were induced by in vitro immunization of lymph node and spleen cells from C57B16(H2b) and BALB/c(H2d) mice to syngeneic and allogeneic MCAF cell lines. The T-cell-dependent cytotoxicity was specific to target cell lines to which the lymphocytes were immunized in vitro. Lymphocytotoxicity to syngeneic MCAF cells or normal syngeneic fibroblasts could not be induced using normal fibroblasts as stimulators. With allogeneic MCAF cells, cytotoxic reactions to alloantigens, but not to tumor-associated antigens, were detected. Phytohemagglutinin (10 μg/6 x 10^7 lymphocytes) induced a nonspecific cytotoxic effect that increased the specific lysis of target cells: allogeneic and syngeneic target cells were destroyed by the effector cells. The cytotoxicity of the in vitro-immunized lymphocytes was inhibited by incubation with membrane protein preparations from syngeneic MCAF cell lines. However, the membrane extract of one individual syngeneic cell line inhibited the lymphocytotoxicity to all other syngeneic cell lines. Membrane protein preparations from allogeneic MCAF cells or from normal syngeneic fibroblasts were not inhibitory. In vitro-immunized cytotoxic lymphocytes did not impair the in vivo tumor growth. (22 refs)

78-2470 **Solubilization and Partial Characterization of Tumour-specific Transplantation Antigens from**

hemically-induced Sarcomas. (Meeting Abstract). (Eng) ikora, K. (Lab. Molecular Biology, Hills Road, Cambridge, ngland); Koch, G.; Lennox, E. *Br J Cancer* 37(3): 472; 1978. no refs)

8-2471 **Methyl Cholanthrene and Cadmium Induced Changes in Rat Prostate.** (Eng) Scott, R. (Dept. rology, Royal Infirmary, Glasgow, Scotland); Aughey, E. *r J Urol* 50(1): 25-28; 1978.

he effects of cadmium and 20-methylcholanthrene (20-MC) n the prostate gland of Wistar rats were compared. There ere three treatment groups: Group 1 (53 rats) received 0-MC directly in the ventral and dorsolateral lobes of the rostate; Group 2 (207 rats) received 0.05 ml of a 1 M soln nd 0.1 ml of a 1 M soln of $CdCl_2$ directly into the ventral nd dorsolateral lobes of the prostate; Group 3 (50 rats) was ubdivided into five groups that received five incremented oses of 0.05 ml of a 1 M soln $CdCl_2$ soln sc. Tumors were resent in 19 Group 1 animals; stratification was noted in 11 nd hyperplasia in 7. In Group 2, 17 tumors, 52 cases of astrate regression, and 80 cases of stratification were detectd. In Group 3, only 1 early tumor was noted; however, 15 ases of castrate regression, 5 of stratification, and 1 tumor n another organ were found. The most common types of umor found in Group 2 animals were tumors of the supportng tissue; the appearance of clear cells and an increase in the umber of cells in the lining of the acini were also noted. Most f the Group 1 tumors were fibrosarcomas and squamous cell arcinomas. The role of Cd in carcinogenesis and its interacion with Zn, which occurs in high concentrations in several issues, including the prostate, is complicated. Zn can obviate ome of the effects of Cd in experimental animals and it can iminish the likelihood of development of testicular tumors. 15 refs)

8-2472 **Immunochemical Evidence of a Tumor-specific Surface Antigen Obtained by Detergent Soluilization of the Membranes of a Chemically Induced Saroma, Meth-A.** (Eng) Natori, T. (Dept. Pathology, Hokkaido niv. Sch. Medicine, Sapporo, 060, Japan); Law, L. W.; Apella, E. *Cancer Res* 38(2): 359-364; 1978.

he immunochemical characterization of a tumor-specific urface antigen (TSSA), which was first found in the final roduct of partially purified detergent-solubilized pecific tumor rejection antigen (TSTA) of a chemially induced sarcoma, Meth-A, is reported. TSSA as isolated by binding to an in vivo-absorbed rabbit nti-TSSA antiserum and was solubilized by detergent, onidet P40. It was further purified through gel iltration, isotachophoresis, and polyacrylamide gel lectrophoresis. The most highly purified TSSA obtained hrough these procedures retained some TSTA activity, suggesting an association between the two antigenic activities. However, the specific activities of both TSSA and TSTA do not suggest copurification. The values obtained show a specific 50-fold increase for TSSA and less than a 1.2-fold increase for TSTA which appears to suggest the existence of two different molecules. Both antigens have a mol wt of 60,000 and an electrophoretic mobility of α-globulin. (18 refs.)

78-2473 **Carcinogen-Protein Complexes in Hamster Colon and Glandular Stomach During Long-Term Administration of 3-Methylcholanthrene.** (Eng) Brodsky, L. H. (Inst. Cancer Res., Fox Chase Cancer Center, 7701 Burholme Ave., Fox Chase, Philadelphia, PA, 19111); Feinberg, R. F.; Sorof, S. *Cancer Res* 38(6): 1654-1660; 1978.

The types and amounts of carcinogen-protein complexes present in the colon and glandular stomach cytosols of male Syrian BIO 87.20 hamsters following long-term administration of 3-methylcholanthrene (3-MC) were investigated. The hamsters were gastrically intubated with 4.0 mg 3-MC three times a week for 0, 1, 3, 6, or 9 wk. On the third day after the final intragastric dose, each hamster received a single ip injection of 100 μCi ^{3}H-3-MC; 24 hr later, the cytosols were prepared and their constituents resolved according to molecular size by Sephadex G-200 gel chromatography. The carcinogen-protein complexes of the colon and glandular stomach cytosols were of similar and unchanging molecular size distributions during the 9 wk of 3-MC administration. The main carcinogen-protein complexes in both organ cytosols had mol wts of 80,000-90,000 daltons. The formation of these complexes apparently required metabolism of the carcinogen, since only small amounts were generated in vitro by incubation of the carcinogen with the organ cytosols at 1-4 C. More than 97% of the complexes in this size class were from the two organs per se and not from the blood they contained. (20 refs)

78-2474 **Anti-Carcinogenic Properties of Vitamin A and its Structural Analogues Studied in Organ Culture (Meeting Abstract).** (Eng) Lasnitzki, I. (Strangeways Res. Lab., Cambridge, England). *Br J Cancer* 37(3): 475; 1978. (no refs)

78-2475 **Biological Activity and Metabolism of the Retinoid Axerophthene (Vitamin A Hydrocarbon).** (Eng) Newton, D. L. (NCI, Bethesda, MD, 20014); Frolik, C. A.; Roberts, A. B.; Smith, J. M.; Sporn, M. B.; Nurrenbach, A.; Paust, J. *Cancer Res* 38(6): 1734-1738; 1978.

The biological activity of axerophthene, a hydrocarbon analog of retinol that may inhibit mammary carcinogenesis, was investigated both in vitro and in vivo. In tracheal organ culture, axerophthene reversed the keratinization caused by a deficiency of retinoid in the culture medium; its potency was of the same order of magnitude as that of retinyl acetate. The analog also supported growth in Syrian golden hamsters fed a vitamin A-deficient diet, but it was less efficient than retinyl acetate. Axerophthene was considerably less toxic than retinyl acetate when administered repeatedly in high doses to rats. Administration of an equivalent po dose of axerophthene caused much less deposition of retinyl palmitate in the liver than did the same dose of retinyl acetate. However, a greater level of total retinoid was found in the mammary gland after administration of axerophthene. The biological activity of axerophthene in tracheal organ culture suggests that enzymatic mechanisms for the activation of retinoids are not confined to the liver and that nonhepatic tissues may play an important role in the metabolism of retinoids. (20 refs)

78-2476 **Spectroscopic Studies of Carcinogen-DNA Complexes. Structure of the Covalent Benzo(a)pyrene 7,8-Dihydrodiol 9,10-Oxide-DNA Adduct (Meeting Abstract).** (Eng) Geacintov, N. E. (Chemistry Dept., New York Univ., New York, NY, 10003); Prusik, T. *Proc Am Assoc Cancer Res* 19: 88; 1978. (no refs)

78-2477 **The Reaction of Trans-7,8-Dihydroxy-Anti-9,10-Epoxy -7,8,9,10- Tetrahydrobenzo(a)pyrene with DNA Involves Attack at the N^7-Position of Guanine Moieties.** (Eng) Osborne, M. R. (Chemical Carcinogenesis Div., Institute Cancer Res., Chalfont St. Giles, Bucks HP8 4SP, England); Harvey, R. G.; Brookes, P. *Chem Biol Interact* 20(1): 123-130; 1978.

The reaction of trans-7,8-dihydroxy-anti-9,10-epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (anti-BPDE) with DNA labeled with ^{14}C- and ^{3}H-purine precursors indicated that in addition to the previously reported N^2-position of guanine, the reaction also involves the N^7-position of guanine. The hydrocarbon-N^7-guanine product was not detected earlier because it is lost from the DNA very readily at pH 7. The same N^7-product was obtained by the reaction of anti-BPDE with guanine in dimethylformamide. (24 refs)

78-2478 **Identification of the Major Adducts Formed by Reaction of Benzo(a)pyrene Diol Epoxide with DNA in Vitro.** (Eng) Straub, K. M. (Lab. Chemical Biodynamics, Univ. California, Berkeley, CA 94720); Meehan, T.; Burlingame, A. L.; Calvin, M. *Proc Natl Acad Sci USA* 74(12): 5285-5289; 1977.

Covalent binding of the benzo(a)pyrene (BP) metabolite (±)7β,8α-dihydroxy -9α,10α- epoxy -7,8,9,10-tetrahydro benzo(a)pyrene to calf thymus DNA was investigated in a microsome-mediated reaction system. Enzymatic hydrolysis of the carcinogen-modified DNA and subsequent separation of the products via reversed-phase high-pressure liquid chromatography resulted in the detection and isolation of seven distinct compounds. High-resolution mass spectrometry indicated that these products are covalent adducts of deoxyguanosine, deoxyadenosine, and deoxycytidine. The deoxyguanosine and deoxyadenosine adducts involve binding between the activated hydrocarbon (BP diol epoxide) and exocyclic amino groups of the respective pyrines, but the deoxycytidine adduct may involve O^2, N-3, or N^4 alkylation. These results provide an unambiguous determination of the overall molecular structures of the products obtained in the alkylation of DNA by BP diol epoxide, but the effects of these modifications on DNA structure or how they might alter the functional properties of nucleic acids in vivo remain unknown. (29 refs)

78-2479 **Chromosomal Aberration Effects of Benzo[a]pyrene in Male and Female Germ Cells of Mice (Meeting Abstract).** (Eng) Generoso, W. M. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Cain, K. T.; Huff, S. W. *Mutat Res* 53(1): 126; 1978. (no refs)

78-2480 **Dihydrodiol Dehydrogenase: An Important Enzyme in Dihydrodiol-Epoxide Pathway - Mediated Benzo(A)Pyrene Mutagenicity (Meeting Abstract).** (Eng) Bentley, P. (Institut fur Pharmacologie, Universitat Mainz, Obere Zahlbacher Strasse 67, D-6500 Mainz, W. Germany); Vogel, K.; Glatt, H. R.; Platt, K.; Oesch, F. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R7; 1978. (no refs)

78-2481 **Scanning Electron Microscopy of DMBA Transformed C3H/10T1/2 Cells of Varying Oncogenic Potential (Meeting Abstract).** (Eng) Saxholm, H. J. (Inst. Pathology, Univ. Oslo, Rikshospitalet, Oslo 1, Norway); Iversen, O. H.; Reith, A. *Proc Am Assoc Cancer Res* 19: 43; 1978. (no refs)

78-2482 **Effect of NO$_2$ and SO$_2$ Inhalation on Benzo(a)pyrene Metabolism in Rat Lung.** (Eng) Husain, M. M. (Industrial Toxicology Res. Centre, P.O. Box 80, Lucknow, India); Dehnen, W. *Arch Toxicol (Berl)* 40(3): 207-210; 1978.

Male Wistar rats were continuously exposed to NO_2 (14.4 ppm) and/or SO_2 (46.5 ppm), and the effect of these gases on lung microsomal aryl hydrocarbon [benzo(a)pyrene(BP)] hydroxylase (AHH) activity was determined. Prior to exposure animals were administered methylcholanthrene (MC) to investigate the effect of the exposure on enzyme inducibility and pattern of BP metabolites. NO_2 significantly increased AHH activity, but no marked change was noted with SO_2. Induction of AHH by MC was inhibited markedly by SO_2, only slightly by $NO_2 + SO_2$, but not at all by NO_2. (15 refs)

78-2483 **Aryl Hydrocarbon Hydroxylase in an Insect, *Spodoptera eridania* (Cramer).** (Eng) Anderson, R. S. (Donald S. Walker Lab., Sloan-Kettering Inst. Cancer Res., 145 Boston Post Road, Rye, NY, 10580). *Comp Biochem Physiol [C]* 59(1): 87-93; 1978.

The ability of the armyworm (*Spodoptera eridania*) to metabolize benzo(a)pyrene (BP) was determined. Aryl hydrocarbon hydroxylase (AHH) activity was measured by a highly sensitive radioisotopic method. *S. eridania* larvae metabolized BP via a microsomal mixed-function oxidase system. AHH activity required the presence of NADPH and O_2 in the medium, but it was inhibited by CO, indicating cytochrome involvement. Optimal reaction conditions were a temperature of 30 C and a pH of 7.6-8.0. Measurable BP products were observed within 2 min following initiation of the reaction. The reaction was linear for 20 min, after which it rapidly leveled off; however, BP metabolism continued throughout the 60-min experimental period. The midgut, or stomach, appeared to be the only tissue rich in BP hydroxylase. Subcellularly, AHH was found to be located primarily in the microsomal fraction. The levels of AHH activity were low in young larvae; they gradually increased with maturity but then declined during the prepupal stage. The activity of BP hydroxylase in mice (male C57BL/6 and male Camm Swiss-Webster) is six times that in *Spodoptera*. This study shows that microsomal BP-metabolizing enzyme systems can be found in phyla lower than the vertebrates and, therefore, that BP may be potentially carcinogenic for insects. (21 refs)

78-2484 **Biological Properties of an Olefin Sulfate.** (Rus) Pyleva, Z. A. (A. N. Sysin Inst. General and Communal Hygiene, USSR Acad. Medical Sciences, Moscow, USSR); Voronin, V. M.; Sautin, A. I. *Vopr Onkol* 24(1): 55-60; 1978.

To determine whether surface-active compounds can enhance polycyclic aromatic hydrocarbon-induced carcinogenesis in experimental animals, CBA x C57BL/6 mice received a single epicutaneous application of benzo(a)pyrene (BP: 100 μg). The animals were then divided into two groups, and 2 wk after BP application the mice in Group 1 received an application of Progress-1 (a preparation with a 16.5% content of a surface-active compound, an olefin sulfate) and the mice in Group 2, an application of Progress-2 (a preparation with a 24.5% content of olefin sulfate). Progress-1 and Progress-2 alone did not induce skin tumors, but 19.4% of the mice exposed to BP alone developed skin tumors (av latent period was 53.7 wk). Combined exposure to BP and Progress-2, however, significantly enhanced tumorigenesis: 68.7% of the mice developed skin tumors with an av latent period of 40.9 wk. The corresponding figures for mice exposed to BP and Progress-1 were 19.6% and 53.3 wk. (19 refs)

78-2485 **Comparison of Benzo(a)pyrene Metabolite Formation in Liver Microsomes and Cultured Primary Rat Liver Parenchymal Cells (Meeting Abstract).** (Eng) Huisingh, J. L. (Environmental Protection Agency and Northrop Services, Res. Triangle Park, NC, 27711); Nesnow, S.; Selkirk, J. K.; Inmon, J. P.; Bergman, H.; Russell, B.; Waters, M. D. *In Vitro* 14(4): 347; 1978. (no refs)

78-2486 **Activation of Mutagens by Small Bowel Mucosa (Meeting Abstract).** (Eng) Halpin, T. C. (Dept. Pediatrics, Case Western Reserve Univ., Cleveland, OH); Speck, W. T. *Gastroenterology* 74(5, part 2): 1126; 1978. (no refs)

78-2487 **Different Tumours Induced by Benzo(a)pyrene and Its 7,8-Dihydrodiol Injected into Adult Mouse Salivary Gland.** (Eng) Wigley, C. B. (Dept. Cellular Pathology, Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2 A3PX, England); Amos, J.; Brookes, P. *Br J Cancer* 37(5): 657-661; 1978.

Tumors induced by the injection of benzo(a)pyrene (BP) or its proposed proximate carcinogen, benzo(a)pyrene 7,8-dihydrodiol (BP 7,8-dihydrodiol), into the submandibular salivary gland of adult C57BL mice were compared. Mice were injected with 0.1, 0.5, or 2.0 mg BP/mouse or 0.1, 0.25, 0.5, or 1.0 mg BP 7,8-dihydrodiol/mouse. The mice were checked regularly for 1 yr for the presence of tumors. BP induced salivary gland tumors at injection site, but BP 7,8-dihydrodiol induced malignant lymphosarcomas at distant sites, particularly the thymus gland. Many of these lymphosarcomas were metastatic to one or more additional sites, such as lung, liver, and kidney, whereas the BP-induced tumors did not metastasize. The highest dose of both compounds induced fewer tumors than the optimal 0.5 mg/mouse. BP was more active than its derivative in this system (90% and 50% tumor incidences, respectively, at 0.5 mg). The different sites of action of the two carcinogens may be due to their different water solubilities and toxicity. (24 refs)

78-2488 **Conformation of Dinucleoside Monophosphates Modified with Benzo[a]pyrene-7,8-dihydrodiol 9,10-Oxide as Measured by Circular Dichroism.** (Eng) Frenkel, K. (Inst. Cancer Res. and Dept. Biochemistry and Medicine, Columbia Univ., Coll. Physicians, New York, NY, 10032); Grunberger, D.; Boublik, M.; Weinstein, I. B. *Biochemistry (Easton)* 17(7): 1278-1282; 1978.

The conformation properties of GpU modified with the reactive derivative of benzo(a)pyrene (BP), (+)-7β,9α-dihydroxy- 9-5a-10α-epoxy- 7,8,9,10-tetrahydrobenzo(a)pyrene (BPDE), were investigated using circular dichroism spectroscopy. Binding of BPDE to the N^2 of guanosine residues in GpU resulted in the formation of four compounds representing two pairs of diastereoisomers. The molar ellipticity values of the modified dimers were two times higher than those of the modified guanosine monomers. These values were decreased appreciably when the spectra of the dimers were obtained at 80 C or in methanol, rather than at 25 C in water, suggesting that under the latter conditions there is a stacking interaction between the carcinogen and the neighboring uridine residue. Based on these results, a conformation is proposed for modified GpU, it includes insertion of the BP moiety, by rotation of the modified guanine residue about its glycoside bond, coplanar to the neighboring uridine and perpendicular to the phosphodiester backbone. Although this is the most likely interpretation, the data do not exclude other mechanisms. Definitive assignments of the conformations of BPDE-modified dimers require further studies. (29 refs)

78-2489 **A Rapid Spectrofluorometric Method for the Analysis of Polycyclic Aromatic Hydrocarbons in Animal Tissues (Meeting Abstract).** (Eng) Mitchell, C. E. (Inhalation Toxicology Res. Inst., P.O. Box 5890, Albuquerque, NM, 87115). *Fed Proc* 37(6): 1710; 1978. (no refs)

78-2490 **Effect of Monooxygenase Inducers on the Binding of Benzo(A)Pyrene Metabolites to Cellular Macromolecules in Perfused Rat Lungs (Meeting Abstract).** (Eng) Klaus, E. (Dept. Pharmacology, Univ. Mainz, Obere Zahlbacher Strasse 67, D-6500 Mainz, W. Germany); Deckers-Schmelzle, B.; Kahl, G. F. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R23; 1978. (no refs)

78-2491 **Morphology of Experimentally Induced Respiratory Tumors in Syrian Golden Hamster. A Histological, Histochemical and Ultrastructural Study.** (Eng) Stenback, F. (Dept. Pathology, Univ. Oulu, Oulu, Finland). *Acta Otolaryngol Suppl (Stockh)* 347: 1-59; 1977.

Respiratory tumors and their precursors induced by intratracheal (it) instillation of polycyclic hydrocarbons [benzo(a)pyrene, 9,10-dimethylbenz(a)anthracene, 7H-dibenzo(c,g)-carbazole, or dibenz(a,i)pyrene] in Syrian golden hamsters were studied and compared with lung tumors and nonneoplastic lesions in man. The early metaplastic changes in the tracheobronchial epithelium were similar to those in humans, and they included squamous and transitional cell metaplasia, lack of cilia, formation of club-shaped or multibranched cilia, dysplastic alterations, cytological abnormalities, and widened intercellular spaces with villous interdigitations and sparse desmosomes. A significantly increased incidence of metaplastic and hyperplastic changes was observed prior to the manifestation of visible tumors. The most frequent malignant tumors in hamsters were squamous cell carcinomas of varying differentiation. Other induced tumor types included epithelial adenocarcinomas, sarcomas, carcinosarcomas, and combined adenoepidermoid carcinomas. Of the 852 tumors observed, 34% were benign and 66% were malignant. The av latency period was 42 wk, with undifferentiated tumors having a shorter latent period. Concerning location, 37% were located in the trachea, 20% in the main bronchi, 9% in the bronchiolar area, and 34% in the peripheral lung. The results show that instillations of carcinogens induce a large number of tumors of different types that are also found in man. (167 refs)

78-2492 **Benzo[a]pyrene Metabolizing Activity of Cultured Cells as Determined by a Simplified Radiometric Method.** (Eng) Miyazawa, T. (Dept. Experimental Radiology, Aichi Cancer Center Res. Inst., Tashiro-cho, Chikusa-ku, Nagoya 464, Japan); Enaka, K.; Umeda, M. *Gann* 68(6): 737-744; 1977.

Benzo(a)pyrene (BP) metabolism in cultured mammalian cells was measured by a simplified method. Cells growing in the bottom of glass test tubes were treated with 2 μM of ^{14}C-BP for 24 hr; unmetabolized BP was then extracted with organic solvents, and the amount of water-soluble products recovered in the aqueous phase was measured by a liquid scintillation counter. Among rodent embryo cells, embryo cells of C3H/He mice and Syrian hamsters showed greater BP-metabolizing activity than those of DDD, AKR, and C57BL/6 mice. Several cell lines sensitive to density-dependent inhibition (Y-AK, DL1, and C3H/10T1/2) actively metabolized BP to water-soluble products. The metabolizing activity of C3H/He mouse embryo cells was markedly enhanced by pretreating the cells with benz[a]anthracene. The amount of water-soluble BP products from phytohemagglutinin-stimulated human lymphocytes was much less than that from most cultured cell lines and embryo cells, but the amount increased with time in culture. (30 refs)

78-2493 **A Human Plasma Protein that Binds Benzo(a)-Pyrene (Meeting Abstract).** (Eng) Busbee, D. L.

(North Texas State Univ., Denton, TX, 76203); McKenzie, M.; Snodgrass, D.; Wray, N. P.; McLemore, T.; Cantrell, E. T. *Fed Proc* 37(6): 1721; 1978. (no refs)

78-2494 **Tentative Identification of Benzo[a]pyrene Metabolite-Nucleoside Complexes Produced In Vitro by Mouse Liver Microsomes.** (Eng) Pelkonen, O. (Developmental Pharmacology Branch, Natl. Inst. Child Health and Human Development, NIH, Bethesda, MD, 20014); Boobis, A. R.; Yagi, H.; Jerina, D. M.; Nebert, D. W. *Mol Pharmacol* 14(2): 306-322; 1978.

Seven of nine column chromatography peaks that are consistently found during the binding of benzo(a)pyrene (BP) to DNA in vitro have been tentatively identified. ^{3}H-BP was incubated in vitro with deproteinized salmon sperm DNA, NADPH, and mouse liver microsomes. The resulting covalently bound BP metabolite-nucleoside complexes were resolved into nine distinct peaks by elution of a Sephadex LH-20 column with a water-methanol gradient. Using synthetic and biologically produced metabolites, 7/9 peaks were assigned to one or more BP metabolites. Four of the peaks contained products of BP quinones that are further metabolized. One peak reflected almost exclusively both the *cis-* and *trans*-7,8-diol 9,10-epoxides of BP. Another peak represented predominantly the K-region metabolite (4,5-oxide), interacting with one or more nucleosides. The seventh identified peak comprised reactive intermediates resulting from the further metabolism of BP phenols. BP may therefore be metabolized to four different types of reactive intermediates capable of binding to DNA: primary arene oxides, diol epoxides, phenols oxygenated further, and quinones oxygenated further (or quinone-derived free radicals). The last three types of microsomally activated intermediates are the result of two- or three-step enzymatic processes in which cytochrome P-450-mediated monooxygenations occur at least two times. (50 refs)

8-2495 **Investigations on the Carcinogenic Burden by Air Pollution in Man. Intratracheal Instillation Studies with Benzo(a)pyrene in Bovine Serum Albumin in Syrian Hamsters.** (Eng) Ketkar, M. (Abteilung fur Experimentelle Pathologie, Medizinische Hochschule Hannover, Karl-Wiechert-Allee 9, 3000 Hannover 61, W. Germany); Reznik, G.; Schneider, P.; Mohr, U. *Cancer Lett* 4(4): 235-239; 1978.

Male and female Syrian golden hamsters were given weekly intratracheal instillations of 0 (Group 1), 0.1 (Group 2), 0.33 (Group 3), or 1.0 mg (Group 4) of benzo(a)pyrene (BP) in 0.2 ml 10% bovine serum albumin for life and examined for the occurrence of tumors. No respiratory tract tumors were noted in Group 1. Of 26 Group 2 males, 5 had lung tumors; of 30 females, 2 had tracheal and 10 had lung tumors. Of 29 Group 3 males, 5 had tracheal and 2 had lung tumors; of 28 females 7 had tracheal and 6 had lung tumors (total of 10 affected females). Of 27 Group 4 males, 5 had tracheal and 2 had lung tumors (6 affected); of 30 females, 3 had tracheal and 6 had lung tumors (6 affected). Survival rates decreased significantly with increasing BP doses. The increased lung tumor incidence was related to longer survival time; tracheal tumor incidence rose with increasing doses. The tracheal neoplasms were all squamous cell papillomas with the exception of a squamous cell carcinoma in Group 2. All lung neoplasms in Group 2 were bronchogenic adenomas; similar tumors occurred in Groups 3 and 4, in addition to one adenocarcinoma in a Group 3 female and squamous cell carcinomas in three Group 4 females. Respiratory tract lesions, ie, metaplastic and dysplastic alterations of the epithelium as well as the occurrence of alveolar macrophages containing brown pigmentation, were more frequent in females than in males. Respiratory tract tumors were also more frequent in females than in males. (9 refs)

78-2496 **Metabolism of Benzo(a)Pyrene by Human Lymphocytes from Donors of Low, Intermediate, and High Aryl Hydrocarbon Hydroxylase Inducibility (Meeting Abstract).** (Eng) Vaught, J. B. (Roswell Park Memorial Inst., Buffalo, NY, 14263); Gurtoo, H. L.; Paigen, B.; Minowada, J.; Sartori, P.; Tabron, D. *Clin Res* 26(3): 595A; 1978. (no refs)

78-2497 **The Relationship Between Transformation and Somatic Mutation in Human and Chinese Hamster Cells.** (Eng) Spandidos, D. A. (Dept. Medical Genetics, Univ. Toronto, Toronto, Ontario, M5S 1A8, Canada); Siminovitch, L. *Cell* 13(4): 651-662; 1978.

The frequencies of benzo(a)pyrene (BP)- and urethane-induced transformation of primary human and Chinese hamster lung fibroblasts were compared with spontaneous and induced frequencies of mutation for resistance to thioguanine and ouabain and for ability to use fructose. The rates and frequencies of mutation were similar in the two cell systems, but transformation to morphologically altered cells was observed only in hamster cells. Optimal concentrations for hamster cell transformation were 1.0×10^{-6} M BP and 1.1×10^{-2} M urethane. The frequency of transformation in these cells was about 10^3 greater than the frequencies of mutation. The chemically transformed hamster cells did not grow in agar (*aga*-) and did not produce tumors when injected into animals. The frequency of transition of these cells to *aga*+ cells, which produce tumors in animals, was similar to the mutationlike events. (41 refs)

78-2498 **Quantitative In Vitro Metabolism of Benzo(a)pyrene (BP) to Intermediates Cytotoxic and Mutagenic to Mammalian Cells (Meeting Abstract).** (Eng) Kouri, R. E. (Dept. Biochemical Oncology, Microbiological Associates, Bethesda, MD, 20016); Joglekar, R.; Schechtman, L. M. *Mutat Res* 53(1): 100; 1978. (no refs)

78-2499 **Accelerated Carcinogen Metabolism in Human Skin Treated with Coal Tar (Meeting Abstract).** (Eng) Bickers, D. R. (Dept. Medicine, Case Western Reserve Univ., Cleveland, OH). *Clin Res* 26(3): 297A; 1978. (no refs)

78-2500 **Benzo(a)pyrene Metabolism and Blast Transformation in Peripheral Blood Mononuclear Cells from Smoking and Nonsmoking Populations and Lung Cancer Patients.** (Eng) Jett, J. R. (Mayo Clinic and Mayo Foundation, Rochester, MN 55901); Moses, H. L.; Branum, E. L.; Taylor, W. F.; Fontana, R. S. *Cancer* 41(1): 192-200; 1978.

Benzo(a)pyrene (BP) metabolism and lymphocyte transformation in peripheral blood mononuclear cells were evaluated in three groups of male patients: 50 healthy nonsmokers, 50 smokers ($>$ 1 pack/day, and 50 lung cancer patients, primarily Stage I, evaluated before radiation therapy or chemotherapy. BP metabolism was assayed by quantitation of the water-soluble products produced from ^{3}H-BP over an 8-hr reaction, and lymphocyte transformation was measured by ^{3}H-thymidine incorporation. The mean level of BP metabolism was significantly higher in the smoking control group, but it was not significantly different in the nonsmoking control and the lung cancer groups. Although 38/57 lung cancer patients had Stage I disease, lymphocyte transformation was significantly lower in the patient group compared with the two control groups. Two factors indicated that the degree of lymphocyte transformation by mitogens influences BP metabolism. First, the mean level of BP metabolites in lung cancer patients with lymphocyte stimulation $< 10^4$ cpm was significantly lower than the level in patients with stimulation $> 10^4$ cpm. Secondly, when mononuclear cells from three control patients were stimulated with variable mitogen concentrations, there was a significant correlation between water-soluble metabolite production and the degree of lymphocyte transformation. (33 refs.)

78-2501 **The Effect of Asbestos-associated Metal Ions on the Binding of Benzo(a)pyrene to Macromolecules In Vitro.** (Eng) Thomson, R. (Natl. Res. Inst. Occupational Diseases, South African Medical Res. Council, P.O. Box 4788, Johannesburg 2000, S. Africa); Kilroe-Smith, T. A.; Webster, I. *Environ Res* 15(2): 309-319; 1978.

The effect of asbestos-associated trace metals on the degree of binding of benzo(a)pyrene (BP) to DNA and other cellular macromolecules was tested in vitro using a rat liver microsomal activation system. Most of the metals tested (nickel, cadmium, chromium, zinc, manganese, and copper) decreased the binding of BP in microsomal incubations containing exogenous DNA. That this represented a reduction in the binding to DNA was confirmed in the cases of zinc, nickel, and chromium. Magnesium caused a slight increase in binding, and iron, both ferrous and ferric, while reducing the binding to DNA, increased the binding to some high-mol-wt component in the incubation mixtures over a narrow concentration range. These results provide evidence that trace metals may play some part in the binding of BP to cellular macromolecules and, therefore, in its carcinogenicity. The effect of iron in increasing the binding of BP in vitro may be particularly significant, because both crocidolite and amosite asbestos have high iron contents. These findings support previous suggestions that metals, such as iron, contaminating crocidolite may be implicated in the development of malignant neoplasia. (42 refs)

78-2502 **Chronic Effects of Ingested Asbestos in Rats.** (Eng) Cunningham, H. M. (Health Protection Branch, Health and Welfare, Ottawa, Ontario, Canada); Moodie, C. A.; Lawrence, G. A.; Pontefract, R. D. *Arch Environ Contam Toxicol* 6(4): 507-513; 1977.

Two lifetime experiments, one using 20 male Wistar rats and one using 80 rats, were performed to determine the effects of chronic po administration of chrysotile asbestos on cancer induction. Half the rats in each study were fed a diet containing 1% asbestos. In the first experiment there were one benign tumor and six malignant tumors in the treated rat compared to one benign and one malignant tumor in the controls. There were 36 nonneoplastic lesions in controls and 34 in the treated group. In the second experiment, there were 38 benign and 11 malignant tumors in the treated groups compared to 37 benign and 11 malignant tumors in the controls. There were 327 nonneoplastic lesions in controls and 324 in the treated rats. In a third experiment, 10/20 rats were fed a 1% asbestos diet for 6 wk, and various tissues and blood were examined for the presence of asbestos fibers. The highest levels of asbestos occurred in the omentum (9.66×10^6 fibers/g vs 1.08×10^6 fibers/g in controls), followed by the brain, lung, liver, blood, and kidney. It is concluded that trace amounts of ingested asbestos can penetrate the walls of the gastrointestinal tract of rats; there is insufficient evidence that these fibers can cause cancer. (20 refs)

78-2503 **Ultrastructure of the Lung in the Rat Following Exposure to Crocidolite Asbestos and Quartz.**

Eng) Miller, K. (Natl. Res. Inst. Occupational Diseases, outh African Medical Res. Council, Johannesburg 2000, S. frica); Webster, I.; Handfield, R. I.; Skikne, M. I. *J Pathol* 24(1): 39-44; 1978.

he effects of crocidolite asbestos and quartz on alveolar acrophages and the subsequent fibrogenic reaction in the ng were studied in male Wistar rats exposed to asbestos ,350 fibers/cm^3) or quartz particles (11 mg/m^3) for 8 hr/ ay, 5 days/wk. The animals were killed after 5-7 mo and e lungs prepared for examination. Lung tissue from rats posed to either substance demonstrated thickening of the terstitial tissue and the presence of collagen fibers. Aggre- ates of macrophages in the alveolar spaces were a dominant ature of asbestos-exposed rats. All macrophages were ma- re cells, indicating that the asbestos was nontoxic. Rats posed to quartz did not have aggregated macrophages in e alveolar spaces, and many macrophages showed a charac- ristic pattern of vacuole formation. Other cells contained tracellular membranous lamellated bodies, similar to those und in Type II pneumocytes. Cells containing lamellated odies were also found in the interstitial tissue. Plasma cells ere present in lung specimens from quartz-dusted rats. hese findings suggest that the two mineral dusts have differ- t biological effects on the macrophage and on the develop- ent of pulmonary fibrosis. (34 refs)

-2504 **Mass and Number of Fibres in the Pathogenesis of Asbestos-related Lung Disease in Rats.** (Eng) avis, J. M. (Inst. Occupational Medicine, Roxburgh Place, dinburgh EH8 9SU, Scotland); Beckett, S. T.; Bolton, R. ; Collings, P.; Middleton, A. P. *Br J Cancer* 37(5): 673-688; 978.

he effects of both fiber number and fiber mass in asbe- os-related lung disease were examined. Five groups of white PF rats of the Han strain were exposed to preparations of ree commercially used types of asbestos (chrysotile, ocidolite, and amosite) for 7 hr/day, 5 days/wk, for a total 224 days during a 1-yr period. Animals were killed 12 and 3 mo and 860 days after the start of exposure. Chrysotile ust produced significantly more lung fibrosis than either nphibole type, even when the fiber numbers in the dust ouds were similar. All malignant pulmonary neoplasms denocarcinomas, squamous carcinomas) occurred in ani- als treated with chrysotile. Only two mesotheliomas were und, one solitary spindle-cell tumor in the pleural cavity an animal treated with crocidolite and an abdominal meso- elioma in an animal that had inhaled chrysotile. When fiber ngth distributions were calculated using a scanning electron icroscope, it was found that the chrysotile clouds contained any more fibers >20 μM long than either of the other ouds. The results, therefore, support previous suggestions at long asbestos fibers are more dangerous than short ones, id they indicate that neither a single mass standard nor fiber umber standards are satisfactory. (32 refs)

78-2505 **Effects of Chrysotile Asbestos Fibers on Syrian Hamster Embryonic Cell Membrane Glycolipids (Meeting Abstract).** (Eng) Saat, Y. A. (Dept. Pathology Radiology, Coll. Medicine, Ohio State Univ., Columbus, OH, 43210); Newman, H. A.; Allison, D. K.; Hart, R. W. *Fed Proc* 37(6): 1645; 1978. (1 ref)

78-2506 **Metabolism and Tissue Binding of Diethylstilbestrol (DES) in the Pregnant Rat: An In Vitro Study (Meeting Abstract).** (Eng) Miller, R. K. (Dept. Obstetrics and Gynecology, Univ. Rochester, Rochester, NY); Laveman, L. B.; Reich, K. A. *Teratology* 17(2): 33A; 1978. (no refs)

78-2507 **Localization of ^{3}H-Estradiol-17β in Diethylstilbestrol-induced Adenosis.** (Eng) Kalland, T. (Inst. Anatomy, Univ. Bergen, Arstadvn 19, Bergen, N-5000, Norway); Fossberg, T. M.; Forsberg, J. G. *Obstet Gynecol* 51(4): 464-467; 1978.

The uptake and distribution of ^{3}H-estradiol-17β was studied in normal and diethylstilbestrol (DES)-treated mice. Newborn females were given five daily injections of 5 μg DES beginning within 24 hr of birth. At 6 mo, all estrogenized animals were ovariectomized and, 24 hr later, each animal received 2 μCi/kg ^{3}H-estradiol-17β sc. The animals were sacrificed 2 hr later. A heavy labeling was noted in the uterine tissue of control animals; stromal and epithelial cells were equally labeled. Estrogenized animals showed the same results in specimens taken from the uterus proper. No difference between control and estrogenized animals was noted in specimens from the uterine cervix and upper vagina; however, the overall picture was of weaker labeling than in the uterus proper. Areas of adenosis in the estrogenized animals showed the same intensity of labeling as the epithelial cells in the uterus. Autoradiograms of these regions showed a marked labeling over the epithelial cells and a nuclear concentration of grains. It is concluded that the adenosis cells in mice are target cells for estrogen. Thus, the development of adenosis in mice and clear cell adenocarcinoma in humans at puberty may be due to the hormonal action of estrogens. Estrogen exposure, including oral contraceptive use, should be avoided by women with vaginal adenosis. (22 refs)

78-2508 **Pathologic Effects Induced in Mice by Diets Containing Diethylstilbestrol or 17β-Estradiol (Meeting Abstract).** (Eng) Highman, B. (Univ. Arkansas Medical Sciences, Little Rock, AR); Norvell, M. J.; Greenman, D. L.; Shellenberger, T. E. *Lab Invest* 38(3): 348; 1978. (no refs)

78-2509 **Diethylstilbestrol-induced Upper Genital Tract Abnormalities (Meeting Abstract).** (Eng) Haney, A. F. (Div. Reproductive Endocrinology, Dept. Obstetrics and Gynecology, Duke Univ. Medical Center, Durham, NC); Hammond, C. B.; Soules, M. R.; Creasman, W. T. *Fertil Steril* 29(2): 235-236; 1978. (no refs)

78-2510 **Squamous and Glandular Dysplasia in Vaginal Adenosis (Meeting Abstract).** (Eng) Antonioli, D. A. (Dept. Pathology, Beth Israel Hosp., Boston, MA, 02215); Rosen, S.; Burke, L.; Donahue, V. *Lab Invest* 38(3): 331; 1978. (no refs)

78-2511 **The Effects of Prenatal Diethylstilbestrol (DES) Exposure on the Genitalia of Pubertal *Macaca Mulatta*** (Meeting Abstract). (Eng) Hendricks, A. G. (Dept. Pathology and Obstetrics, California Primate Res. Center, Univ. California, Davis, CA); Benirschke, K.; Thompson, R. S.; Ahern, J.; Lucas, W. E.; Oi, R. *Teratology* 17(2): 23A; 1978. (no refs)

78-2512 **Estrogenic Activity In Vivo and In Vitro of Some Diethylstilbestrol Metabolites and Analogs.** (Eng) Korach, K. S. (Lab. Environmental Toxicology, Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709); Metzler, M.; McLachlan, J. A. *Proc Natl Acad Sci USA* 75(1): 468-471; 1978.

Three diethylstilbestrol (DES) metabolites and three DES analogs were tested for their estrogenic activity in vivo and in vitro. The metabolites were β-dienestrol, DES α,α′-epoxide (DES epoxide), and α,α′-dihydroxy DES (dihydroxy-DES); the analogs were α-dienestrol, DES-dihydroxydiethylphenanthrene (DES phenanthrene), and 1-(α-ethyl-4α-hydroxyphenyl)indanyl-5-ol (indanyl-DES). As determined by the immature mouse uterine wt bioassay in CD-1 mice, the in vivo estrogenicity of the compounds was in the order DES > α-dienestrol ⩾ DES epoxide > indanyl-DES > dihydroxy-DES > β-dienestrol > DES phenanthrene. In vitro competition equilibrium binding experiments for the uterine estradiol cytosol receptor yielded a similar order of estrogenicity, except that indanyl-DES had a binding affinity comparable to that of DES and α-dienestrol. Sucrose gradient analysis of the competition preparations demonstrated the displacement of ^{3}H-estradiol from the receptor peak. This displacement was receptor-specific, and it correlated with the equilibrium binding concentrations. In addition, the most hormonally active substances had the greatest effect on the association rate reaction of estradiol and the cytoplasmic receptors. These results are discussed in relation to the metabolism and action of DES. (22 refs)

78-2513 **In Utero Exposure to DES. Evaluation and Followup of 199 Women.** (Eng) Fowler, W. C. (Dept. Obstetrics and Gynecology, Div. Gynecologic Oncology, Univ. North Carolina, Chapel Hill, NC, 27514); Edelman, D. A. *Obstet Gynecol* 51(4): 459-463; 1978.

A total of 180 females with known in utero exposure to diethylstilbestrol (DES) and 19 with histories and/or pelvic examinations suggestive of DES exposure were examined. The subjects ranged in age from 12 to 30 yr (mean, 21.6 yr). Only 13.6% had normal colposcopic findings in the vagina and cervix. The incidence of colposcopically detected lesions was not related to the trimester of DES exposure, the patient's age, use of oral contraceptives, or presenting symptoms. Colposcopically directed biopsies of abnormal or suspicious areas revealed 36 cases of cervical intraepithelial neoplasia (CIN), Grade 1 lesions, and 8 cases of CIN, Grade 3 lesions. A total of 28 patients had vaginal adenosis and 62 had one or more of the following cervical abnormalities: cervical bands, cock's combs, and hoods. Areas of punctation, mosaic patterns, white epithelium, and keratosis were not considered areas of adenosis. No cases of clear cell adenocarcinoma were noted. It is suggested that women with in utero DES exposure are at a greater risk than nonexposed women for the development of squamous neoplasia. (15 refs)

78-2514 **Endometrial Carcinoma Following Estrogen Therapy for Breast Cancer. Report of Three Cases.** (Eng) Khandekar, J. D. (Dept. Medicine, Evanston Hosp., 2650 Ridge Ave., Evanston, IL, 60201); Victor, T. A.; Mukhopadhyaya, P. *Arch Intern Med* 138(4): 539-541; 1978.

Three women developed endometrial carcinoma after treatment of their breast cancer with diethylstilbestrol (DES). The first was a 61-yr-old woman who had undergone bilateral salpingo-oophorectomy 29 yr prior to the development of breast cancer. DES therapy (15 mg/day, po) was initiated when bone and liver scans demonstrated metastatic carcinoma. Seven years later, the endometrial carcinoma was diagnosed. The second woman was 69 yr old when she underwent right radical mastectomy for carcinoma in her right breast; 28/29 axillary and 6 right subclavian lymph nodes were positive. DES (15 mg/day, po) was administered upon recurrence in the right axilla 1 yr later. Hysterectomy and bilateral salpingo-oophorectomy, performed 1 yr later upon suspicion of a left ovarian tumor, revealed the endometrial carcinoma. The third was a 64-yr-old woman who received DES (15 mg/day, po) for pleural effusion 2.5 yr after modified radical mastectomy for a tumor. Endometrial biopsy 32 mo after initiation of therapy revealed the carcinoma. These findings support the hypothesis that endometrial carcinoma an occur in breast cancer patients receiving DES therapy. 17 refs)

8-2515 **Exogenous Oestrogens and Ovarian Cancer (Letter to Editor).** (Eng) Hoover, R. (Environ-nental Epidemiology Branch, NCI, Bethesda, MD, 20014); raumeni, J. F.; Gray, L. A. *Lancet* 1(8059): 325-326; 1978.

n a clarification of a previous communication, it is emphazed that not all exogenous estrogens were claimed to be ssociated with ovarian cancer; only diethylstilbestrol DES) has a relative risk of 30.0. When adjustments re made for the prevalence of oophorectomy in the eneral population, this value does not change. Although nese findings were based on only a few cases, the magniude of the risk plus experiments in dogs warrant etiogical consideration of DES in ovarian cancer in postenopausal women. (5 refs)

8-2516 **Hormone-dependent Mammary Tumors in Strain GR/A Mice. IV. Origin and Progression.** Eng) Aidells, B. D. (Dept. Molecular Biology, Univ. Cali-rnia, Berkeley, CA, 94720); Daniel, C. W. *J Natl Cancer ıst* 60(6): 1345-1350; 1978.

wo types of hormone-dependent mammary tumors that ocir in strain GR/A mice, ie, those that appear during pregancy [pregnancy-dependent mammary tumors (PrDT)] and iose induced by treatment with progesterone (P) and estroen (E) [P + E-dependent mammary tumors (P + E-DT)], ere compared in terms of serial transplantation, response pregnancy, and histology. Both types originated as ductal yperplasias. When the P + E-induced ductal hyperplasias ere transplanted into the parenchyma-free mammary fat ıd, their behavior was similar to that of pregnancy-induced ımors. P + E-induced ductal hyperplasias gave rise to ducl outgrowths in the virgin host and to PrDT in the pregnant ost; this indicated that the ductal hyperplasias are preneoastic lesions in strain GR mice. Because no spontaneous rogression of tumors to a pregnancy-independent state had ccurred after eight to nine serial transplant generations in rDT lines, attempts were made to induce this state by forced reeding. In contrast to nontransplanted PrDT, which usual- require three to five forced breedings of mice to inıce pregnancy independence, six to eight matings were eeded to induce progression to pregnancy indepenence in the transplanted lines. The results suggest that rial transplantation in the cleared fat pad may select r less-neoplastic variants. (20 refs)

8-2517 **Oral Contraceptives and Focal Nodular Hyperplasia of the Liver. Two Case Reports with Electron-Microscopic Studies.** (Eng) Balazs, M. (Dept. Pathology, Janos Hosp., Diosarok u. 1, H-1125 Budapest, Hungary); Gergely, R.; Kostic, S. *Acta Hepatogastroenterol (Stuttg)* 25(2): 111-118; 1978.

Case reports are presented for two women (26 and 36 yr old) who developed focal nodular hyperplasia of the liver and who had been taking oral contraceptives for 2 and 6 yr, respectively. In both cases the tumor was detected during cholecystectomy for cholelithiasis. The patients had no other symptoms. Physical examination did not reveal any pathological changes, and laboratory tests were negative. Biopsy specimens from one of the tumors were examined by electron microscopy. Paracrystalloid inclusions were found in the mitochondria of the tumor cells. The most prominent feature seen by electron microscopy was abnormal vascularization in the tumor tissue. It is hypothesized that abnormal regeneration similar to cirrhosis in the circumscribed areas of the liver was responsible for the disturbed microcirculation. The rich vascularization and the special vascular structure of these tumors may induce severe hemorrhage. Whenever possible, radical removal of the tumor is suggested, and oral contraceptives should be stopped because of the danger of recurrence or malignant transformation of the tumor. (50 refs)

78-2518 **Distribution of Sex Hormones in Patients with Carcinoma of the Prostate.** (Rus) Kislyakova, N. D. (Hormonal Lab., Scientific Res. Inst. Oncology, Rostov, USSR); Luntovskaya, V. A.; Polyanichko, M. F.; Volovelsky, I. Z. *Vopr Onkol* 24(1): 86-89; 1978.

Sex hormone levels were studied in 84 patients with prostatic carcinoma and 24 healthy controls. The patients and controls were divided into three groups: 50-59 yr (15 patients and 10 controls), 60-69 yr (37 and 7, respectively) and 70-80 yr (33 and 7). The daily urinary excretion of testosterone was decreased in patients from the first two age groups (39.15 and 15.28 μg/day, respectively, compared to 60.67 and 20.7 μg/day in controls) and increased in the patients in the third age group (12.61 μg/day, compared to 9.13 μg/day in controls). Urinary excretion of total 17-ketocorticosteroids was decreased only in patients from the 50- to 59-yr age group (7.2 mg, compared to 12.3 in controls). (15 refs)

78-2519 **Benign Hepatic Lesions in Women Taking Oral Contraceptives.** (Eng) Davis, T. J. (Dept. Radiology, Presbyterian Hosp., 8200 Walnut Hill Lane, Dallas, TX 75231); Berk, R. N. *Gastrointest Radiol* 2(3): 213-219; 1977.

The literature on benign hepatic lesions associated with oral contraceptives is reviewed, with emphasis on clinical symptoms and on diagnosis by angiography and the radionuclide scan. In addition, four previously unreported cases are described. Patient 1 was a 26-yr-old woman on Orthonovum 1/80 for 7 yr who on routine examination, was found to have a palpable midepigastric abdominal mass. Histological examination of the resected hepatic tissue revealed focal nodular hyperplasia. The second patient was a 23-yr-old woman also on contraceptive medication for 7 yr (Orthonovum 1/80, 3 yr; Ovulen 21, 6 mo; Demulen, 3 yr) who presented with acute abdominal pain, nausea, and vomiting. A right hepatic lobectomy was performed, and a 6-cm hepatic adenoma with surrounding hematoma was removed. Patient 3, a 41-yr-old woman on Ovulen 21 for 6 yr, also presented with pain. Histological examination of the tissue removed on left hepatic lobectomy revealed a hepatic adenoma with two foci of nodular hyperplasia and a retention cyst in the adjacent tissue. Patient 4, a 38-yr-old woman on Ovulen 21 for 4.5 yr, was found to have a liver nodule when she underwent surgery for a suspected ovarian tumor. The histological diagnosis was focal nodular hyperplasia. All four patients did well postoperatively, and they were followed with radionuclide scans. (55 refs)

78-2520 **Size of Random Samples for Aflatoxin Determination in Peanuts.** (Ger) Waibel, J. (NATEC Gesellschaft für naturwissenschaftlich-technische Dienste mbH, Behringstrasse 154, D-2000 Hamburg 50, W. Germany). *Dtsch Lebensm Rundsch* 73(11): 353-357; 1977.

Analysis of 500 peanut samples for aflatoxin content indicated that for random sampling in factory control, the minimum sample size should be at least 5 kg. For official survey checking, the accumulation of data from each individual producer is adequate. (8 refs.)

78-2521 **Conversion of Aflatoxin B_1 to Aflatoxin D_1 in Ammoniated Peanut and Cottonseed Meals.** (Eng) Lee, L. S. (Southern Regional Res. Center, Agricultural Res. Service, US Dept. Agriculture, New Orleans, LA, 70179); Cucullu, A. F. *J Agric Food Chem* 26(4): 881-884; 1978.

Cultured and spiked peanut and cottonseed meals, high in aflatoxin, were ammoniated under laboratory conditions paralleling those employed for the model ammoniation of pure aflatoxin B_1, as well as under conditions used in commercial detoxification with ammonia. In the model reaction, 10% aflatoxin B_1 remained after ammoniation and 10% was converted to aflatoxin D_1. In all ammoniation studies conducted on spiked and cultured peanut and cottonseed meals, an av of 0.36% aflatoxin B_1 was converted to aflatoxin D_1, and an av of 0.37% of the B_1 compound remained unreacted. (10 refs)

78-2522 **Toxic Responses in Gerbils Treated Topically with Aflatoxin B_1 and Dimethylformamide.** (Eng) Llewellyn, G. C. (Dept. Biology, Virginia Commonwealth Univ., Richmond, VA, 23284). *Bull Environ Contam Toxicol* 19(6): 747-754; 1978.

The effects of topically applied aflatoxin B_1 (AFB_1) and/or dimethylformamide (DMF) were studied in adult male and female Mongolian gerbils. The animals were painted with DMF alone (0.1 ml, 1x/wk or 2x/wk) or AFB_1 dissolved in DMF (500 μg/0.1 ml DMF, 1x/wk or 2x/wk) over a 35-day period. Animals were sacrificed after 174 days, and the liver, kidney, bladder, and treated skin were examined histologically. No pathological responses were detected in any of the organs examined. In the male treatment groups, the control animals showed the highest gain in body wt. The males treated with AFB_1-DMF 2x/wk failed to gain wt initially, showed fluctuations over the duration of the study, but by 120 days had attained a body wt similar to that of the control group. AFB_1 seems to have an effect on membranes and this effect may influence the animals' response to DMF and cause the wt gain differences. It is concluded that gerbils do not absorb appreciable levels of AFB_1 through the skin and, therefore, do not attain the necessary levels of AFB_1 or activated product to result in toxic or carcinogenic effects. (21 refs)

78-2523 **Metabolism and Toxicokinetics of Aflatoxin B_1 in the Monkey, Rat and Mouse: Indications of Species Susceptibility (Meeting Abstract).** (Eng) Wong, Z. A. (Dept. Environmental Toxicology, Univ. California, Davis, CA 95616); Wei, C.; Hsieh, D. P. *Proc Am Assoc Cancer Res* 19: 99; 1978. (no refs)

78-2524 **Modification of Hepatotoxic Effects of Aflatoxin B_1 in Rabbits by Immunization.** (Eng) Ueno, I. (Univ. Tokyo, Tokyo, Japan); Chu, F. S. *Experientia* 15(1): 85-86; 1978.

The usefulness of immunization with bovine serum albumin (BSA)-aflatoxin B_1 conjugate against aflatoxicosis was tested in female albino rabbits. Six weeks after immunization with BSA-aflatoxin B_1 conjugate (210 μg/rabbit), which contained 13 moles of aflatoxin B_1/mole of BSA, immunized and control animals were challenged with a single dose of aflatoxin B_1 at LD_{50} or slightly higher doses. All immunized rabbits were protected from the acute toxic effect, but the expected mortality rate was seen in the unimmunized rabbits. Protection was not absolute, however, since immunized rabbits challenged with twice the LD_{50} dose 17 days after the first challenge showed a greater than 50% mortality. Serum isocitric dehydrogenase activity remained near normal following challenge of the immunized rabbits, but it increased significantly in the control rabbits after they received aflatoxin B_1. Histological examination of the liver of immunized rabbits that survived the challenge showed no abnormality. These results indicate that immunization might be used prophylactically against aflatoxicosis, but it is not known whether imnunization might inhibit the aflatoxin B_1-induced hepatocarcinogenic effect in animals. (11 refs)

8-2525 **Isolation and Characterization of Intact Adult Liver Parenchymal Cells Capable of Metaboism of Promutagens and Procarcinogens (Meeting Abtract).** (Eng) Casciano, D. A. (Div. Mutagenic Res., Natl. Center for Toxicological Res., Jefferson, AR, 72079); Alhaus, Z. R. *Mutat Res* 53(1): 99-100; 1978. (no refs)

8-2526 **Mutagenicity of Plant Flavonols in Microbial Systems (Meeting Abstract).** (Eng) Hardigree, . A. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 7830); Epler, J. L. *Mutat Res* 53(1): 89; 1978. (no refs)

8-2527 **A Scanning Electron Microscopic Study of IAA-induced Tumours in Bean (*Phaseolus vularis* L.) Embryos.** (Eng) Sen, S. (Dept. Biology, Bethune-Cookman Coll., Daytona Beach, FL, 32015); Stevenson, E. . *Cytobios* 18(69): 27-36; 1977.

ndole-3-acetic acid (IAA)-induced tumors in bean (*Phaseous vulgaris*) embryos were studied electron microscopically. The tumors formed between the epicotyl and radicle, and hey produced total rupture and ultimate destruction of the pidermis. The hypocotyl portion showed enlarged, filamenous, thin-walled parenchymatous tissue along the tumor surace, and the filamentous cells showed pustules on the walls. The leaf of the tumorous embryo gradually atrophied with ime, indicating that IAA inhibits epicotyl growth. Furthernore, the stomas demonstrated less crystalline deposition han control embryos. When the IAA was depleted, epicotyl rowth resumed, indicating that the tumors were IAA-ependent. (9 refs)

8-2528 **Mutagenicity of Flavone Derivatives.** (Eng) Sugimura, T. (Natl. Cancer Center Res. Inst., sukiji, Chuo-ku, Tokyo 104, Japan); Nagao, M.; Matsuima, T.; Yahagi, T.; Seino, Y.; Shirai, A.; Sawamura, M.; latori, S.; Yoshihira, K.; Fukuoka, M.; Kuroyanagi, M. *Proc pn Acad [B]* 53(4): 194-197; 1977.

f several flavone derivatives of plants tested on *Salmonella phimurium* for mutagenicity, kaempferol, quercetin, and alangin had high mutagenic activities and fisetin was weakly utagenic. It is suggested that the hydroxyl groups on e phenyl moiety are essential for mutagenic activity nd that the 3,5,7-trihydroxyflavone structure inhibits is activity. (12 refs)

78-2529 **An Analysis of Meiotic Chromosomes of Inbred Male Mice and Their F_1 Sons After Long-Term Treatment of Sires With Triethylenemelamine.** (Eng) Pecevski, J. (Lab. Radiobiology, Boris Kidric Inst. Nuclear Sciences, Belgrade, Yugoslavia); Maric, N.; Savkovic, N.; Radivojevic, D.; Green, S. *Mutat Res* 54(1): 55-60; 1978.

Male C_3H mice were treated with triethylenemelamine (0.02 and 0.04 mg/kg po for 8 wk), to determine the dose-dependent effects of the chemical on fertility and the induction of chromosomal translocations in these mice and their F_1 offspring. Semisterility and sterility plus reciprocal translocations were noted only in the F_1 males, and the changes occurred to a greater degree in the offspring of mice given the higher dose. (10 refs)

78-2530 **Salbutamol: Lack of Evidence of Tumour Induction in Man (Letter to Editor).** (Eng) Poynter, D. (Allen and Hanburys Res. Ltd., Ware, Herts, England); Harris, D. M.; Jack, D. *Br Med J* 1(6104): 46-47; 1978.

Experimental studies to date on the carcinogenicity of salbutamol (SB, or albutamol, a β-adrenoreceptor stimulant) are discussed. Early work with soterenol, another β-adrenoreceptor stimulant, revealed mesovarian leiomyoma formation in Sprague-Dawley rats. Extension of these tests with salbutamol sulfate revealed similar results. When Charles River CD rats were given a max of 50 mg/kg SB, benign leiomyomas resulted. SB was not oncogenic in mice at daily po doses 2,000 times the human daily po intake; it was not mutagenic in vitro at doses 1,000,000 times its effective concentration. Because mesovarian leiomyomas are extremely rare in humans, any increase in their frequency as a result of SB treatment would have been noticed. Higher doses, such as those that cause animal tumors, would produce intolerable cardiovascular effectors and muscle tremor. Since there is little evidence that SB is carcinogenic and it is a selective β_2-adrenergic bronchodilator, there is no rational basis for discontinuing its use. In the US, clinical trials have been halted pending the results of new toxicity data. (2 refs)

78-2531 **Prior Psychiatric Treatment and the Development of Breast Cancer.** (Eng) Overall, J. E. (Univ. Texas Medical Branch, Galveston, TX). *Psycho Pharmacol Bull* 14(1): 19-20; 1978.

The records of 377 women with breast cancer and 236 women with other cancers were studied for prior psychiatric treatment with major neuroleptic drugs, which increase prolactin levels. Prolactin plays a key role in virus- and carcinogen-induced mammary tumors in rodents. Only 1.33% of the breast cancer patients and 1.27% of the other cancer patients had a prior psychiatric diagnosis, and none had a prior diagnosis of schizophrenia. It is concluded that in terms of risk/benefit ratio, the risk of breast cancer should not be a major consideration in the clinical use of psychotherapeutic drugs. (no refs)

78-2532 **Mutagenicity of Aclacinomycin A and Daunomycin Derivatives.** (Eng) Umezawa, K. (Dept. Molecular Oncology, Inst. Medical Science, Univ. Tokyo, Shirokanedai, Minato-ku, Tokyo 108, Japan); Sawamura, M.; Matsushima, T.; Sugimura, T. *Cancer Res* 38(6): 1782-1784; 1978.

The mutagenicity of the antitumor antibiotics aclacinomycin A and daunomycin and some of their derivatives was investigated using *Salmonella typhimurium* strain TA98. Neither aclacinomycin A, N-monodemethylaclacinomycin A, nor N-demethylaclacinomycin A was mutagenic in the absence of metabolic activation by the S-9 mix. In the presence of the mix, only N-demethylaclacinomycin A was mutagenic. 1-Deoxypyrromycin, a hydrolysis product of aclacinomycin A, and N-monodemethyl-1-deoxypyrromycin were not mutagenic in the presence or absence of the mix; N-demethyl-1-deoxypyrromycin was mutagenic, but only with metabolic activation. Daunomycin, N-methyldaunomycin, and N-dimethyldaunomycin were highly mutagenic, weakly mutagenic, and nonmutagenic, respectively, both in the presence and absence of the S-9 mix. Aclavinone and daunomycinone, the aglycones of aclacinomycin A and daunomycin, respectively, were not mutagenic either in the presence or absence of the mix. It is concluded that the amino moiety of anthracycline glycosides is essential for mutagenesis. (11 refs)

78-2533 **Host-mediated Assays and Bone-Marrow Cytogenetics in Rhesus Monkeys (*Macaca mulatta*) with Mitomycin C (Meeting Abstract).** (Eng) Maier, P. (Environmental Mutagenesis Branch, Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC 27709); Huang, S.; Michelmann, H. W.; Ficsor, G.; Malling, H. V.; Feldman, D. B. *Mutat Res* 53(1): 93; 1978. (no refs)

78-2534 **Mutagenicity of the Pyrrolizidine (Senecio) Alkaloid, Lasiocarpine in the Salmonella/Microsome Test (Meeting Abstract).** (Eng) Koletsky, A. (Northwestern Univ. Medical Sch., Chicago, IL, 60611); Oyasu, R.; Reddy, J. K. *Lab Invest* 38(3): 352; 1978. (no refs)

78-2535 **Effects of Cytochalasin B and Colchicine on Phagolysosomal Proteins of Mouse Cells (Meeting Abstract).** (Eng) Evans, R. M. (Univ. Colorado Medical Center, Denver, CO, 80262); Fink, L. M. *Fed Proc* 37(6): 1598; 1978. (no refs)

78-2536 **Experimental Colon Cancer.** (Jpn) Miyano, Y. (First Dept. Surgery, Sch. Medicine, Tottori Univ., Nishi-machi, Yonago, Tottori, 683, Japan); Inoue, J.; Takeda, R.; Inoue, M.; Adachi, H.; Koga, N. *In: Program and Abstracts, 32nd General Meeting of Japan Society of Coloproctology held in Kashihara-shi, Nara Prefecture, Nov 5-6, 1977.* Japan Society of Colon-proctology. (Ota-ku, Tokyo): pp: 21; 1977. (no refs)

78-2537 **Effect of Selenium on Chemically-induced Sister Chromatid Exchanges (Meeting Abstract).** (Eng) Ray, J. H. (Univ. Texas Health Science Center, Medical Genetics Center, Houston, TX, 77030); Altenburg, L. C.; Jacobs, M. M. *Mutat Res* 53(1): 73-74; 1978. (3 refs)

78-2538 **Structure of Mutagen Nucleic Acid Complexes in Solution. Proton Chemical Shifts in 9-Aminoacridine Complexes with dG-dC, dC-dG, and dA-dT-dG-dC-dA-dT.** (Eng) Reuben, J. (Dept. Chemistry, Univ. Houston, Houston, TX, 77004); Baker, B. M.; Kallenbach, N. R. *Biochemistry* 17(14): 2915-2919; 1978.

The influence of self-complementary oligodeoxynucleotides on the chemical shifts of protons of the mutagenic acridine dye 9-aminoacridine was measured. Upfield shifts indicative of intercalative binding were found with dG-dC, dC-dG, and dA-dT-dG-dC-dA-dT, but not with dA-dT. (28 refs)

78-2539 **The Role of DNA Repair in Germ Cell Toxicity (Meeting Abstract).** (Eng) Lee, I. P. (Lab. Environmental Toxicology, NIEHS, Research Triangle Park, NC, 27709); Dixon, R. L. *Environ Health Perspect* 20: 239; 1977. (no refs)

78-2540 **Mutagenicity of Carcinogens as Determined in a Quantitative Mammalian Cell Mutation System, CHO/HGPRT: Study of 80 Agents (Meeting Abstract).** (Eng) Hsie, A. W. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Couch, D. B.; San Sebastian, J. R.; Sun, W. N.; Riddle, J. C.; Brimer, P. A.; Machanoff, R.; Forbes, N. L.; Kenney, F. T.; Hsie, M. H. *Proc Am Assoc Cancer Re* 19: 93; 1978. (no refs)

3-2541 **Structure-Activity Relationships in Chemical Mutagenesis: Preliminary Studies on the Ames' almonella Test Systems (Meeting Abstract).** (Eng) Sigman, . C. (Life Sciences Div., Stanford Res. Inst., Menlo Park, A, 94025); Johnson, H. L.; Cory, M. J. *Mutat Res* 53(1):)-90; 1978. (no refs)

8-2542 **In Vitro DNA Damage/Alkaline Elution Assay for Predicting Carcinogenic Potential (Meeting bstract).** (Eng) Petzold, G. (Upjohn Co., Kalamazoo, MI,)001); Harbach, P.; Bedell, M.; Swenberg, J. A. *Mutat Res* 3(1): 110; 1978. (no refs)

8-2543 **Mutagenicity of Selected Dental Therapeutic Agents Using *Salmonella typhimurium* leeting Abstract).** (Eng) Wheeler, L. (Dental Res. Inst., ch. Medicine & Dentistry, UCLA, Los Angeles, CA); ewman, M.; Rose, K.; Saperstein, M.; Halula, M. *J Dent es* 57(A): 211; 1978. (no refs)

8-2544 **Mutagenicity Testing in Mammalian Cells; the Development of Drug Resistance Markers Havg Practical Application for Screening Potential Mutagens Meeting Abstract).** (Eng) Carver, J. H. (Lawrence Liverore Lab., Livermore, CA, 94550); Wandres, D. L.; Adair, . M.; Branscomb, E. W. *Mutat Res* 53(1): 96; 1978. (no refs)

8-2545 **Mutagenicity Studies on Chemical Carcinogens (Meeting Abstract).** (Eng) Clive, D. (Genetic oxicology Lab., Burroughs Wellcome Co., Research riangle Park, NC). *Mutat Res* 53(1): 96; 1978. (no fs)

8-2546 **Mutagenicity of Fume Particles from Stainless Steel Welding.** (Eng) Hedenstedt, A. (Environ. oxicol. Unit, Univ. Stockholm, Stockholm, Sweden); enssen, D.; Lidesten, B. M.; Ramel, C.; Rannug, U.; tern, R. M. *Scand J Work Environ Health* 3(4): 203-11; 1977.

ume particles collected from different welding procedures vere tested for mutagenicity in *Escherichia coli* (the inhibiion zone in *pol A* was compared with that in *pol A*$^+$) nd in *Salmonella typhimurium* strain TA100. Although 10 mutagenicity was found with mild steel welding, mutagenic effect was established with samples from tainless steel welding. This mutagenicity was particularly associated with manual metal arc (MMA) welding and, to a lesser extent, with metal inert-gas welding. The effect was diminished or eliminated in the presence of a liver microsomal metabolizing system (S-9 mix). More-detailed investigations of the MMA samples showed that the mutagenic agent(s) is water-soluble. An increased mutagenicity associated with the induction of frameshift mutations was found with *S. typhimurium* TA98. The same fume sample was used for a mutagenicity test (resistance to 6-thioguanine) in V79 hamster cells. Because of the high toxicity of these welding fume particles, only very low concentrations could be tested, but the increase in mutations was significantly higher then that in control cells. It is suggested that hexavalent chromium may be involved in the mutagenic effect of the welding fumes. (13 refs)

78-2547 **DNA Targets for Carcinogens Within Living Human Bone Marrow Cells (Meeting Abstract).** (Eng) Frenster, J. H. (Dept. Medicine, Stanford Univ., Stanford, CA); Landrum, S. R.; Masek, M. A.; Nakatsu, S. L. *Clin Res* 26(3): 434A; 1978. (3 refs)

78-2548 **Malignant Neoplasms in Rats Fed Lasiocarpine.** (Eng) Rao, M. S. (Dept. Pathology, Northwestern Univ. Medical Sch., 303 E. Chicago Ave., Chicago, IL, 60611); Reddy, J. K. *Br J Cancer* 37(2): 289-293; 1978.

The effects of chronic dietary administration of lasiocarpine, a pyrrolizidine alkaloid, were studied in 20 male F-344 rats fed 50 ppm for 55 wk. Malignant tumors developed in 17/20 animals between 48 and 59 wk. There were 16 liver tumors, 9 angiosarcomas and 7 hepatocellular carcinomas. Other tumors were a malignant adnexal tumor of the skin (1 rat, that also had an angiosarcoma) and a lymphoma (1 rat). Metastasis to the lungs was observed in four animals with angiosarcomas and one animal with a hepatocellular carcinoma. An angiosarcoma from one animal was successfully transplanted through four generations. Previous studies with repeated ip lasiocarpine injections and with other alkaloids administered by various routes gave rise to other histiogenetic types of liver tumors. These differences illustrate the effect of the dose and mode of administration of various alkaloids on the susceptibility of different cell types in a given target organ. (10 refs)

78-2549 **Carcinogenic and Mutagenic Activities of Milk from Cows Fed Bracken Fern (*Pteridium aquilinum*).** (Eng) Pamukcu, A. M. (Div. Clinical Oncology, Dept. Human Oncology, Univ. Wisconsin Center Health Sciences, 1300 University Ave., Madison, WI, 53706); Erturk, E.; Yalciner, S.; Milli, U.; Bryan, G. T. *Cancer Res* 38(6): 1556-1560; 1978.

The carcinogenicity, toxicity, and mutagenicity of the milk of Brown Swiss cows fed bracken fern (BF) and hay grain (HG: normal diet) were determined. The BF-fed cows were fed 1 g/kg/day for a total biennial BF intake/cow of 218-252 kg. The milk was fed to male and female Norwegian strain rats as either fresh or freeze-dried powdered milk mixed with grain. Of the rats fed milk from BF-fed cows, 9/34 fed whole milk and 11/56 fed powdered milk developed small intestinal, kidney, or urinary bladder carcinomas within 117 wk, vs 0/70 rats fed either whole or powdered milk from cows receiving a HG diet and 9/20 rats fed a basic grain diet. The pentane, methanol, benzene, diethyl ether, ethyl acetate and residue fractions of milk from BF- and HG-fed cows were prepared. The diethyl ether and ethyl acetate fractions displayed the highest murine acute toxicity. The murine bladder carcinogenicity of prepared fractions was assessed by pellet implantation (20 mg containing 1:3 wt/wt concentrations of each fraction). Diethyl ether fractions from BF-fed cows induced bladder carcinomas in 10/24 mice, compared with 3/19 mice fed the same fraction from HG-fed cows. The pentane, methanol, chloroform, and chloroform:methanol fractions of milk from BF- and HG-fed cows were tested for mutagenicity in *Salmonella typhimurium* strains TA98 and TA100. Only the chloroform:methanol fraction from BF-fed cows was mutagenic, and then only with TA100. Cystoscopic examination of the BF-fed cows revealed urinary bladder papillomas in all animals (4); no changes were noted in cows fed HG. The significance of these results in terms of the human risk of gastointestinal tract or urinary bladder cancer following ingestion of milk from BF-fed cows is not known. (18 refs)

78-2550 **Carcinogenesis Studies in the Western New York Great Lakes Aquatic Environment (Meeting Abstract).** (Eng) Black, J. J. (Roswell Park Memorial Inst., Buffalo, NY, 14263); Holmes, M.; Wenner, C.; Zapisek, W. *Proc Am Assoc Cancer Res* 19: 60; 1978. (no refs)

78-2551 **Development of a Technique for the Rapid Screening of Chemicals for Carcinogenicity (Meeting Abstract).** (Eng) Cobon, A. M. (Dept. Biology as Applied to Medicine, Middlesex Hosp. Medical Sch., Cleveland St., London W1P 6DB, England); Lilly, L. J.; Newton, M. F. *Br J Pharmacol* 62(3): 455P-456P; 1978. (4 refs)

78-2552 **A Convenient Thin Layer Chromatographic Method for the Detection of Cyclochlorotine and Simatoxin, Toxins from *Penicillium islandicum* Sopp.** (Eng) Ghosh, A. C. (SISA Inc., Cambridge, MA, 02138); Manmade, A.; Bousquet, A.; Townsend, J. M.; Demain, A. L. *Experientia* 34(6): 819-820; 1978.

A rapid thin-layer chromatographic method for the detection of cyclochlorotine, a carcinogen produced by the storage mold *Penicillium islandicum* Sopp, is based on the color reaction of the peptide with chlorine o-tolidine. The method also led to the detection of a new toxic metabolite, simatoxin. (18 refs)

78-2553 **Low Dose Extrapolation in Carcinogenesis (Meeting Abstract).** (Eng) Hoel, D. (Biometry Branch, NIEHS, Research Triangle Park, NC, 27709); Guess, H. *Environ Health Perspect* 20: 249; 1977. (no refs)

78-2554 **Evidence for In Situ Environmental Mutagenesis and Other Toxic Effects from a Lead Smelting Operation (Meeting Abstract).** (Eng) Lower, W. (Environmental Trace Substances Res. Center, Univ. Missouri, Columbia, MO, 65201); Plewa, M. *Mutat Res* 53(1): 86-87; 1978. (no refs)

78-2555 **Spin Trapping in Biological Systems. Free Radicals Produced by Nitrosamine Carcinogens upon Interaction with Rat Liver Microsomes and Nuclei and During Oxidation of 6-Hydroxy-Dopamine (Meeting Abstract).** (Eng) Floyd, R. A. (Oklahoma Medical Res. Foundation, Oklahoma City, OK, 73104). *Fed Proc* 37(6): 1725; 1978. (no refs)

78-2556 **Mutagenicity of Nitrosopiperidines in Yeast (Meeting Abstract).** (Eng) Larimer, F. W. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Ramey, D. W.; Epler, J. L. *Mutat Res* 53(1): 86; 1978. (no refs)

78-2557 **Pathogenesis of Pancreatic Carcinoma in Hamsters Induced by N-Nitro Bis(2-oxopropyl)amine (BOP) (Meeting Abstract).** (Eng) Scarpelli, D. G. (Northwestern Univ., Chicago, IL, 60611); Rao, M. S. *Fed Proc* 37(3): 232; 1978. (no refs)

78-2558 **Study on Cytological Effects of Carofur, a New Mutagen (Meeting Abstract).** (Eng) Vig, B. K. (Dept. Biology, Univ. Nevada, Reno, NV, 89557); Natarajan, A. T.; Zimmermann, F. K. *Mutat Res* 53(1): 79-80; 1978. (no refs)

3-2559 **Deuterium Isotope Effects in Nitrosamine Carcinogenesis (Meeting Abstract).** (Eng) Lijinsky, /. (Chemical Carcinogenesis Program, NCI Frederick Can- er Res. Center, Frederick, MD, 21501). *Proc Am Assoc Can- er Res* 19: 213; 1978. (no refs)

8-2560 **Occurrence of Methylguanidine and Agmatine, Nitrosatable Guanidino Compounds, in Foods.** Eng) Kawabata, T. (Dept. Biomedical Res. on Food, NIH, hinagawa-ku, Tokyo 141, Japan); Ohshima, H.; Ino, M. *J gric Food Chem* 26(2): 334-338; 1978.

sing a recently developed method for the determination and lentification of some basic guanidino compounds in foods, e nitrosated methylguanidine (MG) and nitrosated agma- ne (AG) contents of various fresh and processed foods were etermined. The results were confirmed by gas chromatogra- hy-mass spectrometry. The MG content of fresh foods inged from not detectable (ND) in most foods to 2.5 mg/kg chicken. It appears unlikely that MG occurs naturally in esh foods in the high concentrations reported previously. xcept for smoked-dried fish products, none of the processed ods tested contained more than trace amounts of MG, the noked-dried fish products contained from 18.1 to 178 g/kg MG, although it seems unlikely that these pro- ucts are responsible for the high incidence of gastric ancer in Japan. The AG levels in fresh foods ranged om ND to 18 mg/kg in most cases, but they were onsiderably higher (up to 224 mg/kg) in abalone and op-shell muscle. Most processed foods also contained nly small amounts of AG, although some salted- ried seafood products contained levels up to 650 g/kg. In the processed foods, there was no sign of deteriora- on with high AG concentrations. (16 refs)

8-2561 **Inhibition of Nitroso Chemical Carcinogen Activation of Rat Liver Guanylate Cyclase Activi- / by Antitumor Agents (Meeting Abstract).** (Eng) Vesely, . L. (Dept. Medicine, Univ. Miami Sch. Medicine, Miami, L); Levey, G. S. *Clin Res* 26(3): 537A; 1978. (no refs)

3-2562 **Experimental Cancer of the Stomach Induced in Rats with N-Methyl-N′-nitro-N-ni- osoguanidine.** (Jpn) Nishizato, Y. (Dept. Internal Medi- ne, Tokyo Medical Coll., Shinjuku-ku, Tokyo 160, Japan). *okyo Ika Daigaku Zasshi* 35(5): 759-770; 1977.

umor development was studied in Wistar rats fed N-meth- -N′-nitro-N-nitrosoguanidine (167 μg/ml) continuously in e diet. The majority of the gastric tumors developed in the sser curvature of the glandular stomach, and many were evated. Histologically, most tumors were adenocarcinomas that invaded as deep as the serosa. There did not appear to be any correlation between tumor formation and degree of intestinal epithelial metaplasia. In some animals, the right gastric artery, the gastroduodenal artery, and the gastroepiploic artery were ligated before carcinogen administration. Animals thus treated had striking erosions and proliferation of the pit epithelium and prominent upward and downward adenomatous hyperplasia. (27 refs)

78-2563 **Chemically Induced Gastric Carcinogenesis of the Rat after Vagotomy and Resection.** (Ger) Rumpf, P. (Chirurgische Universitatsklinik A, Moorenstrasse 5, D-4000 Dusseldorf, W. Germany); Schacht, U.; Palomba, P.; Kremer, K.; Schmitz, H.; Borchard, F. *Z Gastroenterol* 16(2): 85-94; 1978.

The effects of truncal vagotomy with pyloroplasty and Billroth II (B-II) gastric resection without Braun's anastomosis on the carcinogenic effect of N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) were studied in inbred male Wistar rats. Six-week-old animals received drinking water containing 30 or 100 mg/liter MNNG ad libitum for 480 days, and they were then sacrificed. Vagotomy or gastric resection was performed after the first 7 wk of treatment. The controls received MNNG only. The first carcinoma was discovered on day 392 in a control rat treated with 100 mg/liter. Malignant gastric tumors (mainly tubular and papillary adenocarcinomas plus 2 carcinosarcomas and 2 signet ring cell carcinomas) were found in 6/40 control rats treated with 30 mg/liter, in 21/40 controls treated with 100 mg/liter, in 2/22 vagotomized rats treated with 30 mg/liter, in 11/21 vagotomized rats treated with 100 mg/liter, in 8/16 B-II rats treated with 30 mg/liter, and in 15/18 B-II rats treated with 100 mg/liter. The findings indicate that the incidence of gastric carcinoma was significantly higher in B-II rats than it was in the controls, but vagotomy did not increase the incidence. (17 refs)

78-2564 **In Vitro Studies of Chemical Carcinogenesis in the Rat Colon (Meeting Abstract).** (Eng) Shamsuddin, A. K. (Dept. Pathology, Univ. Maryland Sch. Medicine, Baltimore, MD, 21201); Barrett, L. A.; Autrup, H.; Harris, C. C.; Trump, B. F. *Lab Invest* 38(3): 364-365; 1978. (no refs)

78-2565 **Soluble Adenylate Cyclase and Guanylate Cyclase in Bovine Adrenal Cortex: Activation by Sodium Nitroprusside and Nitrosamines - Inhibition by Phospholipase A and Free Fatty Acids (Meeting Abstract).** (Eng) Struck, C. J. (Pharmakologisches Institut, Justus-Liebig-Universitat Giessen, Frankfurter Strasse 107, D-6300

Lahn-Giessen 1, W. Germany); Glossmann, H. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R25; 1978. (no refs)

78-2566 **Cytologic Studies of Cultured Pancreatic Ducts (Meeting Abstract).** (Eng) Resau, J. H. (Dept. Pathology, Univ. Maryland Sch. Medicine, Baltimore, MD, 21201); Jones, R. T. *Lab Invest* 38(3): 362; 1978. (1 ref)

78-2567 **Direct and Transplacental Chemical Mutagenesis in Syrian Hamster Embryo Cultures (Meeting Abstract).** (Eng) Jonmaire, P. W. (Kettering Lab., Univ. Cincinnati, Cincinnati, OH, 45267); Soukup, S.; Christian, R. T. *In Vitro* 14(4): 382; 1978. (no refs)

78-2568 **Phenotypic Lag and Mutation to 6-Thioguanine Resistance in Diploid Human Lymphoblasts.** (Eng) Thilly, W. G. (Toxicology Group, Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Room E18-666, Cambridge, MA, 02139); Deluca, J. G.; Hoppe, H.; Penman, B. W. *Mutat Res* 50(1): 137-144; 1978.

A mutation assay at the hypoxanthine-guanine phosphoribosyltransferase (HGPRT) locus, expressed as 6-thioguanine resistance (6TG-r), was used to determine the time required for expression of the mutant phenotype after treatment of diploid human lymphoblasts with various mutagens. 6TG mutants appeared 7 and 13 generations after exposure of the cells for 24 hr to 37.5 and 50 μM methylnitrosourea (MNU), respectively; 6, 7, and 9 generations after exposure to 0.075, 0.15, and 0.225 μM N-methyl-N′-nitro-N-nitrosoguanidine (MNNG); 15 generations after exposure to 50 μM 5-bromodeoxyuridine; and 16, 16, and 15 generations after exposure to 0.625, 1.25, and 2.50 μM ICR-191, a substituted, acridine half-mustard. For MNU and MNNG, the length of the phenotypic lag appeared to be concentration-dependent. It is hypothesized that expression of 6TG-r may require the removal of essentially all preexisting HGPRT molecules via division, dilution, and protein turnover. Each potential mammalian cell mutation assay should be characterized carefully with regard to the length and possible variability of phenotypic lag. (30 refs)

78-2569 **Suppression of In Vitro Chemically Induced** Morphological Transformation by 5′-Bromo-2′-deoxyuridine (BUDR)-Photolysis (Meeting Abstract). (Eng) Mironescu, S. G. (Dept. Pathology, Univ. South Carolina, Columbia, SC, 29208). *Proc Am Assoc Cancer Res* 19: 59; 1978. (no refs)

78-2570 **Conditions for the Selection of Azaguanine- or Thioguanine-resistant Human Diploid Fibroblasts (Meeting Abstract).** (Eng) Jacobs, L. (Univ. Wisconsin, Madison, WI, 53706); DeMars, R. *Mutat Res* 53(1): 97-98; 1978. (no refs)

78-2571 **The Effect of Iodoacetamide-induced Fundic Ulcers on Gastric Carcinogenesis Produced by N-Methyl-N′-nitro-N-nitrosoguanidine in Rats.** (Eng) Majima, S. (First Dept. Surgery, Kyoto Prefectural Univ. Medicine, Kyoto 602, Japan); Fukushima, M.; Fujita, Y.; Ouchi, T. *Tohoku J Exp Med* 123(4): 381-388; 1978.

The effect of iodoacetamide (IAM)-induced ulcers on the subsequent development of gastric carcinoma produced by N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) was examined in male Wistar rats. IAM (0.1% soln) was given in the drinking water for 8 wk. MNNG (50 or 100 mg/liter in the drinking water) was given from weeks 6 to 26. Two additional groups of rats were given IAM or MNNG alone. Fifty-six of the 62 ulcers induced by IAM (alone or with MNNG) were located in the fundic gland area along the limiting ridge. There were no ulcers in the rats treated with MNNG alone. The incidence of fundic carcinoma was 16% in the groups treated with IAM + MNNG, 0% in the group treated with MNNG alone. The incidence of pyloric gland carcinoma was 18% in the groups treated with IAM + MNNG, 54% in the group treated with MNNG. No tumors were found in the group treated with IAM alone. These results indicate that if an ulcer is present, carcinoma develops even in the fundic mucosa, which is usually resistant to the carcinogenic stimulation of MNNG. It is concluded that gastric ulcer predisposes to the development of gastric carcinoma. (14 refs)

78-2572 **Responses of the Hepatic Guanylate Cyclase System to Nitrosoguanidine (NG) After Partial Hepatectomy (Meeting Abstract).** (Eng) Briggs, R. G. (Dept. Medicine, Veterans Admin. Hosp., Pittsburgh, PA, 15240); DeRubertis, F. R. *Fed Proc* 37(6): 1689; 1978. (no refs)

78-2573 **The Effects of Dimethylhydrazine and Small Bowel Resection on Crypt Cell Proliferation in the Rat Colon (Meeting Abstract).** (Eng) Tilson, M. D. (Dept. Medicine, Yale Univ. Sch. Medicine, New Haven, CT); Buck, M.; Livstone, E. M. *Gastroenterology* 74(5, part 2): 1105; 1978. (no refs)

78-2574 **Metal Ion Effects in Microbial Screening Systems (Meeting Abstract).** (Eng) Tindall, K. R.

Montana State Univ., Bozeman, MT, 59715); Warren, G. R.; Skaar, P. D. *Mutat Res* 53(1): 90-91; 1978. (no refs)

78-2575 **The Effects of Urea and Cell Surface Protein on a Normal and a Transformed Cell Line Meeting Abstract).** (Eng) Phelps, A. W. (Univ. Tennessee, Memphis, TN, 38163); Dillingham, E. O.; Autian, J. *In Vitro* 4(4): 335-336; 1978. (no refs)

8-2576 **Chemical Induction of Unscheduled DNA Synthesis in Human Skin Epithelial Cell Cultures.** Eng) Lake, R. S. (Dept. Pathology, Children's Hosp. Medical Center, Akron, OH, 44308); Kropko, M. L.; Pezzutti, M. R.; Shoemaker, R. H; Igel, H. J. *Cancer Res* 38(7): 2091-2098; 1978.

A semiquantitative in vitro method for measuring chemical nduction of unscheduled DNA synthesis (UDS) was deveoped to evaluate the range of chemical sensitivity and DNA epair response of human skin epithelial cells. Cryopreserved oreskin epithelial cells were grown from explants and replanted in replicate culture wells. The cultures were then reated for 3 days in arginine-deficient medium and further nhibited in S-phase DNA synthesis by a 2-hr hydroxyurea reatment (10 μM). Carcinogen and ^{3}H-labeled thymidine were added simultaneously, and UDS was accumulated over a 24-hr incubation period determined by direct scintillation counting of acid-precipitable whole-cell radioactivity. All ultimate and proximate carcinogens elicited a doserelated UDS response, as did several classes of procarcinogens. Upon serial subculture of organotypic primary skin cultures, the UDS response induced by the procarcinogen 3-methylcholanthrene decreased in parallel with a decreased ability of cells to metabolize polycyclic hydrocarbons to water-soluble metabolites. Arginine deprivation and hydroxyurea did not impair the ability of epithelial cells to metabolize procarcinogens to DNA-damaging metabolites. It is hypothesized that those procarcinogens that are efficiently metabolized by skin will be detected with UDS as an end point. Similar UDS measurements in a panel of organotypic epithelial cell cultures may provide a relevant technique for detecting specific human carcinogens. (41 refs)

78-2577 **Distribution of O^6-Alkylguanine in Rat DNA Sequences (Unique and Repetitive) and in a Selected Proliferative Cell Pool Following Inoculation of Methyl- and Ethylnitrosourea (MNU and ENU) (Meeting Abstract).** (Eng) Chang, M. J. (Ohio State Univ., Columbus, OH, 43210); Webb, T. E. *Proc Am Assoc Cancer Res* 19: 177; 1978. (no refs)

78-2578 **Relative Carcinogenic Effectiveness of N-Methyl-N-nitrosourea (MNUA): and N-Ethyl-N-nitrosourea (ENUA): Quantitative Relation to Alkylation of O-6 of Guanine in DNA (Meeting Abstract).** (Eng) Frei, J. V. (Chester Beatty Res. Inst., Chalfont St. Giles, England); Lawley, P. D.; Swenson, D. H.; Warren, W. *Proc Am Assoc Cancer Res* 19: 169; 1978. (no refs)

78-2579 **Tumor Induction in the Trachea of Hamsters with N-Nitroso-N-methylurea.** (Eng) Yarita, T. (Univ. Tennessee-Oak Ridge Graduate Sch. Biomedical Sciences and Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Nettesheim, P.; Williams, M. L. *Cancer Res* 38(6): 1667-1676; 1978.

The tracheal epithelium of male Syrian golden hamsters was exposed to a 1% soln of N-nitroso-N-methylurea (MNU) once or twice weekly for a period of 5-20 wk (10-30 exposures), and tumor incidence was determined. Carcinomas, all types, were noted in 0/12, 5/12, 10/15, 15/17, and 16/17 hamsters given 10, 15, 20, 25, and 30 twice-weekly exposures, respectively. With 10 exposures, 2/12 hamsters developed benign tracheal tumors. Mean tumor induction time decreased when the frequency of exposure was increased from 50 wk with 10-15 exposures to 28 wk with 25-30 exposures. Tumors were also noted in 20/22 hamsters receiving 20 once weekly exposures; the mean tumor induction time was 35 wk. The major histological types of invasive carcinomas were epidermoid carcinomas (54%), anaplastic large cell and small cell carcinomas (26%), adenocarcinomas (13%), and combined epidermoid-adenocarcinomas (7%). In animals receiving 10-20 twice-weekly exposures, only metaplastic lesions with varying degrees of cellular atypia were noted at the time of the last exposure. (9 refs)

78-2580 **A Comparative Study of the Effects of Various Transplacental Carcinogens in Rabbits.** (Rus) Beniashvili, D. S. (Res. Inst. Oncology, GSSR Ministry Health, Tbilisi, USSR). *Vopr Onkol* 24(3): 77-83; 1978.

Transplacental carcinogenesis was studied in 60 pregnant rabbits. The rabbits were divided into five groups and, 25-26 days prior to parturition, Groups 1-4 were inoculated with ethylnitrosourea (80 mg/kg), methylnitrosourea (40 mg/kg), 7,12-dimethylbenz(a)anthracene (20 mg/kg), and benzo(a)pyrene (30 mg/kg), respectively; Group 5 rabbits served as untreated controls. The offspring of the exposed does were followed for 4 yr: 34/183 rabbits developed 38 tumors (16 tumors were located in the peripheral nervous system, and 12 tumors were located in the kidneys). The number of rabbits developing tumors in the five groups was 16/36, 6/38, 7/35, 5/36, and 0/38, respectively. The av latent period was 436 days in Group 1, 622 days in Group 2, 596 days in Group 3, and 635 days in Group 4. The offspring of the Group 1 does had 8 neurogenic tumors, compared to 1 in Group 2, 4 in Group 3, and 3 in Group 4. (26 refs)

78-2581 **Neoplasms in Purebred Boxer Dogs Following Long-Term Administration of N-Methyl-N-nitrosourea.** (Eng) Denlinger, R. H. (Pathology and Toxicology Res. Unit, Upjohn Co., Kalamazoo, MI, 49001); Koestner, A.; Swenberg, J. A. *Cancer Res* 38(6): 1711-1717; 1978.

The carcinogenicity of N-methyl-N-nitrosourea (MNU) was studied in purebred Boxer dogs, a breed with a high incidence of spontaneous tumors (including brain tumors). Twenty dogs were given weekly iv injections of 5 mg/kg MNU for 36 wk; they were observed for approx 3 yr. Nineteen dogs developed tumors 19-36.5 mo after the first injection; the mean latent period was 30 mo. All tumor-bearing dogs had two or more neoplasms, including metastases, and 14 dogs had tumors of more than one tumor type. Malignant neurinomas of the small intestine were present in 11 dogs, poorly differentiated intestinal sarcomas in 6. Metastasis occurred to the liver, spleen, lymph nodes, lung, and adrenal glands. Two cases of malignant neurinoma developed in the heart, and one each developed in the stomach and colon. Two well-differentiated neurinomas occurred in the small intestine, and one each occurred in a cervical (C_8) spinal root, the stomach, and the colon. Hemangiosarcomas were noted in the spleen of 3 dogs, the heart of 2, a spinal root [the base of the dorsal arch of vertebra T_4 (T_4)] of 1, and a lumbar mass of 1. Three developed hemangiomas of the skin. Malignant. lymphoma developed in the liver of 2, the spleen of 2, and the orbit of 1. One each developed a carcinoma of the intestine and lung; one dog developed a fibrosarcoma of the femur. Because of the high incidence rate, young age at development, and unique tumor types, it is believed that these tumors, especially the neurinomas, resulted from MNU exposure. Although the lack of brain involvement was unexplained, MNU had a neurooncogenic effect on the peripheral nerves of the gastrointestinal tract. (19 refs)

78-2582 **The Stability of Methyl and Ethyl Phosphotriesters in DNA In Vivo.** (Eng) Shooter, K. V. (Chemical Carcinogenesis Div., Inst. Cancer Res., Royal Cancer Hosp., Pollards Wood Res. Station, Nightingales Lane, Chalfont St. Giles, Bucks HP8 4SP, England); Slade, T. A. *Chem Biol Interact* 19(3): 353-361; 1977.

Studies were conducted to determine whether a phosphotriester in DNA is a lesion that is excised and repaired by cellular enzymes in vivo. C57BL male mice were inoculated ip with N-methyl-N-nitrosourea (MNU) or N-ethyl-N-nitrosourea (ENU), and the concentration of alkyl phosphotriesters in the DNA of lung, liver, brain, kidney, spleen, and thymus was determined from the extent of degradation induced in isolated DNA by alkali. The same total dose (2 mg MNU or 4 mg ENU) was given either as a single injection or as weekly injections carried out over 5-20 wk. Methyl phosphotriesters induced in liver, lung, and kidney by the single injection were lost, with a half-life (t1/2) of 7 days; in brain, the t1/2 was 2-3 days. During the multiple MNU injections, t1/2 was 16 days. Ethyl phosphotriesters formed in the DNA of lung, liver, kidney, and brain were much more stable, t1/2 being 10-15 wk. Phosphotriesters formed in the DNA of spleen and thymus disappeared very quickly after the single injection; there was no accumulation of phosphotriesters in the DNA of these tissues during the multiple injections. The relative stability of methyl and ethyl phosphotriesters suggests that these lesions are not excised enzymatically, since it is unlikely that the repair of methylated but not ethylated lesions would occur. The results suggest that phosphotriesters are not lesions that are eliminated by cellular repair enzymes. (23 refs)

78-2583 **Effect of Functional Disturbances of the Thyroid on the Development of Experimental Tumors of Peripheral Nervous System.** (Rus) Beniashvili, D. S. (Scientific Res. Inst. Oncology, Tbilisi, USSR). *Soobshch Akad Nauk Gruz SSR* 87(1): 189-192; 1977.

The role of functional disturbances of the thyroid in the genesis of methylnitrosourea (MNU)-induced tumors of the peripheral nervous system was studied in albino random bred rats and rabbits. All animals received weekly iv injections with NMU (10 mg/kg). Three series of experiments were carried out. Animals in group 1 were given thyroidin (250 mg/kg, daily, for 1.5 yr), animals in group 2 received the antithyroidin drug mercazolyl (5 mg/kg, po, daily, for 18 mo) and animals in group 3 received NMU alone (controls). The incidence of neurogenic tumors was significantly decreased in the animals with hyperfunction of the thyroid: 6/41 rats and 1/12 rabbits in group 1 developed tumors, compared to 23/48 and 7/14, respectively, in group 2, and 15/48 and 3/16, respectively, in group 3. An av latent period of tumor development in groups 1, 2 and 3 was 359.5, 172.1 and 323.4 days, respectively. (12 refs.)

78-2584 **A Study of Chemical Carcinogenesis. 4. Synthesis of the Carbon-14 Labelled Carcinogens 5-Methylchrysene, 2-Methylaniline and 3-Methyl-2-naphthylamine.** (Eng) Weiss, L. (Div. Environmental Carcinogenesis, Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY, 10595); Loy, M.; Hecht, S. S.; Hoffmann, D. *J Labelled Compd Radiopharm* 14(1): 119-131; 1978.

Experimental procedures for synthesizing the ^{14}C-labeled forms of the carcinogens 5-methylchrysene, 2-methyl-iline, and 3-methyl-2-naphthylamine are presented. $^{14}CO_3$ was used to introduce the label. The produres can be used to prepare these compounds in fficient yields for metabolism studies. (26 refs)

-2585 **Tumor-initiating Activity of Fluorinated 5-Methylchrysenes.** (Eng) Hecht, S. S. (Naylor Dana st. Disease Prevention, American Health Foundation, Vallla, NY, 10595); Hirota, N.; Loy, M.; Hoffmann, D. *Canr Res* 38(6): 1694-1698; 1978.

series of fluorinated 5-methylchrysene derivatives was preured and tested for tumor-initiating activity on Ha/ICR male outbred Swiss albino mouse skin. The compounds sted were 1-fluoro-5-methylchrysene, 3-fluoro -5-ethylchrysene, 6-fluoro-5-methylchrysene, 7-fluoro-methylchrysene, 9-fluoro-5-methylchrysene, 11-fluoro-methylchrysene, 12-fluoro-5-methylchrysene, 5,12-methylchrysene, and 12-methoxy-5-methylchrysene. ich compound was assayed at total initiating doses 30 or 100 μg, with promotion thrice weekly by 5 μg of tetradecanoylphorbol acetate. Of the comunds tested, 12-fluoro-5-methylchrysene, 1-fluoro-methylchrysene, 3-fluoro -5- methylchrysene, 5,12-methylchrysene, and 12-methoxy -5- methylchrysene ere less active than 5-methylchrysene, and 6-fluoro-5-meylchrysene, 7-fluoro-5-methylchrysene, 9-fluoro-5-meylchrysene, and 11-fluoro-5-methylchrysene were as otent as 5-methylchrysene. Thus, positions 12, 1, d 3 of 5-methylchrysene are involved in metabolic tivation to the ultimate carcinogen. In this series, e requirements favoring carcinogenicity were a baygion methyl group and an unhindered *peri* position ljacent to an unsubstituted angular ring. (23 refs)

8-2586 **Evidence for Bay-Region Activation of Chrysene 1,2-Dihydrodiol to an Ultimate Carcinoen.** (Eng) Levin, W. (Dept. Biochemistry and Drug Metabosm, Hoffmann-LaRoche Inc., Nutley, NJ, 07110); Wood, . W.; Chang, R. L.; Yagi, H.; Mah, H. D.; Jerina, D. M.; onney, A. H. *Cancer Res* 38(6): 1832-1835; 1978.

n attempt was made to identify the ultimate carcinogenic etabolite of chrysene by testing the compound and its three etabolically possible *trans*-dihydrodiols (at the ,2-, 3,4-, and 5,6-positions) for tumorigenicity in emale CD-1 mice. A single topical application of 0.4, .25, or 4.0 micromoles of each compound was followd 7 days later by twice-weekly applications of 12-O-etradecanoylphorbol-13-acetate for 25 wk. Chrysene ,2-dihydrodiol had approx twice the tumorigenic ctivity of chrysene at all doses tested. Chrysene ,4-dihydrodiol and chrysene 5,6-dihydrodiol had no signifiant tumorigenic activity. 1,2-Dihydroxy-1,2,3,4-tetrahydrochrysene, a compound related to chrysene 1,2-dihydrodiol but without the conjugated nonaromatic double bond at the 3,4-position, had < 25% of its tumorigenic activity. These findings indicate that chrysene 1,2-dihydrodiol is a proximate carcinogenic metabolite of chrysene and that a chrysene 1,2-diol-3,4-epoxide, in which the epoxide group forms part of the bay region in the molecule, is a likely candidate for the ultimate carcinogenic metabolite of chrysene. (24 refs)

78-2587 **Investigations on the Carcinogenic Burden by Air Pollution in Man XVII. Synthesis of the Carcinogen Cyclopenteno(cd)pyrene.** (Eng) Jacob, J. (Biochemisches Institut fur Umweltcarcinogene, Sieker Landstrasse 19, D-2070 Arhrensburg, W. Germany); Grimmer, G. *Zentralbl Bakteriol [Orig B]* 165(3/4): 305-308; 1977.

An experimental procedure for synthesizing the carcinogen cyclopenteno(cd)pyrene (CPP) is described in which acetylpyrene is converted to 1-pyrenylethanol, which in turn is converted to CPP. Previous studies have shown that when CPP is injected sc in mice, it has half the activity of benzo(a)pyrene. (9 refs)

78-2588 **The Inability of Aminoazotoluene to Induce Somatic Crossing-Over or Mutation in *Glycine max* (L.) Merrill.** (Eng) Arenaz, P. (Dept. Biology, Univ. Nevada, Reno, NV, 89557). *Mutat Res* 56(3): 295-298; 1978.

The mutagenicity of aminoazotoluene (AAT) was examined in seeds from soybean (*Glycine max*) varieties T219 and L65-1237, which are heterozygous for the gene combination $Y_{11}y_{11}$ controlling chlorophyll development. The seeds were soaked for varying periods of time in 6, 12.5, 25, or 50 ppm AAT. Leaf analysis indicated that the drug did not increase the frequency of mutation or somatic crossing-over in this system. (10 refs)

78-2589 **o-Aminoazotoluene-induced Tumors in Interscapular Fat in Mice.** (Rus) Kaledin, V. I. (Lab. Cancer Genetics, Inst. Cytology and Genetics, Novosibirsk, USSR); Alekseeva, G. V. *Vopr Onkol* 24(1): 95-98; 1978.

The exposure of A/He and CC57BR mice to o-aminoazotoluene (OAT: 10 mg sc, 1x/mo, 8 doses) induced tumors in the interscapular fat of 49/60 CC57-BR mice. The first tumor appeared 260 days after the initiation of carcinogen administration. Histologically, the tumors were diagnosed as hyperplastic capillary hemangiomas. In addition, 6 mice developed tumors in the femur, 2 in the mesenteric fat, and 1 in the liver (all were typical hemangioendotheliomas). OAT did not induce interscapular fat tumors in A/He, but it did result in the development of liver tumors: 52/60 A/He mice died of liver tumors within 435 days of the start of the experiment. (4 refs)

78-2590 **Murine Liver Tumors Induced by Prenatal and Postnatal Administration of o-Aminoazotoluene.** (Rus) Kolesnichenko, T. S. (Dept. Carcinogenic Agents, Cancer Res. Center, Moscow, USSR); Popova, N. V.; Shabad, L. M. *Biull Eksp Biol Med* 85(2): 199-202; 1978.

The hepatocarcinogenic effect of o-aminoazotoluene (AAT) was studied in the embryos and mature animals of CBA strain mice. Three-mo-old mice or pregnant mice (4-5 days before delivery) were given AAT (12 mg/animal, intragastrically); 12 mo later, the exposed mice and their offspring were sacrificed. Postnatal administration of AAT resulted in liver tumors in 78.7% of the males (compared with 68.5% in controls; CBA males have high frequency of spontaneous liver tumors) and in 89.3% of the females (compared with 11.5% in controls). Administration of AAT to pregnant mice induced liver tumors in 81.8% of their male offspring (35.2% in controls) and in 54.8% of their female offspring (5.3% in controls). Prenatal administration of AAT increased the frequency of malignant liver tumors in males 94.3% vs 57.9% in postnatally exposed males. (11 refs)

78-2591 **Studies on the Cytochrome P-450-Product Complexes Formed During the Metabolism of NN-Dimethylaniline (Meeting Abstract).** (Eng) Hlavica, P. (Pharmakologisches Institut, Universitat Munchen, Nussbaumstrasse 26, D-8000 Munich 2, W. Germany); Aichinger, G. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R8; 1978. (2 refs)

78-2592 **Carcinogen Induction of Cytochrome-P448-Associated Enzymes: Detection by In Vivo Breath Analysis (Meeting Abstract).** (Eng) Hepner, G. W. (Dept. Medicine, Univ. California Los Angeles/Harbor General Hosp., Torrance, CA); Piken, E. *Clin Res* 26(3): 498A; 1978. (no refs)

78-2593 **Azocarcinogen, Nuclear Ribonucleoprotein Particles and the Transport of Nuclear RNA into the Cytoplasm (Meeting Abstract).** (Eng) Patel, N. T. (Univ. Texas Medical Branch, Galveston, TX, 77550); Holoubek, V. *Fed Proc* 37(6): 1735; 1978. (no refs)

78-2594 **3-(Glutathion-S-yl) -N- methyl-4-aminoazobenzene: Metabolic Formation and Affinity for Hepatic Ligandin (Meeting Abstract).** (Eng) Kadlubar, F. F. (Natl. Center Toxicological Res., Jefferson, AR, 72079); Ketterer, B. *Proc Am Assoc Cancer Res* 19: 36; 1978. (no refs)

78-2595 **Carcinomas of the Liver and Forestomach in Mice Ingesting Chlorobenzilate.** (Eng) Reuber, M. D. (NCI Frederick Cancer Res. Center, Frederick, MD, 21501). *Digestion* 16(4): 308-316; 1977.

The max tolerated dose of chlorobenzilate (CB) was given by continuous po administration to both sexes of two hybrid strains of mice, (C57BL/6 x C3HAnf)F_1 (strain X) and (C57BL/6 x AKR)F_1 (strain Y). Seven-day-old mice were administered 215 mg/kg CB in 0.5% gelatin daily by stomach tube. After the mice were weaned at 28 days of age, 603 ppm CB was mixed in the diet and fed ad libitum. The mice were killed after 83 wk. Eight of 17 male mice in each strain developed carcinomas of the liver, and one male mouse in each strain developed a hyperplastic nodule. Hyperplasia and carcinomas of the forestomach were found in 13/18 female strain X mice. Stomach tumors were also present in one male strain X mouse and in one female strain Y mouse. In addition to liver and stomach tumors, there were small numbers of malignant lymphomas and carcinomas of the lung in the CB-treated mice. The liver carcinomas were similar histologically to those observed in mice ingesting other organochlorine pesticides. The forestomach tumors varied from hyperplastic lesions to squamous cell carcinomas with invasion. It is probable that the forestomach lesions go through the various stages of preneoplasia before becoming malignant neoplasms, as do lesions in the liver. (17 refs)

78-2596 **Serum Alpha-Fetoprotein in Experimental Hepatocellular Carcinomas in Rats.** (Eng) Gopal Naidu, N. R. (Dept. Immunopathology, Postgraduate Inst. Medical Education and Res., Chandigarh, India); Aikat, B. K.; Dash, R. J.; Sehgal, S. *Eur J Cancer* 14(3): 295-301; 1978.

Serum α-fetoprotein (AFP) levels were measured by radioimmunoassay during the evolution of chemically induced liver cancer in Wistar rats, and the morphological type of lesion was correlated with AFP synthesis. Animals were given 0.06% 4-dimethylaminoazobenzene (DAB) or 3-methyl-4-dimethylaminoazobenzene (3-Me-DAB) in the diet for 6 mo or 25 μg aflatoxin B_1 po 5 days/wk for 8 wk. Thirty of the animals were fed the two azodyes for only 10 wk. Rats fed the azodyes developed hepatocellular carcinoma (HCC) by 8-24 mo, and they could be divided into two distinct groups, quick and slow responders, on the basis of their capacity to synthesize AFP. The quick responders developed tumors by

8-10 mo, they had high serum AFP values (250-1,110 μg/ml), and their tumors were mainly poorly differentiated HCC. The slow responders developed HCC by 16-24 mo, the tumors were better differentiated, and their serum AFP levels were $< 15\ \mu$g/ml. Both groups showed a biphasic response of AFP during the evolution of HCC. None of the animals in the aflatoxin B_1 group showed a rise in AFP levels of > 1-$2\ \mu$g/ml, and only 2/10 animals developed tumors after 26 mo. Twelve animals that were fed the carcinogenic diet for only 10 wk developed HCC after 20 mo. It is speculated that the transitory AFP elevation in patients with liver cirrhosis or hepatitis indicates a primary reaction and that these patients may eventually develop HCC under the influence of some unknown carcinogenic factors. (19 refs)

78-2597 **Studies on the Mechanism of Irreversible Protein Binding of Aromatic Hydrocarbons, Mediated by Rat Liver Microsomes: Binding of ^{14}C-Naphthalene and ^{14}C-1-Naphthol (Meeting Abstract).** (Eng) Hesse, S. (Abteilung Toxikologie, Gesellschaft fur Strahlen- und Umweltforschung, D-8042 Neuherberg/Munchen, W. Germany); Mezger, M. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R23; 1978. (1 ref)

78-2598 **Further Improvements in the Hepatocyte Primary Culture DNA Repair Test for Carcinogens: Detection of Carcinogenic Biphenyl Derivatives.** (Eng) Williams, G. M. (Naylor Dana Inst. Disease Prevention, American Health Foundation, 1 Dana Road, Valhalla, NY, 10595). *Cancer Lett* 4(2): 69-75; 1978.

Improvements in the protocol for the detection of autoradiographic unscheduled DNA synthesis (UDS) in hepatocyte primary cultures (HPC) are reported, and they were used to study the relationship between UDS and carcinogenicity in a series of biphenyl derivatives. With this protocol, simultaneous exposures to the test compound and ^{3}H-thymidine were commenced at the end of a 1.5-hr attachment period and continued overnight; use of an automatic counter made measurement of UDS simpler and more precise. Repeat assays indicated that the noncarcinogens biphenyl and 2-aminobiphenyl did not elicit UDS, but the carcinogens 4-aminobiphenyl, dimethylaminobiphenyl, and p-diaminobiphenyl were positive. Carcinogen-induced UDS could also be measured by liquid scintillation counting as increased acid-precipitable counts, but the background was high. The background could be reduced by hydroxyurea, but the increase produced by dimethylaminobiphenyl remained proportionally the same. In HPC exposed to label only, the autoradiographic background was lowest in areas of the emulsion where no cells were present, and it was slightly higher over the cytoplasm than the nucleus. The finding of comparable backgrounds over the cytoplasm and nucleus was consistent with binding to macromolecules other than DNA. (15 refs)

78-2599 **Perinatal Pharmacology of PCBs and PBBs (Meeting Abstract).** (Eng) Lucier, G. W. (Lab. Environmental Toxicology, NIEHS, Research Triangle Park, NC, 27709); McDaniel, O. S.; Schiller, C. M.; Matthews, H. B. *Environ Health Perspect* 20: 240-241; 1977. (no refs)

78-2600 **Cytogenetic Effects of M-Phenylene Diamine (MPDA) and Methylene Dianiline (MDA) on Human Leukocytes In Vitro (Meeting Abstract).** (Eng) Ho, T. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Tipton, S. C.; Epler, J. L. *In Vitro* 14(4): 350; 1978. (no refs)

78-2601 **Effect of Mixed-Function Oxidase Modifiers on Metabolism and Toxicity of the Oncogen Dioxane.** (Eng) Woo, Y. T. (Seamen's Memorial Res. Lab., U.S. Public Health Service Hosp., New Orleans, LA, 70118); Argus, M. F.; Arcos, J. C. *Cancer Res* 38(6): 1621-1625; 1978.

The effect of inducers and inhibitors of hepatic mixed-function oxidases (MFO's) excretion of p-dioxane-2-one, the major urinary metabolite of dioxane, was studied. Pretreatment of male Sprague-Dawley rats with inducers such as phenobarbital (PB), polychlorinated biphenyls (PCB), and, to a much lesser extent, 3-methylcholanthrene (MC) increased metabolite excretion and shortened the time of onset of peak excretion. On the other hand, MFO inhibitors such as 2,4-dichloro-6-phenylphenoxyethylamine and cobaltous chloride decreased metabolite excretion. These findings implicate the involvement of MFO's in the in vivo metabolism of dioxane. p-Dioxane-2-one (0.5-1.2 g/kg) and dioxane (5.0-5.8 g/kg) were then injected ip into rats. The former was found to be considerably more toxic (LD$_{50}$ 0.79 g/kg, vs 5.3 g/kg for dioxane), suggesting that increased metabolism of the latter may bring about increased toxicity. The LD$_{50}$ of dioxane in PCB-, MC-, and PB-treated rats was 4.4, 5.2, and 5.4 g/kg, respectively. Thus, there was an apparent correlation between the metabolism and toxicity of dioxane in PCB- and MC-pretreated rats, but PB had no effect on the toxicity of the solvent. The lack of a PB effect may be due to the fact that p-dioxane-2-one is further metabolized to exert its toxic effect. Indeed, pretreatment of rats with PCB and MC further increased the toxicity of p-dioxane-2-one, but PB appeared to have the opposite effect. It is proposed that the generation of a toxic substance from dioxane involves a multistep mechanism with p-dioxane-2-one as an intermediate. (33 refs)

78-2602 **Carcinomas, Sarcomas and Other Lesions in Osborne-Mendel Rats Ingesting Endrin.** (Eng) Reuber, M. D. (NCI Frederick Cancer Res. Center, Frederick, MD, 21510). *Exp Cell Biol* 46(3): 129-145; 1978.

Osborne-Mendel male and female rats ingesting 0.1, 1.0, 5, 10, and 25 ppm endrin (1,2,3,4,10,10-hexachloro- 6,7-epoxy-1,4,4a,5,6,7,8,8a- octahydro- 1,4:5,8-endoendodimethanonaphthalene) developed malignant tumors in a variety of organs, as well as tumors of the liver, at a high incidence rate. The incidence of carcinomas and sarcomas was highest in male rats ingesting 0.1 ppm and in female rats ingesting 0.1 or 1.0 ppm endrin. The tumors were highly malignant and they included some rare and unusual types, such as Kupffer cell sarcomas of the liver and leiomyosarcomas of the mammary gland and stomach. Carcinomas or sarcomas were also present in the adrenal cortex, mammary gland, thyroid, uterus, lymph nodes, and other organs. In addition male rats developed an increased incidence of chronic renal disease. Tumors also developed, but at a lower rate, in male and female rats ingesting 1 ppm dieldrin. They included leiomyosarcoma of the parotid gland, adenocarcinoma of the lung, carcinoma of the pancreas, and hepatocellular carcinoma of the liver. Most of the carcinomas in the females were in the mammary gland. It is concluded that endrin is a carcinogen when given to Osborne-Mendel rats in the diet. (23 refs)

78-2603 **Novel Synthesis of the Dihydroarene Precursors of Carcinogenic Arene Dihydrodiols and Diolepoxides.** (Eng) Fu, P. P. (Ben May Lab. Cancer Res., Univ. Chicago, Chicago, IL, 60637); Lee, H. M.; Harvey, R. G. *Tetrahedron Lett* (6): 551-554; 1978.

A two-step procedure is reported for the synthesis of the dihydroarene precursors of carcinogenic arene dihydrodiols and diol epoxides directly from the parent hydrocarbons. The procedure involves regioselective hydrogenation over a platinum/carbon catalyst to the tetrahydroarene, followed by dehydrogenation with 2,3-dichloro-5,6-dicyano-1,4-benzoquinone to the conjugated dihydroarene. (18 refs)

78-2604 **Cell and Microsome Mediated Binding of 7,12-Dimethylbenz(a)anthracene to DNA Studied by Fluorescence Spectroscopy.** (Eng) Ivanovic, V. (Inst. Cancer Res., Columbia Univ. Coll. Physicians and Surgeons, New York, NY, 10032); Geacintov, N. E.; Jeffrey, A. M.; Fu, P. P.; Harvey, R. G.; Weinstein, I. B. *Cancer Lett* 4(3): 131-140; 1978.

The fluorescence spectra of DNA isolated from hamster embryo cells incubated with 7,12-dimethylbenz(a)anthracene (DMBA) were compared with the spectra of DNA modified by DMBA or 7-hydroxymethyl-12-methylbenz(a)anthracene in a microsomal system and with the spectra of various model compounds. The spectra indicate that the 7-hydroxymethyl derivative bound to DNA in all cases and that it has a 9,10-dimethylanthracene-like chromophore. They also provide evidence of the similarity of the DNA-bound products of DMBA and its 7-hydroxymethyl derivative. The results are consistent with an activation mechanism that involves saturation of the 1,2,3,4-ring positions. (26 refs)

78-2605 **Evidence for the Involvement of a Diol-Epoxide in the Binding of 7,12-Dimethylbenz(a)anthracene to DNA in Cells in Culture.** (Eng) Dipple, A. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501); Nebzydoski, J. A. *Chem Biol Interact* 20(1): 17-26; 1978.

The binding of 7,12-dimethylbenz(a)anthracene (DMBA) to DNA was investigated in primary NIH Swiss mouse embryo cells cultured with 1,1,1-trichloropropene 2,3-oxide (TCPO), which inhibits epoxide hydrase. DMBA (0.07 μg/ml) was added to the culture medium, and the cells were harvested after 24 hr. TCPO inhibited the binding of DMBA to the murine cell DNA under conditions that did not appreciably decrease the overall metabolism of the carcinogen. This suggests that the formation of a trans-dihydrodiol is a necessary step in the metabolic pathway leading to DNA binding and that binding probably occurs through the generation of a reactive diol-epoxide. Chromatography of the DMBA-DNA product produced both in the presence and absence of TCPO suggests that two stereoisomeric diol-epoxides are involved in the binding of DMBA to DNA. (21 refs)

78-2606 **Differences Between Products of Binding of 7,12-Dimethylbenz(a)anthracene to DNA in a Target Tissue and in In Vitro Activating Systems (Meeting Abstract).** (Eng) Bigger, C. A. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501); Dipple, A. *Proc Am Assoc Cancer Res* 19: 162; 1978. (no refs)

78-2607 **Some Properties of Vicinal Diol-Epoxides Derived From Benz(a)Anthracene and Benzo(a)Pyrene.** (Eng) Phillips, D. H. (Chester Beatty Res. Inst., Fulham Rd., London SW3 6JB, England); Grover, P. L.; Sims, P. *Chem Biol Interact* 20(1): 63-75; 1978.

The alkylating properties of pairs of syn- and anti-isomers of two diol-epoxides derived from benzo(a)pyrene (BP) and of one derived from benz(a)anthracene (BA) were investigated. Of the anti-diol-epoxides, anti-BP 7,8-diol-9,10-oxide was the most reactive compound toward DNA, sodium p-nitrothiophenolate (in a nonaqueous solvent system), and 4-(p-nitrobenzyl)pyridine (in aqueous soln); anti-BP 9,10-diol-7,8-oxide was of intermediate reactivity and anti-BA 8,9-diol-10,11-oxide was the least reactive. The syn-diol-epoxides gave unsatisfactory results with DNA and 4-(p-nitrobenzyl)pyridine because of their rapid solvolysis in aqueous soln; with sodium p-nitrothiophenolate, their reactivity increased in the order syn-BP 7,8-diol-9,10-oxide > syn-BA 8,9-diol-10,11-oxide > syn-BP 9,10-diol-7,8-oxide. The products of the reaction between diol-epoxides and nucleic acids were examined by Sephadex LH-20 chromatography followed by high-pressure liquid chromatography (HPLC). The diol-epoxides reacted principally with the guanosine and adenosine moieties of RNA. (49 refs)

78-2608 **Cocarcinogenic and Irritant Factors of *Euphorbia Esula* L. Latex.** (Eng) Upadhyay, R. R. (Faculty Pharmacology, Dept. Pharmacognosy, Azarabadegan Univ., Tabriz, Iran); Bakhtavar, F.; Ghaisarzadeh, M.; Tilabi, J. *Tumori* 64(1): 99-102; 1978.

The latex of the plant *Euphorbia esula* contains the potent skin irritant ingenol-3-$\Delta^{2,4,6,8,10}$ pentene tetradecanoate and a cocarcinogen, ingenol-3-dodecanoate. The latter produced squamous cell papillomas when it was applied in a single dose with 7,12-dimethylbenz(a)anthracene to the back skin of NMRI mice. (15 refs)

78-2609 **Binding of Benzo(a)Pyrene and 7,12-Dimethylbenzanthracene to Oncornaviruses (Meeting Abstract).** (Eng) Harris, R. (Worcester Foundation Experimental Biology, Worcester, MA, 01545); Kupfer, D.; Luftig, R. B. *Fed Proc* 37(6): 1745; 1978. (no refs)

78-2610 **Prevention of 7,8,12- Trimethylbenz(a)anthracene-induced Leukemia in Rat (Meeting Abstract).** (Eng) Ueda, N. (Ben May Lab. Cancer Res., Univ. Chicago, Chicago, IL, 60637); Huggins, C. B. *Proc Am Assoc Cancer Res* 19: 5; 1978. (no refs)

78-2611 **The Structure of Tumours Derived from Mouse Submandibular Gland Epithelium Transformed In Vitro.** (Eng) Franks, L. M. (Dept. Cellular Pathology, Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2A 3PX, England); Knowles, M. A. *Br J Cancer* 37(2): 240-247; 1978.

The morphology and ultrastructure of 48 primary tumors derived from 5 cell lines of adult mouse salivary gland epithelial cells transformed in vitro are described. Four of the tumor-producing cell lines developed after treatment of mice with 7,12-dimethylbenzanthracene, and one arose without carcinogen treatment. There were no significant differences between the tumors from the carcinogen-treated and untreated cell lines. Tumors from four of the cell lines were adenocarcinomas with a wide range of structural variation. The fifth cell line produced tumors with carcinomatous and sarcomatous elements. The morphology and ultrastructure of the tumors derived from the in vitro-transformed cells were almost identical to those of common human salivary gland tumors, particularly their more malignant variants. (12 refs)

78-2612 **The Metabolic Activation of 7-Methylbenz(a)anthracene in Mouse Skin: High Tumour-initiating Activity of the 3,4-Dihydrodiol.** (Eng) Chouroulinkov, I. (L'Institut de Recherches Scientifiques sur le Cancer, B.P. No. 8, 94800 Villejuif, France); Gentil, A.; Tierney, B.; Grover, P.; Sims, P. *Cancer Lett* 3(5/6): 247-253; 1977.

7-Methylbenz(a)anthracene (MBA) and its 1,2-, 3,4-, 5,6-, and 8,9-dihydrodiols were tested for their ability to initiate tumors following topical application to female CDI mice. The test compounds were applied as a single 25-μg dose; 1 wk after initiation, tumor formation was promoted with 12-O-tetradecanoylphorbol-13-acetate (TPA) three times a week. A dose of 0.5 μg TPA was used for the first 22 wk, 1 μg for the following 14 wk. The 3,4-dihydrodiol initiated the formation of more papillomas than MBA, and they arose at an earlier stage. The 1,2- and 5,6-diols were almost devoid of tumor-initiating activity. These findings support the suggestion that the metabolic activation of MBA involves the formation of a biologically active vicinal diol-epoxide, in this case 3,4-dihydro-3,4-dihydroxy-7-methylbenz(a)anthracene. (21 refs)

78-2613 **Changes in the Surface of Normal Rat Cells Cultured In Vitro after Single Exposure to 7,12-Dimethylbenz(a)anthracene.** (Rus) Belisheva, N. K. (Lab. Tumor Cell Genetics, Inst. Cytology, Leningrad, USSR); Fridlianskaia, I. I. *Tsitologiia* 20(3): 329-332; 1978.

Cells from normal rats were incubated 72 hr with 7,12-dimethylbenz(a)anthracene (DMBA: 0.1 and 0.5 μg/ml), and changes in the cell surface were determined by agglutination reactions with concanavalin A. Control cultures were incubated with dimethylsulfoxide. Incubation with 0.5 μg/ml DMBA considerably increased the agglutination of the cells considerably compared to control cells; this remained significant for 30 days, after which the DMBA-treated cells died. Incubation with 0.1 μg/ml also increased cell agglutination during the first days, but there was no significant difference between treated and control cells after 4 days. The long-term increase in the agglutinability of the cells treated with 0.5 μg/ml DMBA, cells in which mitosis was strongly inhibited, was due to the presence of DMBA in the cells. The short-term increase seen after the lower dose was due to dilution of the carcinogen as a result of mitosis. (13 refs)

78-2614 **Dibenz(a,c)anthracene: A Potent Inhibitor of Skin-Tumor Initiation by 7,12-Dimethylbenz(a)anthracene.** (Eng) Slaga, T. J. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Viaje, A.; Buty, S. G.; Bracken, W. M. *Res Commun Chem Pathol Pharmacol* 19(3): 477-483; 1978.

The mechanism by which the weak tumor initiator dibenz(a,c)anthracene [DB(a,c)A] inhibits the skin tumor-initiating activity of 7,12-dimethylbenz(a)anthracene (DMBA) was investigated in 7- to 9-wk-old female Charles River CD1 mice. DB(a,c)A [200 nanomoles (nmol)] was a potent inhibitor of DMBA skin tumor initiation when given either 5 min or 1, 12, or 36 hr before DMBA. Pretreatment of mice with 200 nmole unlabeled DB[a,c]A at either 1, 12, or 36 hr before killing increased the in vitro epidermally mediated covalent binding of ^{3}H-DMBA to DNA more than pretreatment with unlabeled DMBA at comparable times. In vitro covalent binding of ^{3}H-DMBA to DNA was substantially decreased when epidermal homogenates were isolated from mice pretreated with 200 nmole each of DMBA and DB(a,c)A rather than with DMBA only. The results suggest that some competition at the level of polycyclic hydrocarbon metabolism or at the genome level may exist between metabolites of the weak carcinogen and those of the strong carcinogen. (14 refs)

78-2615 **[^{3}H]Tamoxifen Binding Studies in Dimethylbenzanthracene-induced Mammary Carcinomata (Meeting Abstract).** (Eng) Nicholson, R. I. (Tenovus Inst. Cancer Res., Welsh Natl. Sch. Medicine, Heath Park, Cardiff CF4 4XX, Wales); Daniel, C. P.; Davies, P.; Griffiths, K. *J Endocrinol* 75(3): 26P-27P; 1977. (no refs)

78-2616 **Interaction of Androgens with Oestradiol-17β Receptor Proteins in DMBA-induced Mammary Tumours--A Possible Oncolytic Mechanism.** (Eng) Nicholson, R. I. (Tenovus Inst. Cancer Res., Welsh Natl. Sch. Medicine, Heath Park, Cardiff, CF4 4XX, Wales); Davies, P.; Griffiths, K. *Eur J Cancer* 14(4): 439-445; 1978.

The direct action of testosterone and other C_{19} steroids on the binding of ^{3}H-estradiol-17β to its receptor protein in both cytoplasmic and nuclear compartments was studied in Sprague-Dawley rats with mammary tumors induced by 7,12-dimethylbenz(a)anthracene (single dose of 20 mg via intubation). Androst-5-ene-3β,17β-diol and androstenediol were the most potent reducers of binding capacity, with substantial inhibition of ^{3}H-estradiol-17β binding being observed at concentrations as low as 25 nanomoles/liter. Compounds that did not compete with ^{3}H-estradiol-17β for cytoplasmic estradiol-17β binding sites were unable to instigate the nuclear translocation of the estrogen receptor. Androgen-transferred estrogen receptors had a relatively short nuclear retention time and readily bound ^{3}H-estradiol-17β at 4 C. It is suggested that the ability of compounds such as androstenediol to translocate the estrogen receptor to the nucleus may be the basis for the antitumor activity of androgens. (26 refs)

78-2617 **7,12-Dimethylbenz(a)anthracene-induced Alteration of Mammary Epithelial Cell RNA-Synthetic Patterns in Rat Strains of High and Low Tumor Incidence.** (Eng) DeAngelo, A. B. (Chemical Carcinogenesis, NCI-Frederick Cancer Res. Center, Frederick, MD 21701); Hudgins, W. R.; Kerby, S. B. *Cancer Res* 38(2): 384-389; 1978.

The influence of rat strain on 7,12-dimethylbenz(a)anthracene (DMBA)-induced alterations in the incorporation of [^{3}H]uridine into mammary epithelial cell RNA was studied using female Sprague-Dawley and Long-Evans rats. From 1 to 4 days after 20 mg DMBA was given intragastrically to 51-day-old Sprague-Dawley rats, a high tumor incidence strain, the specific activities of cytoplasmic and nucleolar RNA extracted from mammary gland epithelial cells decreased to 64 and 69% of the control value, respectively. This decrease was followed by a 40-100% stimulation of cellular RNA specific activity over control values throughout the remainder of the 28-day test period. In DMBA-treated Long-Evans rats, a low tumor incidence strain, the specific activity of RNA extracted from mammary epithelial cells did not differ from control values at any of the times studied with the exception of a smaller early time period decrease. DMBA did not alter the RNA specific activities in liver, kidney, duodenum, or mammary fibroblasts derived from either strain. Exposure of epithelial cells to DMBA (0.05 μg/ml) for 20 hr in vitro showed a similar early decrease of RNA specific activity in the Sprague-Dawley rats. The results suggest that RNA synthesis may be an early and distinguishable event characterizing tissues undergoing transformation. (23 refs.)

78-2618 **Polycyclic Aromatic Hydrocarbon (PAH) Metabolism in Normal and Neoplastic Human Breast Epithelial Cells in Culture (Meeting Abstract).** (Eng) Greiner, J. W. (Frederick Cancer Res. Center, Frederick, D, 21501); Janss, D. H.; Shepherd, R. E. *In Vitro* 14(4): 39-340; 1978. (no refs)

-2619 **Factors Affecting the Binding of Polycyclic Aromatic Hydrocarbons to Human Embryo ells, and Transformable and Non-Transformable Hamster mbryo Cells (Meeting Abstract).** (Eng) Raineri, R. (Chemi- l Carcinogenesis Section, Frederick Cancer Res. Center, rederick, MD, 21501); Poiley, J.; Hillesund, T. *In Vitro* 4(4): 337-338; 1978. (no refs)

-2620 **Biochemical Activation of Aryl Hydrocarbon Hydroxylase Activity, Cellular Distribution of olynuclear Hydrocarbon Metabolites, and DNA Damage Polynuclear Hydrocarbon Products in Human Cells In itro.** (Eng) Milo, G. E. (Dept. Veterinary Pathobiology, hio State Univ., 1900 Coffey Road, Columbus, OH, 43210); lakeslee, J.; Yohn, D.; DiPaolo, J. A. *Cancer Res* 38(6): 538-1644; 1978.

12-Dimethylbenzanthracene (DMBA), 3-methylcholanth- ne (3-MC), and benzo(a)pyrene (BP) were added to cul- res of human skin fibroblasts, and the sequence of events ading to transformation was studied. BP (at concentrations 10 μg/ml) induced mixed-function hydroxylase activity, tered cell proliferation kinetics, and caused DNA damage, measured by altered grain count and bromodeoxyuridine corporation. 3-MC, at concentrations as high as 15 μg/ml, as ineffective. DMBA, at concentrations ≥6 μg/ml, in- uced mixed-function oxygenase and stimulated DNA syn- esis and cell proliferation, but caused little or no cytotoxici- or DNA damage at these concentrations. The oncarcinogenic analogs 6,8,12-trimethylbenzanthracene, -fluorodimethylbenzanthracene, anthracene, and phenanth- ne had no detectable effect on the human cells. These re- ılts indicate that BP is highly suspect as a direct carcinogen cocarcinogen for human cells. (32 refs)

8-2621 **Separation of Glutathione S-Transferase Activities with Expoxides from the Mouse Liver -Protein, a Major Polycyclic Hydrocarbon-Binding rotein.** (Eng) Sarrif, A. M. (Dept. Pharmacology and omprehensive Cancer Center, Univ. Alabama in irmingham Medical Center, Univ. Station, Birming- am, AL, 35294); McCarthy, K. L.; Nesnow, S.; Heidel- erger, C. *Cancer Res* 38(5): 1438-1443; 1978.

he relationship between the *h*-protein and enzymes of iouse liver was studied in C3H mice. Carboxymethylcel- ulose and isoelectrofocusing columns allowed the separation f *h*-protein from glutathione S-transferase activities with 1,2-epoxy-(3-p-nitrophenoxy)propane and benz(a)anthracene-5,6-oxide as substrates. It was also possible to purify the *h*-protein as a ^{3}H-7,12-dimethylbenz(a)anthracene conjugate and in its free form. These findings do not suggest an involvement of the *h*-protein in hydrocarbon carcinogen detoxification. (27 refs)

78-2622 **Plant Metabolic Activation of 1,2-Dibromoethane (EDB) to a Mutagen of Greater Potency.** (Eng) Scott, B. R. (Science Park, Res. Div., Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., P.O. Box 418, Smithville, TX, 78957); Sparrow, A. H.; Schwemmer, S. S.; Schairer, L. A. *Mutat Res* 49(2): 203-212; 1978.

An investigation was conducted to confirm the mutagenicity of 1,2-dibromoethane (DBE) in liquid tests and to determine whether the addition of plant mixtures to the microbial cultures would elevate the levels of mutagenicity detected. The plant extract consisted of cuttings from the Tradescantia hybrid clone 4430 that had been dried, ground into powder, and centrifuged. With both *Aspergillus nidulans* and *Escherichia coli* WP2 try-, inactivation and induction of mutation due to DBE were higher in the presence of the plant extract than in its absence. This effect was not due to a synergistic effect between the mutagen and the plant mix, because with ethyl methanesulfonate, the degree of mutagenicity and inactivation was depressed in the presence of the plant extract. The increase in the mutagenicity of DBE was energy-dependent, because omission of NADPH from the treatment mixture abrogated the effect. With *E. coli*, residual DBE exhibited a greater effect than the DBE distillate for inducing both inactivation and reversion to tryptophan independence. In the presence of the plant mix, however, elevated biological end-point values were observed for both fractions. The plant mix alone exerted no significant effect on inactivation. Apparently, DBE can be converted into a mutagen either by a pathway involving a breakdown product in vitro or by a pathway involving metabolic activation by plants. (31 refs)

78-2623 **Carcinogenesis in Rats of Combined Ethylene Dibromide and Disulfiram (Letter to Editor).** (Eng) Plotnick, H. B. (Natl. Inst. Occupational Safety and Health, Cincinnati, OH). *JAMA* 239(16): 1609; 1978.

Administration of ethylene dibromide (20 ppm, by inhalation) plus disulfiram (0.05% by wt, in diet) to Sprague-Dawley rats increased morbidity, mortality, and tumor formation. Hemangiosarcomas of the liver, spleen, omentum, and kidney were noted in both sexes; adenocarcinomas of the mammary gland were noted in females; and tubular atrophy of the testes was noted in males. It is not known if this industrial chemical-drug interaction is of clinical significance in humans. (no refs)

78-2624 **Induction of Respiratory Deficient Mutants in *Saccharomyces cerevisiae* by Mono- and Diazido Analogs of Ethidium.** (Eng) Morita, T. (Lab. Molecular Biology, Univ. Alabama, University Station, Birmingham, AL, 35294); Yielding, K. L. *Mutat Res* 56(1): 21-30; 1977.

The ability of ethidium bromide (EB), ethidium monoazide (EA_1), and ethidium diazide (EA_2) to induce respiratory-deficient (RD) mutants in *Saccharomyces cerevisiae* was compared under dark conditions and with photolysis in situ. EA_1 and EA_2 did not induce RD mutants in the dark at concentrations up to 6 μM, but they induced more than 90% RD mutants in the light at concentrations of 2 and 6 μM, respectively. The highest proportion of RD mutants for EA_1 and EA_2 was seen after 30 and 10 min exposure in the light, respectively. EB competed with either EA_1 or EA_2 for RD induction when applied before, but not after, the photolysis step, suggesting that the initial mutagenic binding sites for the azides are identical with those of EB. There was no self-rescue or recovery in azide mutagenesis as there is with EB, and recovery from azide mutagenesis could not be provoked by EB. Thus, a simple competition between the binding of EB and its azide analogs accounts for the prevention by EB of the azide-induced mutations. (17 refs)

78-2625 **Sister Chromatid Exchanges in Human Lymphoid Cell Lines Cultured in Diffusion Chambers in Mice.** (Eng) Huang, C. C. (Dept. Experimental Biology, Roswell Park Memorial Inst., Buffalo, NY, 14263); Furukawa, M. *Exp Cell Res* 111(2): 458-461; 1978.

A host-mediated assay system for evaluating the mutagenic potential of a compound was developed. It involves culturing cells from human lymphoid lines in diffusion chambers that are subsequently implanted into mice. The system was tested using two cell lines, B35M, a Burkitt's lymphoma line, and Jeff, a line derived from a normal individual. One day after implantation of the diffusion chambers, the hosts received 12 hourly ip injections of 5-bromodeoxyuridine (BUdR; 0.5 x 10^{-2} M). A single ip injection of cyclophosphamide at 7.5, 15, or 30 μg/g was given 13 hr after the first injection of BUdR, and the diffusion chambers were harvested 30-36 hr after the first BUdR injection. Chromosome preparations were examined for metaphases with differentially stained chromatids, and the number of sister chromatid exchanges (SCE)/metaphase was enumerated. The number of SCE/cell in all the cyclophosphamide-treated diffusion chambers was significantly higher than that in controls and was dose-dependent. The Burkitt cell line seemed to be more sensitive to cyclophosphamide treatment than the normal line. This host-mediated assay system may be useful for screening certain classes of mutagens or carcinogens. However, simplification of the procedure is needed, especially the BUdR administration. (17 refs.)

78-2626 **Association Between Chloroform Levels in Finished Drinking Water Supplies and Various Site-Specific Cancer Mortality Rates (Meeting Abstract).** (Eng) Hogan, M. D. (Biometry Branch, NIEHS, Research Triangle Park, NC, 27709); Chi, P.; Mitchell, T. J.; Hoel, D. G. *Environ Health Perspect* 20: 247-248; 1977. (no refs)

78-2627 **Dominant Lethal Studies with the Halogenated Olefins Vinyl Chloride and Vinylidene Dichloride in Male CD-1 Mice.** (Eng) Anderson, D. (Central Toxicology Lab., Imperial Chemical Industries, Ltd., Alderley Park nr. Macclesfield, Cheshire SKIO 4TJ, England); Hodge, M. C.; Purchase, I. F. *Environ Health Perspect* 21: 71-78; 1977.

Vinyl chloride (VC) and vinylidene dichloride (VDC) were assessed by the dominant lethal test for mutagenic activity in fertile male CD-1 mice. Compared with control males exposed to air, VC and VDC did not cause dominant lethal mutations at 3,000, 10,000, and 30,000 ppm and at 10, 30, and 50 ppm, respectively. (17 refs)

78-2628 **Report on Bioassay of 1,1-Dichloroethane for Possible Carcinogenicity.** (Eng) Fredrickson, D. S. (NIH, Bethesda, MD, 20014). *Fed Register* 43(143): 32190-32191; 1978.

1,1-Dichloroethane was administered by gavage to male and female Osborne-Mendel rats (382-950 mg/kg/day) and B6C3F1 mice (1,442-3,331 mg/kg/day) 5 days/wk for 78 wk. Rats were sacrificed 33 wk later, mice 13 wk later. Survival rates were poor in all rats and in several mouse groups. There were dose-related marginal increases in adenocarcinomas and in hemangiosarcomas among female rats and there was a statistically significant increase in the incidence of endometrial stomal polyps among female mice. (no refs)

78-2629 **The Mutagenic Effect of 1,2-Dichloroethane on *Salmonella Typhimurium* I. Activatio Through Conjugation with Glutathione In Vitro.** (Eng Rannug, U. (Environmental Toxicology Unit, Univ Stockholm, S-106 91 Stockholm, Sweden); Sundvall

A.; Ramel, C. *Chem Biol Interact* 20(1): 1-16; 1978.

The weak mutagenicity of 1,2-dichloroethane (1,2-DE) for *Salmonella typhimurium* TA1535 was increased by the addition of a metabolizing system, ie, the S-9 postmitochondrial rat liver fraction. This activation was further increased by the addition of reduced glutathione (GSH). No activation occurred when GSH was added in the presence of a totally denatured S-9 fraction or in the absence of this fraction. Glutathione S-transferases A and C significantly increased the mutagenicity of 1,2-DE in the presence of GSH. A synthetic conjugate, S-(2-chloroethyl)-L-cysteine, gave a strong direct mutagenic effect at 0.2-5.0 μmol/plate, concentrations at which no effects were seen with 1,2-DE. Thus, it is concluded that 1,2-DE is activated by conjugation to GSH. (24 refs)

78-2630 **Pharmacokinetics of Halogenated Ethylenes (Meeting Abstract).** (Eng) Filser, J. G. (Institut fur Toxikologie, Universitat Tubingen, Wilhelmstrasse 56, D-7400 Tubingen, W. Germany); Bolt, H. M. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R22; 1978. (2 refs)

78-2631 **Alkylation of DNA and RNA Metabolites of Vinyl Chloride and Vinyl Bromide (Meeting Abstract).** (Eng) Laib, R. J. (Institut fur Toxikologie, Universitat Tubingen, Wilhelmstrasse 56, D-7400 Tubingen, W. Germany); Ottenwalder, H. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R21; 1978. (1 ref)

78-2632 **Inhalation Toxicity of Vinyl Chloride and Vinylidene Chloride.** (Eng) Lee, C. C. (Pharmacology and Toxicology, Midwest Res. Inst., 425 Volker Blvd., Kansas City, MO, 64110); Bhandari, J. C.; Winston, J. M.; House, W. B.; Peters, P. J.; Dixon, R. L.; Woods, J. S. *Environ Health Perspect* 21: 25-32; 1977.

Experiments on the inhalation toxicity and carcinogenicity of vinyl chloride (VC) and vinylidene chloride (VDC) in mice and rats are reported. The exposure of mice to 1,000 ppm VC for 6 hr/day, 5 days/wk caused some acute deaths with toxic hepatitis and marked tubular necrosis of the renal cortex. During the sixth month, mice exposed to 1,000, 250, or 50 ppm VC became lethargic, lost weight quickly, and died; only a few mice exposed to 50 ppm survived for 12 mo. In mice exposed to 50, 250, or 1,000 ppm VC, there was a high incidence of bronchioloalveolar adenoma, mammary gland tumors (including ductular adenocarcinoma), squamous and anaplastic cell carcinomas with metastasis to the lung, and hemangiosarcoma (HS) in the liver and, to a lesser extent, in other organs. The incidence and severity of these tumors were proportional to the levels and duration of exposure. Malignant lymphoma involving various organs was observed in a few mice. Rats were more resistant to the toxic effects of VC, and exposure to 1,000 ppm slightly depressed the body wt of females. Exposures of 250 and 1,000 ppm VC caused a number of deaths and hepatic HS during the 9th mo. Most rats with hepatic HS also developed HS in the lung. HS occasionally occurred in other tissues, including omentum, mesentery, or sc tissue, of rats exposed to 50, 250, or 1,000 ppm VC. The exposure of mice to 55 ppm VDC also caused a few acute deaths and a few hepatic HS. Inflammatory, degenerative, and mitotic changes occurred in the liver. No mouse exposed to VDC developed any mammary gland tumors. Several mice had a bronchioloalveolar adenoma. The exposure of rats to 55 ppm VDC slightly depressed body wt; HS occurred in the mesenteric lymph node or sc tissue of two rats. (25 refs)

78-2633 **Formation of $3,N^4$-Ethenocytidine Moieties in RNA by Vinyl Chloride Metabolites In Vitro and In Vivo.** (Eng) Laib, R. J. (Institut fur Toxikologie, Universitat Tubingen, Wilhelmstrasse 56, D-7400 Tubingen, W. Germany); Bolt, H. M. *Arch Toxicol (Berl)* 39(3): 235-240; 1978.

Rats were exposed to ^{14}C-vinyl chloride, and the in vivo liver metabolites were examined. In addition to RNA and $1,N^6$-ethenoadenosine, $3,N^4$-ethenocytidine was also radioactively labeled. In vitro experiments using vinyl chloride-exposed liver microsomes and NADPH produced the same results. These alkylation mechanisms are consistent with the mutagenic and carcinogenic properties of vinyl chloride. (22 refs)

78-2634 Disposition and Metabolism of [^{14}C]1,1-Di**chloroethylene after Single Oral Administration in Rats (Meeting Abstract).** (Eng) Reichert, D. (Dept. Toxicology, Univ. Wurzburg, Versbacher Landstrasse 9, D-8700 Wurzburg, W. Germany); Werner, H. W. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R22; 1978. (1 ref)

78-2635 **1,1-Dichloroethylene Hepatotoxicity: Proposed Mechanism of Action and Distribution and Binding of ^{14}C Radioactivity Following Inhalation Exposure in Rats.** (Eng) Jaeger, R. J. (Toxicology Unit, Dept. Physiology, Kresge Center Environmental Health, Harvard Sch. Public Health, 665 Huntington Ave., Boston, MA, 02115); Shoner, L. G.; Coffman, L. *Environ Health Perspect* 21: 113-119; 1977.

The mechanism of 1,1-dichloroethylene (1,1-DCE)-induced hepatotoxicity and its potentiation by fasting were determined in male Holtzman rats, and results on tissue distribution and binding of radioactivity (RA) were related to the proposed mechanism of action. Trichloropropane epoxide (TCPE: 0.1 ml/kg po), an epoxide hydrase inhibitor, significantly increased the toxicity of 1,1-DCE (100-150 ppm by inhalation, x 4 hr) in 18-hr-fasted (FA) rats, as measured by serum sorbitol dehydrogenase (SDH) elevation. When FA rats were exposed to 250 ppm 1,1-DCE alone, hepatic citric acid levels were increased 416%, SDH activity 636.4-fold (vs increases of 122% and 100-fold in fed [FE] rats). It was hypothesized that mitochondrial injury was associated with inhibition of the tricarboxylic acid cycle and that monochloroacetic acid is the toxic metabolite of 1,1-DCE. When FE and FA rats were exposed for 2 hr to 2,000 ppm ^{14}C-1,1-DCE in a recirculating exposure chamber, there was no difference in rate of uptake between the two groups. FA rats had more water- or trichloroacetic acid (TCA)-soluble 1,1-DCE metabolites in their tissues than FE rats. At 30 min postexposure, most of the total RA (mainly TCA-soluble) was detected in the kidneys of both groups. FA rats had more tissue-bound or TCA-insoluble RA in their mitochondrial and microsomal fractions than FE rats, but both groups had approx the same rate of disappearance of RA (half-time of 2-3 hr). At 26 hr, the urinary excretion of RA was similar in FE and FA rats (36.6%). The results suggest that although there are no significant metabolic differences between FE and FA rats, there does appear to be a significant difference in the in vivo pathway for the products of metabolism. The fact that mice develop renal tumors after long-term 1,1-DCE exposure but rats only show significant hepatocellular injury is explained. (27 refs)

78-2636 **The Biological Fate of Vinylidene Chloride in Rats.** (Eng) Jones, B. K. (Central Toxicology Lab., Imperial Chemical Industries, Ltd., Alderley Park, Cheshire SK10 4TJ, England); Hathway, D. E. *Chem Biol Interact* 20(1): 27-41; 1978.

The fate of vinylidene chloride (DCE) following intragastric (ig), iv, or ip administration to male Alderley Park rats was determined. In rats given 500-μg/kg or 350-mg/kg doses of labeled DCE by all three routes, almost all the radioactivity was recovered within 72 hr of dosing; with 500 μg/kg ig, however, small amounts of label were still being recovered 72-108 hr after dosing. With the higher ig dose, nearly 70% of the dose was excreted as unchanged DCE and 1% as DCE-related CO_2 by the lungs; with the lower dose, urinary excretion accounted for 80%, and $<1\%$ of the unchanged DCE plus 4%-6% of DCE-CO_2 were eliminated by the lungs. This change in excretion pattern appeared to be due to a saturable drug metabolism and an arterial-alveolar transfer of unchanged DCE from the blood. In comparison, 80% of the small iv dose was excreted unchanged from the blood within 1 hr ($>60\%$ within 5 min). The excretion pattern after a small ip dose was intermediate between that resulting from iv and that from ig administration. For groups of rats administered various dose levels ig within a 1,300-fold dose range, the plot of the pulmonary excretion of unchanged DCE against the log of the reciprocal doses was biphasic. Radiography following ig doses revealed the presence of large amounts of the ^{14}C label in the kidneys and liver after 30 min and a more general distribution of ^{14}C throughout the soft organs at 1 hr. The kidneys and liver retained the label for the longest times. Biotransformation resulted in thiodiglycollic acid and an N-acetyl-S-cysteinylacetyl derivative as the major urinary metabolites, together with substantial amounts of chloroacetic acid, dithioglycollic acid, and thioglycollic acid. (12 refs)

78-2637 **Carcinogenicity Studies on Vinylidene Chloride.** (Eng) Viola, P. L. (Regina Elena Inst. Cancer Res., Rome, Italy); Caputo, A. *Environ Health Perspect* 21: 45-47; 1978.

The possible carcinogenicity of vinylidene chloride (VDC) was determined in two experiments, one using Wistar rats, one using Sprague-Dawley rats. The Wistar rats inhaled VDC for 4 hr/day, 5 days/wk, for 12 mo at a concentration of 200 ppm for 5 mo, 100 ppm thereafter. The animals were allowed to die spontaneously (life-span was 22-24 mo). Tumors, all occurring in the abdominal cavity except for one in the liver and one in the lung, were found in 8/51 males and 9/23 females. The diagnosis was reticulum cell sarcomas of a nonsyncytial type. However, many abdominal tumors were also found in control rats (5/30 males, 10/30 females). This prompted the second experiment, in which Sprague-Dawley rats were exposed to 75 or 100 ppm. Tumor incidence in the controls was practically identical to that found in the 100 ppm group and higher than that in the 75 ppm group. Microscopic examination of the tissues and organs from all animals is incomplete. Nevertheless, it seems clear that there is no grossly observable correlation between tumor formation and VDC inhalation. (7 refs)

78-2638 **Metabolism of Trichloroethylene and Tetrachloroethylene in Human Subjects.** (Eng) Ikeda, M. (Dept. Environmental Health, Tohoku Univ. Sch. Medicine, Sendai 980, Japan). *Environ Health Perspect* 21: 239-245; 1977.

The metabolism of trichloroethylene (3C-E) and tetrachloroethylene, (4C-E) in humans and the urinary concentration of metabolites of these compounds in persons occupationally exposed to their vapors was determined. There was a linear correlation between 3C-E concentration in the work environments and the level of total trichloro compounds in the urine of workers. The trichloroethanol level was also linearly related to 3C-E concentration, but trichloroacetic acid levels deviated from linearity at 3C-E levels > 50 ppm. With 4C-E, the trichloroethanol, trichloroacetic acid, and total trichloro compound levels all plateaued at 4C-E levels < 100 ppm. The respiratory and urinary half lives of 3C-E were 25 and 42 hr, respectively, but those of 4C-E were 65 and 144 hr, respectively. Urinalysis of a 38-yr-old man addicted to 3C-E revealed a urinary half-life of > 70 hr. An automated system for the determination of total trichloro compounds in human urine is described. (35 refs)

78-2639 **Transforming Activities of Trichloroethylene and Proposed Industrial Alternatives.** (Eng) Price, P. J. (Microbiological Associates, Torrey Pines Res. Center, 2945 Science Park Road, La Jolla, CA, 92037); Hassett, C. M.; Mansfield, J. I. *In Vitro* 14(3): 290-293; 1978.

The carcinogenicity of trichloroethylene (TCE) and three industrial substitutes, 1,1,1-trichloroethane, tetrachloroethylene, and methylene chloride, was tested in vitro using the F1706 Fischer rat embryo cell system. All compounds induced transformation in the rat cells. Toxicity studies indicated that TCE was the least toxic of the four compounds by a factor of at least 100. Sc injection of transformed cells into newborn Fischer rats resulted in undifferentiated fibrosarcomas at the injection site in 100% of the animals in 27-68 days. (4 refs)

78-2640 **Carcinogenicity Studies on Halogenated Hydrocarbons.** (Eng) Weisburger, E. K. (Carcinogen Metabolism and Toxicology Branch, NCI, NIH, Bethesda, MD, 20014). *Environ Health Perspect* 21: 7-16; 1977.

The acute and chronic carcinogenicities of a series of halogenated hydrocarbons were studied in Osborne-Mendel rats and B6C3F1 mice. The test chemical was given by gavage 5 days/wk for 6 wk (acute studies) or 78 wk (chronic studies). Trichloroethylene had little effect on rats but caused hepatocellular carcinomas (HC) in mice. Chloroform caused HC in mice, kidney tumors in male rats, and thyroid tumors in female rats. 1,1,1-Trichloroethane had no effect on either species, whereas iodoform produced a slight increase in thyroid tumors in male rats. 1,2-Dibromoethane and 1,2-dibromo-3-chloropropane (DBCP) caused squamous cell carcinomas of the stomach with metastases in both species: DBCP also caused mammary tumors in female rats. 1,2-Dichlorethane produced some stomach tumors in male rats, HC in male mice, and lung tumors in males and females of both species. 1,1-Dichloroethane had little effect on rats, but caused a slight increase in HC in male mice. 1,1,2-Trichloroethane and hexachloroethane also had little effect on rats but increased the number of HC in mice, whereas 3-chloropropene decreased the hepatomagenic effect in mice. Tetrachloroethylene increased the number of HC in mice but had no effect in rats. Carbon tetrachloride, used as a positive control, produced HC in rats and mice as well as thyroid tumors in mice. The results suggest that po administration of many halogenated aliphatics might pose a carcinogenic hazard to humans on continued exposure. (9 refs)

78-2641 **Elevated CEA Levels in a Group of Polyvinyl Chloride Workers.** (Fre) Page, M. (Service de Recherche et de Biochimie, Hotel-Dieu de Quebec, Quebec, Canada); Theriault, L.; Delorme, F. *Ann Immunol (Paris)* 128 C(6): 1101-1103; 1977.

Plasma levels of carcinoembryonic antigen (CEA) were assayed in 200 polyvinyl chloride (PVC) workers and compared to levels in a normal population of 10,000 subjects that included smokers and nonsmokers. The results for the PVC workers were tabulated according to length of time in the industry and type of work performed. Elevated CEA levels (above 5 nanograms/ml) were found in 56% of the workers but only 9.2% of the nonworkers. It is concluded that screening PVC workers for elevated CEA levels could detect benign or malignant hepatic lesions. (8 refs.)

78-2642 **Effects of the Epoxide Hydrase Inhibitor, 1,1,1-Trichloropropane-2,3-oxide on the Genetic Activity of Aflatoxin B_1 Metabolites in In Vitro Activation Test Systems.** (Eng) Callen, D. F. (Environmental Mutagenesis Branch, Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC, 27709); Ong, T. M. *Mutat Res* 49(3): 371-376; 1978.

Studies were conducted to determine if the epoxide hydrase inhibitor 1,1,1-trichloropropane 2,3-oxide (TCPO) is genetically active and if its addition to an in vitro activation system would alter the genetic activity of aflatoxin B_1 (AFB_1) to either *Neurospora crassa* or *Saccharomyces cerevisiae*. TCPO was genetically active to cells of *S. cerevisiae* and conidia of *N. crassa*. The genetic activity could be eliminated by addition of the supernatant fraction of hamster liver homogenate (S-9) to the treatment soln. This can be explained by the rapid binding of TCPO to epoxide hydrase and other proteins within the S-9 fraction, which results in a partial or complete absence of unbound TCPO able to penetrate the indicator cell and bind to DNA. The addition of TCPO to an in vitro activation system containing AFB_1 resulted in an increase in the genetic activity of AFB_1 that was dependent on the TCPO dose. This effect can be interpreted as genetic evidence for the proposal that AFB_1 is metabolized to aflatoxin B_1 2,3-oxide, which can be further metabolized by epoxide hydrase to a dihydrodiol that is not genetically active. The inhibition of epoxide hydrase activity by TCPO results in an increase in the availability of AFB 2,3-oxide to interact with DNA, which is reflected in the increased AFB mutagenicity. Alternatively, competition between TCPO, which is itself genetically active, and the active metabolite of AFB$_1$ within the S-9 fraction could also result in an increase in mutagenicity. (12 refs)

78-2643 **Alkylating and Mutagenic Effects of Allyl and Allylogenic Compounds (Meeting Abstract).** (Eng) Eder, E. (Dept. Toxicology, Univ. Wurzburg, Versbacher Landstrasse 9, D-8700 Wurzburg, W. Germany); Neudecker, T. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R21; 1978. (1 ref)

78-2644 **Effect of Carbon Tetrachloride on Carcinogenicity of *Petasites japonicus* and Transplantability of Induced Tumors.** (Eng) Mori, H. (Dept. Pathology, Gifu Univ. Sch. Medicine, 40 Tsukasamachi, Gifu 500, Japan); Ushimaru, Y.; Tanaka, T.; Hirono, I. *Gann* 68(6): 841-845; 1977.

The effect of carbon tetrachloride on the carcinogenicity of the flower stalk of *Petasites japonicus* was examined in three groups of inbred ACI rats. Group 1 received a 4% petasites diet and CCl$_4$ (intragastrically, once every 2 wk) over a 30-wk period. Group 2 received only the 4% petasites diet and Group 3 received CCl$_4$ and a basal diet. Both Groups 1 and 2 showed the same incidence of hemangioendothelial sarcoma of the liver (6/22). However, the incidence of hyperplastic liver nodules (19/22) and liver cell adenomas (8/22) in Group 1 was significantly higher than that in Group 2 (2/22, 0/22). No hyperplastic liver nodules or liver cell adenomas were noted in Group 3. These results indicate that CCl$_4$ probably enhanced the carcinogenic activity of *Petasites japonicus* in hepatocellular tumorigenesis. Three hepatic hemangioendothelial sarcomas induced in rats of Groups 1 and 2 were transplanted sc and established as transplantable lines. (7 refs)

78-2645 **Chronic Toxicity and Reproduction Studies of Hexachlorobutadiene in Rats.** (Eng) Kociba, R. J. (Toxicology Res. Lab., Health and Environmental Res., Dow Chemical Co., Midland, MI, 48640); Schwetz, B. A.; Keyes, D. G.; Jersey, G. C.; Ballard, J. J.; Dittenber, D. A.; Quast, J. F.; Wade, C. E.; Humiston, C. G. *Environ Health Perspect* 21: 49-53; 1977.

The carcinogenic, reproductive, and toxic effects of po hexachlorobutadiene (HCBD), an unwanted by-product of processes associated with the chlorination of hydrocarbons, were assessed in Sprague Dawley rats. The LD$_{50}$ values following a single po dose ranged from 504 to 667 mg/kg in adult male rats and from 200 to 400 mg/kg in adult female rats. In 21-day-old male and female weanling rats, the LD$_{50}$ values were 46-91 and 26-81 mg/kg, respectively. In a chronic toxicity study, the ingestion of 20 mg/kg/day HCBD for up to 2 yr caused multiple toxicologic effects, primarily of the kidney. These included the development of renal tubular adenomas and adenocarcinomas. The ingestion of 2 mg/kg/day HCBD caused lesser toxicity and no evidence of neoplasia. No toxic effects were observed in rats chronically exposed to a po dose of 0.2 mg/kg/day. Thus, a dose-response relationship was observed for HCBD-induced toxicity,which affected primarily the kidney; HCBD-induced neoplasms occurred only at a dose level higher than that causing discernible renal injury. In a reproduction study, 20 or 2.0 mg/kg/day HCBD induced slight maternal toxicity (primarily of the kidney), but caused no adverse effects on reproductive parameters (percent pregnancy and neonatal survival/development). A decreased neonatal body wt was noted at 20 mg/kg/day. No toxicologic effects were observed among adult rats at 0.2 mg/kg/day or among neonates at 0.2 or 2.0 mg/kg/day. (14 refs)

78-2646 **The Gas Chromatographic Analysis of Bis(chloromethyl) Ether in Air.** (Eng) Baba, Y. (Natl. Res. Inst. Pollution and Resources, Kawaguchi, Saitama 332, Japan); Tanaka, T. *Bull Chem Soc Jpn* 51(1): 317-318; 1978.

A gas chromatographic method for the determinatior of bis(chloromethyl) ether (BCME) concentration was devised. BCME was converted into bis(ethylthio methyl) ether by reaction with sodium ethanethiolat in the hexane soln. The sulfur-containing derivativ was then detected using a flame photometric detector This method allows BCME determination in sample of air containing BCME and other compounds. (refs)

78-2647 **Study of the Mutagenic Potential of Bis(2 chloroethyl) and Bis(2-chloroisopropyl Ethers in Mice by the Heritable Translocation Tes (Meeting Abstract).** (Eng) Jorgenson, T. A. (Stanfor Res. Inst., Menlo Park, CA); Rushbrook, C. J.; Newell G. W.; Tardiff, R. G. *Mutat Res* 53(1): 124; 1978 (no refs)

78-2648 **Butyric Acid Suppression of the In Vitro Neoplastic State of Syrian Hamster Cells.** (Eng Leavitt, J. (Dept. Biochemistry, Johns Hopkins Univ., Sch Hygiene and Public Health, Baltimore, MD 21205); Barrett J. C.; Crawford, B. D.; Ts'o, P. O. *Nature* 271(5642): 262-265 1978.

The effects of butyric acid on altered growth properties associated with the neoplastic state were examined in a highl tumorigenic Syrian hamster fibroblast cell line, BP6T, anc two clonal isolates, BP6T-But 1 and BP6T-But 2 (isolated from BP6T cells grown in the presence of 2 mM butyric acid). The addition of 2 mM butyric acid to the growth medium decreased the doubling time by 4-7 hr and reduced the cell saturation density by 20%-70% in all three cell lines. Cloning efficiencies ranged from 14% to 25% in medium containing butyric acid and 65-85% in normal medium. In contrast to the slight effect on general growth, butyric acid produced marked effects on colony morphology, anchorage-dependent growth, and fibrinolytic activity of BP6T cells. All colonies displayed morphologies similar to those of BP6T-But 1 and BP6T-But 2; ie, they were oriented in a uniform, parallel pattern within the colony, a pattern similar to that of untransformed Syrian hamster cells. In the presence of butyric acid, none of the cell lines was able to form colonies in semisolid agar, compared to a 50% cloning efficiency without butyric acid. BP6T-But 1 and BP6T-But 2 cells grown in the presence of butyric acid produced levels of extracellular fibrinolytic activity that were 85%-95% lower than those of BP6T cells grown in the absence of butyric acid. BP6T and BP6T-But 1 cells grown in the presence of butyric acid (the former for 48 hr, the latter for 14 passages) had a 70%-90% reduction in intracellular fibrinolytic activity. All of these effects were reversible. It is suggested that this system could be of value in investigations of the cellular control mechanism of neoplasia. (21 refs.)

78-2649 **A Linear Relationship Between Tumorigenic Potency In Vivo and Mutagenic Potency at the Heterozygous Thymidine Kinase (TK$^{+/-}$) Locus of L5178Y Mouse Lymphoma Cells Coupled with Mammalian Metabolism (Meeting Abstract).** (Eng) Clive, D. (Genetic Toxicology Lab., Burroughs Wellcome Co., Research Triangle Park, NC). *Mutat Res* 53(1): 95; 1978. (no refs)

78-2650 **Loss of Activity of an N'-Methyl-4-pyridone-5-carboxamide-forming N'-Methylnicotinamide Oxidase in Livers of Rats Fed 2-Acetylaminofluorene.** (Eng) Ohkubo, M. (Div. Biochemistry, Chiba Cancer Center Res. Inst., Nitona-cho 666-2, Chiba 280, Japan); Fujimura, S. *Cancer Res* 38(3): 697-702; 1978.

N^{1}-Methylnicotinamide oxidase (MNO) activity was compared in the livers of control and 2-acetylaminofluorene (2-AAF)-treated Wistar rats. In the liver homogenates from the 39-wk-old controls, three enzyme fractions were eluted by diethylaminoethyl cellulose column chromatography. Fraction I produced 1-methyl-2-pyridone-5-carboxamide (2-PY) and was identified as xanthine oxidase. Fraction II formed 2-PY (72%) in preference to 1-methyl-4-pyridone-5-carboxamide (37%), but Fraction III produced only 4-PY. The 4-PT- and 2-PY-forming activity of Fraction II (MNO-I) increased with an increase in pH, but the activity of Fraction III (MNO-II) was max at pH 8.5. In the liver homogenate of a rat fed a diet containing 0.05% 2-AAF for 24 wk (beginning at age 6 wk), followed by a normal diet for 9 wk, MNO activities were observed only in Fractions I and II. MNO-II activity was lacking, but the elution character of Fractions I and II was similar to that of the corresponding fractions from the control liver. In Fraction II from control or 2-AAF-treated liver, the Km's for the MNO of 2-PY and 4-PY formation were in the same range. In the control liver, the Km for the MNO of 4-PY formation was one-fifth of that of the Fraction II enzymes. (13 refs.)

78-2651 **A Glycolipid Cascade in Experimental Liver Tumorigenesis (Meeting Abstract).** (Eng) Morre, D. J. (Purdue Univ., West Lafayette, IN, 47907); Merritt, W. D.; Richardson, C. L.; Kloppel, T. M.; Keenan, T. W. *Fed Proc* 37(6): 1767; 1978. (no refs)

78-2652 **Alteration of the Ability of Liver Microsomes to Activate N-2-Fluorenylacetamide to a Mutagen of *Salmonella typhimurium* During Hepatocarcinogenesis.** (Eng) Stout, D. L. (Dept. Anatomic and Res. Pathology, M. D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); Becker, F. F. *Cancer Res* 38(8): 2274-2278; 1978.

Male Sprague-Dawley rats were exposed to a hepatocarcinogenic regimen of N-2-fluorenylacetamide (FAA: 0.06% in the diet) consisting of cycles of 3 wk on the carcinogen followed by 1 wk on a normal diet, with four cycles representing a full carcinogenic exposure. At weekly intervals, postmitochondrial (S9) and postmicrosomal liver fractions were isolated from the rats and tested for their ability to activate FAA and N-hydroxy-FAA to metabolites mutagenic for *Salmonella typhimurium* TA1538. FAA exposure resulted in an almost immediate enhancement of S9 (mutation-producing) activity, which reached a max of 300% by the end of Cycle 1. Although still elevated through Cycle 2, S9 activity declined rapidly and was severely depressed during the remainder of the feeding regimen. The diminished ability of the microsomal system to activate the parent compound was striking when hepatic nodules were tested separately and compared with surrounding parenchyma or control liver. Throughout the same exposure period, the activity of the postmicrosomal fraction was relatively unaltered. The decrease of S9 activity was accompanied by a decline in microsomal protein levels. The reduced mutagenizing activity of the liver microsomes is in accord with recent results indicating a decline in drug-metabolizing activity during chemical hepatocarcinogenesis. (22 refs)

78-2653 **Gangliosides of Liver Tumors Induced by N-2-Fluorenylacetamide. I. Ganglioside Altera-**

tions in Liver Tumorigenesis and Normal Development. (Eng) Merritt, W. D. (Section Cytology, Yale Univ. Sch. Medicine, New Haven, CT, 06510); Richardson, C. L.; Keenan, T. W.; Morre, D. J. *J Natl Cancer Inst* 60(6): 1313-1327; 1978.

Ganglioside patterns were studied in hyperplastic liver nodules and hepatomas induced in Wistar rats by a low-protein diet containing 0.05% of the carcinogen N-2-fluorenylacetamide (2-AAF). The patterns were compared with those in the livers of fetal, newborn, and normal adult rats. Ganglioside sialic acid levels were elevated in the nodules and hepatomas, and the levels were higher in fetal than in adult liver. Monosialogangliosides and disialogangliosides were evenly distributed in normal adult liver, but the monosialogangliosides predominated in 2-AAF-treated livers. Superficially at least, the ganglioside changes during tumorigenesis mimicked the reversal of liver differentiation. The enzyme that converts lactosylceramide to G-M3 was one of the earliest to show increased activity in hyperplastic tissues and the tissues surrounding nodules; a transient rise in G-M2 in newborn rat liver was duplicated by the high G-M2 levels in well-circumscribed, poorly differentiated hepatomas; and G-M3 levels were highest in fetal liver and in well-differentiated hepatomas. G-D1a levels were also elevated in fetal tissues and hepatomas, compared with normal adult liver. It is concluded that the ratio of higher monosialogangliosides to higher disialogangliosides is highest in fetal tissue, lowest in the adult, and correlated with increasing tumorigenicity. (61 refs)

78-2654 **Effects of Dietary Protein and Fat Levels on Liver and Urinary Bladder Neoplasia in Mice Fed 2-Acetylaminofluorene (Meeting Abstract).** (Eng) Frith, C. H. (Univ. Arkansas Medical Sciences, Little Rock, AR); Norvell, M. J. *Lab Invest* 38(3): 345; 1978. (no refs)

78-2655 **Progesterone Inhibition of 2-AAF Induction of Urogenital Tumors in Rabbits (Meeting Abstract).** (Eng) Coogan, P. S. (Rush-Presbyterian-St.Luke's Medical Center, Chicago, IL, 60612); Maganini, H. T.; Newton, J. J.; Hass, G. M. *Lab Invest* 38(3): 339; 1978. (no refs)

78-2656 **Steroid Hormone Receptors in N-Hydroxy-N-2-fluorenylacetamide (N-Hydroxy-2-FAA)-induced Mammary Adenocarcinoma in the Rat (Meeting Abstract).** (Eng) Li, S. A. (Veterans Admin. Hosp., Minneapolis, MN, 55417); Malejka-Giganti, D.; Li, J. J. *Proc Am Assoc Cancer Res* 19: 47; 1978. (no refs)

78-2657 **Effects of Selenium on the Mutagenicity of 2-Acetylaminofluorene (AAF) and AAF Metabolites (Meeting Abstract).** (Eng) Jacobs, M. M. (Univ. Texas System Cancer Center, Houston, TX, 77030); Matney, T. S.; Griffin, A. C. *Mutat Res* 53(1): 73; 1978. (no refs)

78-2658 **Estimation of N-Acetoxy-2-acetylaminofluorene (NA-AAF)-induced Mutation as a Function of Binding at the Hypoxanthine-guanine-phosphoribosyl-transferase Locus in Human Diploid Cells (Meeting Abstract).** (Eng) Huang, S. L. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709). *Mutat Res* 53(1): 117; 1978. (no refs)

78-2659 **Removal of Acetylaminofluorene from the DNA of Three Repair-deficient Human Cell Lines (Meeting Abstract).** (Eng) Amacher, D. E. (Pfizer Central Res., Groton, CT, 06340). *Mutat Res* 53(1): 117-118; 1978. (no refs)

78-2660 **Sister Chromatid Exchanges (SCE) as Sensitive Indicators of the Effects of Mutagenic Carcinogens (Meeting Abstract).** (Eng) Takehisa, S. (Lab. Radiobiology, Univ. California, San Francisco, CA, 94143); Wolff, S. *Mutat Res* 53(1): 77; 1978. (no refs)

78-2661 **Distribution of Chromosomal Breaks Induced by Three Chemicals (Meeting Abstract).** (Eng) Meyne, J. (Univ. Texas Medical Branch, Galveston, TX, 77550); Rinkus, S.; Legator, M. S. *Mutat Res* 53(1): 80; 1978. (no refs)

78-2662 **The Effect of Mutagenic Treatment on Quantitative Traits in the F_1 and F_2 Generations of Mice (Meeting Abstract).** (Eng) Favor, J. (Georgia Inst. Technology, Atlanta, GA, 30332); Crenshaw, J. W. *Mutat Res* 53(1): 105; 1978. (no refs)

78-2663 **Carcinogenic and Antitumor Effects of Aminotriazole on Acatalasemic and Normal Catalase Mice.** (Eng) Feinstein, R. N. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL, 60439); Fry, R. J.; Staffeldt, E. F. *J Natl Cancer Inst* 60(5): 1113-1116; 1978.

The carcinogenic and antitumor effects of 3-amino-1H-1,2,4-triazole (AT) were evaluated in two inbred mouse strains, C3H and C57BL/6, and their corresponding acatalasemic (a-cat) substrains in order to determine the effect of catalase on tumor susceptibility. Natural tumor incidences, susceptibility to γ-radiation induction (4 weekly doses of 175 rads, 45 rads/

min, from a ^{60}Co source) of thymic lymphomas, and the effect of neutron radiation (1 dose of 80 rads, 15 rads/min) on thyroid tumors were compared in relation to the presence or absence of the mutation at the gene locus for catalase and the effect of AT (1% in the diet ad libitum). Prolonged consumption of an AT diet shortened the lives of all mice, although significant differences existed between strains and substrains. The a-cat mice of both strains survived longer (mean, 35 wk) than their normal catalase (n-cat) counterparts (mean, 25 wk). A larger proportion of the former (21/87), especially those of strain C3H, developed liver tumors that were detected earlier (35 wk) than the n-cat mice (6/85, 50 wk). Thyroid tumors were less common because they required a longer period of development. The antitumor effects of AT included a delay in the appearance of mammary tumors, a striking reduction in γ-radiation induced lymphomas, and a sharp reduction in neutron radiation-induced harderian gland and ovarian tumors. The fact that on the AT diet, C3H a-cat mice developed more liver tumors, starting earlier, than the n-cat substrain indicates a possible relevance of catalase and H_2O_2 in carcinogenesis. The increased incidence of liver tumors in AT-treated a-cat mice was probably due to the diminished rate of degradation of endogenous H_2O_2. (30 refs)

78-2664 **Increased Frequencies of Aberrant Sperm as Indicators of Mutagenic Damage in Mice (Meeting Abstract).** (Eng) Soares, E. R. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC 27709); Sheridan, W.; Segall, M. *Mutat Res* 53(1): 123; 1978. (no refs)

78-2665 **Potentiation of Ribavirin-induced Teratogenesis by Natural Purines.** (Eng) Willhite, C. C. (Dept. Pharmacology/Toxicology, Dartmouth Medical Sch., Hanover, NH, 03755); Ferm, V. H. *Exp Mol Pathol* 28(2): 196-201; 1978.

The effect of the purine bases adenine and guanine on malformations produced in the hamster embryo by ribavirin (RV), an antineoplastic agent, was investigated. On day 8 of gestation, golden hamsters were given ip injections of RV (2.5 or 5.0 mg/kg) alone, either of the purine bases (100 mg/kg) alone, or combinations of RV + adenine and/or guanine. RV produced a broad spectrum of anomalies in the developing hamster, but adenine or guanine produced no gross external developmental malformations. RV + guanine + adenine increased the percentage of resorbed implantation sites and the percentage of malformed hamster fetuses significantly compared with RV alone. The simultaneous injection of 2.5 mg/kg RV + adenine increased only slightly the number of embryonic deaths, but 5.0 mg/kg RV + adenine significantly increased both the number of embryonic deaths and the number of malformed fetuses. A similar experimental protocol with RV + guanine did not result in a highly significant increase in the number of resorptions or the number of malformed hamsters. However, the total number of malformations of the eyes, limbs, and ribs in offspring of hamsters treated with 5.0 mg/kg RV + guanine was greater than the number in fetuses recovered from dams treated with RV alone. The fetal anomalies produced by the RV-purine combinations were generally more severe than those produced by an equivalent amount of RV alone. (10 refs)

78-2666 **Cell Division-dependent Mutagenesis of Yeast by Hydrazine: Loss of Induced Mutability During Reversible Posttreatment Growth Inhibition (Meeting Abstract).** (Eng) Lemontt, J. F. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830). *Mutat Res* 53(1): 85-86; 1978. (no refs)

78-2667 **Genetic Activity of 1,1-Dimethylhydrazine and Methylhydrazine in a Battery of In Vitro and In Vivo Assays (Meeting Abstract).** (Eng) Matheson, D. (Dept. Genetics, Litton Bionetics Inc., Kensington, MD, 20795); Brusick, D.; Jagannath, D. *Mutat Res* 53(1): 93-94; 1978. (no refs)

78-2668 **The Effect of Bran on Dimethylhydrazine-induced Colon Carcinogenesis in the Rat.** (Eng) Barbolt, T. A. (Center Experimental Pathology and Toxicology, Albany Medical Coll., Albany, NY, 12208); Abraham, R. *Proc Soc Exp Biol Med* 157(4): 656-659; 1978.

The role of dietary fiber in dimethylhydrazine (DMH)-induced colon carcinogenesis was investigated in rats. Four groups of 10 male Sprague-Dawley rats each were used: Group I - control; Group II - 30 mg/kg DMH po weekly for 10 doses; Group III - 20% wheat bran in the standard diet; and Group IV - bran and DMH. Intestinal transit times and fecal output were estimated during the 16th, 17th, and 20th weeks. The rats were killed at 21 wk, and the intestinal tract was examined grossly and microscopically for tumors. Intestinal transit times were decreased and fecal outputs increased in rats given the 20% bran diet. In Group II, all 10 rats had tumors, with a mean of 6.4 tumors/rat. In Group IV, 6/9 rats had tumors, with a mean of 1.8 tumors/rat/group and 2.7 tumors/rat that had tumors. The size and distribution of these tumors were similar in both groups. Bran in the diet had little effect on the incidence, number, or distribution of duodenal tumors. The close proximity of the duodenal tumors to the opening of the common bile duct suggests that an active metabolite of DMH was being excreted directly into the bile or that the local high bile salt concentration was acting to promote tumorigenesis in this area. (15 refs)

78-2669 **Contribution of Dietary Fat and Cholesterol to Large Bowel Tumorigenesis or Vascular Lipido-**

sis (Meeting Abstract). (Eng) Broitman, S. A. (Boston Univ. Sch. Medicine, Boston, MA); Vitale, J. J.; Vavrousek-Jakuba, E.; Gottlieb, L. S. *Digestion* 16(3): 234; 1977. (no refs)

78-2670 **Diet and Bile Salt Metabolism in the Aetiology of Carcinoma of the Colon (Meeting Abstract).** (Eng) Castleden, W. M. (Dept. Surgery, Univ. Western Australia, Australia). *Clin Oncol* 4(1): 106; 1978. (3 refs)

78-2671 **Effect of Chemically Defined Dietary Fiber on Rat Colonic Neoplasia Induced by 1,2-Dimethylhydrazine (Meeting Abstract).** (Eng) Freeman, H. J. (Gastroenterology Lab., Veterans Admin. Hosp. and Univ. California, San Francisco, CA); Kim, Y. S.; Spiller, G. A. *Gastroenterology* 74(5, part 2): 1036; 1978. (no refs)

78-2672 **Histogenesis of Symmetrical 1,2-Dimethylhydrazine-induced Neoplasms of the Colon in the Mouse.** (Eng) Chang, W. W. (Dept. Anatomy, Mount Sinai Sch. Medicine, City Univ. New York, One Gustave L. Levy Place, New York, NY, 10029). *J Natl Cancer Inst* 60(6): 1405-1418; 1978.

CF-1 female adult mice were given weekly sc injections of 20 mg/kg symmetrical 1,2-dimethylhydrazine (DMH)·2HCl and killed at intervals of 9-36 wk after the initial injection. ^{3}H-thymidine (TdR) was given before sacrifice. The histogenesis of colon neoplasms was investigated by means of autoradiographs prepared from sections of Epon-embedded descending colon, which were stained with the PAS reaction and iron hematoxylin. By 9 wk after initiation of DMH treatment, the distal 5 cm of the colon was enlarged, the mucosa thickened, and the crypts were elongated and hyperplastic. Although the number of proliferating cells increased in the hyperplastic crypts, the distribution of ^{3}H-TdR-labeled cells in these crypts was similar to that in the normal crypts. Differentiation and transformation of epithelial cells occurred, but somewhat aberrantly. The cryptal hyperplasia was diffuse, but the neoplasms that began to appear by 9 wk after the initial treatment were localized lesions. An isolated crypt from which a neoplasm developed was first repopulated by what appeared to be altered, undifferentiated 'stem' cells. These cells did not differentiate,they continued to divide, and eventually they accumulated in the upper part of the crypts, where the earliest identifiable neoplastic lesion was observed. The early lesions expanded in various directions, depending on the local environments, and formed polypoid or discoid lesions. The biologic behavior of a neoplasm seemed to be determined by the downward progression of its leading edge. When it penetrated the muscularis mucosae, the neoplasm became highly invasive. In the murine model, invasive adenocarcinomas were observed 26 wk after the start of DMH treatment. (43 refs)

78-2673 **Mutagenic and Killing Activities of Two Alkylating and Two Nonalkylating Agents in Heterokaryons of *Neurospora crassa* Homokaryotic for *uvs-2*$^+$ or *uvs-2* (Meeting Abstract).** (Eng) Brockman, H. E. (Illinois State Univ., Normal, IL 61561); Hung, C. Y.; Ong, T. M.; de Serres, F. J. *Mutat Res* 53(1): 81; 1978. (no refs)

78-2674 **Alkyl Methane Sulfonate Mutation of Diploid Human Lymphoblasts and *Salmonella typhimurium*.** (Eng) Hoppe, H. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA, 02139); Skopek, T. R.; Liber, H. L.; Thilly, W. G. *Cancer Res* 38(6): 1595-1600; 1978.

The concentration dependence of mutation in equigenerational exposures to methyl, ethyl, propyl, and butyl methanesulfonate (M-, E-, P-, and BMS) was determined in MIT-2 diploid human fibroblasts and *Salmonella typhimurium*. Forward mutations were measured at the hypoxanthine-guanine phosphoribosyltransferase locus in human lymphoblasts and at the putative guanine phosphoribosyltransferase locus in *S. typhimurium*. Reverse mutations at the *his* G46 locus were also measured in *S. typhimurium*. Studies with the human cells indicated that there was decreasing toxicity with increasing chain length. The concentration required to reduce the surviving fraction to 0.10 was 0.05 mM for MMS, 0.44 mM for EMS, 1.2 mM for PMS, and 1.4 mM for BMS. A similar relationship between mutagenic potential and chain length was observed for EMS, PMS, and BMS; the MMS mutant fraction increased with concentration initially, but the extreme toxicity prevented accurate determination of the mutant fraction. With *S. typhimurium*, the concentration necessary to reduce the surviving fraction to 0.10 was 10 mM for MMS and >20 mM for BMS, PMS, and EMS. For both MMS and EMS, concentration dependence differed significantly from that expected from single-hit kinetics on a target of fixed size. In the reverse mutation studies, the concentration required to induce a mutant fraction approx tenfold over controls was 5 mM for MMS, 10 mM for EMS, 30 mM for PMS, and 10 mM for BMS. This study and previous reports support the conclusion that *S. typhimurium* and mammalian cells are essentially equisensitive to the mutagenic effects of EMS when concentration and exposure time are taken into account. The forward and reverse mutation assays in *S. typhimurium* revealed no important differences in sensitivity among the four compounds. (39 refs)

78-2675 **Mutagenicity of the Fungicide Dexon (Meeting Abstract).** (Eng) Rogers, S. J. (Montana State Univ., Bozeman, MT, 59715); Skaar, P. D.; Tindall, K. R.; Vyse, E. R.; Warren, G. *Mutat Res* 53(1): 91; 1978. (1 ref)

78-2676 **Inhibiting Effect of Caffeine on Spontaneous and Urethan-induced Lung Tumors in Strain A**

Mice. (Eng) Theiss, J. C. (Dept. Community Medicine, Sch. Medicine, Univ. California at San Diego, La Jolla, CA, 92093); Shimkin, M. B. *Cancer Res* 38(6): 1757-1761; 1978.

The anticarcinogenic effect of caffeine on the development of spontaneous and urethan-induced lung tumors in strain A mice was investigated. Ip injections of 8, 20, or 40 mg/kg caffeine thrice weekly for 8 wk suppressed spontaneous pulmonary adenoma development. They also suppressed urethan-induced (0.25 and 1.0 mg/g sc) lung tumor development when they were begun 1 wk before urethan administration, but this suppression was not significant when caffeine treatment was initiated 1 wk after urethan injection. The most pronounced suppression of lung tumor formation occurred when caffeine was given as only two injections 3 hr before and 3 hr after urethan administration. The incorporation of ^{3}H-thymidine into lung tissue DNA of caffeine-treated mice was impaired at the time of urethan administration. Caffeine also partially antagonized the effects of urethan on lung tissue. It is suggested that caffeine-induced suppression of DNA synthesis interferes with pulmonary adenoma induction by decreasing the affinity of lung tissue DNA for urethan. The finding that chronic caffeine treatment produced continued suppression of thymidine incorporation into lung tissue DNA suggests that caffeine-induced inhibition of spontaneous pulmonary adenoma formation is due to a general suppression of lung DNA synthesis. (18 refs)

78-2677 **Cytogenetic Analysis of Mice Before and after Urethane Treatment (Meeting Abstract).** (Eng) Cumming, R. B. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Sotomayor, R. E.; Lewis, L. R.; Green, S. *Mutat Res* 53(1): 127; 1978. (no refs)

78-2678 **Alkali-labile Lesions, DNA Synthesis, and Mutagenesis in Cultured Mammalian Cells (Meeting Abstract).** (Eng) Peterson, A. R. (McArdle Lab., Univ. Wisconsin, Madison, WI, 53706); Heidelberger, C. *Mutat Res* 53(1): 118-119; 1978. (no refs)

78-2679 **In Vivo Determination of Alkylation of Amino Acids in Hemoglobin and Urine to Detect Potential Mutagenic-Carcinogenic Effects (Meeting Abstract).** (Eng) Truong, B. L. (Univ. Texas Medical Branch, Galveston, TX, 77550); Legator, M. S. *Mutat Res* 53(1): 121; 1978. (no refs)

78-2680 **In Vitro Plasmid DNA Breakage by Mutagens and Pesticides (Meeting Abstract).** (Eng) Griffin, D. E. (Univ. Southwestern Louisiana, Lafayette, LA, 70504); Hill, W. E. *Mutat Res* 53(1): 87; 1978. (no refs)

78-2681 **Histological Examination of Perinatal Eye Development in the Rat after Ingestion of Sodium Cyclamate and Sodium Saccharin During Pregnancy.** (Eng) Luckhaus, G. (Institut fur Toxikologie der Bayer AG, Postfach 101709, D5600 Wuppertal 1, W. Germany); Machemer, L. *Food Cosmet Toxicol* 16(1): 7-11; 1978.

Pregnant female Wistar rats were fed a diet containing 5% sodium cyclamate or 0.4% sodium saccharin for 20 days after mating. The daily ingested dose was 1,495 mg for the cyclamate animals, 98.8 mg for the saccharin group. Examination of the fetuses on day 20 of pregnancy or the young at 3 wk of age revealed no histological changes in the lens, vitreous body, insertion of the optic nerve, or other parts of the eye. (11 refs)

78-2682 **The Fate of Saccharin Impurities. The Excretion and Metabolism of [^{14}C]Toluene-4-sulphonamide and 4-Sulphamoyl[^{14}C]benzoic Acid in the Rat.** (Eng) Ball, L. M. (Pharmacology Branch, NIEHS, Research Triangle Park, NC, 27709); Williams, R. T.; Renwick, A. G. *Xenobiotica* 8(3): 183-190; 1978.

The fate of ^{14}C-labeled toluene-4-sulfonamide (TS) and 4-sulfamoylbenzoic acid (SBA), byproducts of saccharin synthesis by the Remsen-Fahlberg process, was determined in female albino Wistar rats. The rats were given 29 or 200 mg/kg TS or 22 mg/kg SBA by intragastric intubation. Approx 94% of the SBA dose was eliminated within 24 hr. By 6 days, 73%-83% of the dose was recovered in the urine, but significant amounts (18%-32%) were eliminated in the feces because of incomplete absorption. The ^{14}C in the urine and feces was unchanged SBA. No labeled CO_2 was detected in the expired air. Following TS administration, 66%-89% of the dose was rapidly eliminated in the urine, with only 2%-8% being eliminated in the feces. The label in the feces was SBA; it probably originated in the tissues, since the gut flora could not effect this biotransformation. The urine of rats given TS contained SBA as the major metabolite (93% of the urinary label), together with small amounts of unchanged TS (1.5%-2.3% of the label), 4-sulfamoylbenzyl alcohol (2.0%-3.9%), 4-sulfamoylbenzaldehyde (0%-1.5%: at the lower dose only), and N-acetyltoluene-4-sulfonamide (2.1%-2.3%: at the higher dose only). Thus, prolonged intake of saccharin containing traces of TS and SBA should not lead to their accumulation in the body, providing that the larger amounts of saccharin do not interfere with the oxidation of TS to SBA or the renal elimination of the latter. (14 refs)

78-2683 **Methyl Methanesulphonate Mutagenesis in L5178Y Mouse Lymphoma Cells.** (Eng) Cole, J. (M.R.C. Cell Mutation Unit, Univ. Sussex, Falmer, Brighton BN1 9QG, England); Arlett, C. F. *Mutat Res* 50(1): 111-120; 1978.

The effect of short (2 hr) and long (20 hr) treatment times on methyl methanesulfonate (NMS) mutagenesis was determined in murine lymphoma L5178 cells. At time 0 posttreatment, treatment of the cells for 20 hr with 0.048 mM MMS reduced cloning efficiency to 74.0%; treatment for 2 hr with 0.12, 0.24, 0.48, and 0.96 mM MMS reduced cloning efficiency to 99.6%, 90.2%, 47.5%, and 1.5%, respectively. Recovery, measured by cloning efficiency at 24-hr intervals after treatments that inflicted >25% lethality, was delayed for 72-96 hr. There was no detectable cell multiplication for 72 hr in the population with a 98.5% level of kill. There was a dose-dependent induction of thioguanine-resistant (TG-r) and thymidine-resistant (TdR-r) variants, but not ouabain-resistant (Oua-r) variants. TdR-r variants were expressed much earlier (48-96 hr) than TG-r variants (144 hr). (24 refs)

78-2684 **Systems Development for Mutagenicity Testing; Syrian Hamster Embryo Cells in Bulk Cell Culture Vessels (Meeting Abstract).** (Eng) Carver, J. H. (Lawrence Livermore Lab., Livermore, CA, 94550); Wandres, D. L.; Branscomb, E. W. *Mutat Res* 53(1): 97; 1978. (no refs)

78-2685 **Reversion of Carcinogen-induced Cytoplasmic Abnormalities (Mallory Bodies) in Rat Liver Cells (Meeting Abstract).** (Eng) Borenfreund, E. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Higgins, P. J. *In Vitro* 14(4): 347; 1978. (1 ref)

78-2686 **Post-Treatment with Caffeine and the Induction of Gene Mutations by Ultraviolet Irradiation and Ethyl Methanesulphonate in V-79 Chinese Hamster Cells in Culture.** (Eng) van Zeeland, A. A. (Dept. Radiation Genetics and Chemical Mutagenesis, State Univ. Leiden, Wassenaarseweg 72, Leiden, Netherlands). *Mutat Res* 50(1): 145-151; 1978.

A determination was made of the effect of caffeine posttreatment on the induction of 6-thioguanine-resistant mutants in V-79 Chinese hamster cells by UV radiation and ethyl methanesulfonate (EMS). Caffeine (1 mM) potentiated the killing effect of UV radiation and EMS. However, it did not alter the induction of mutations at the hypoxanthine-guanine phosphoribosyltransferase locus by UV radiation (up to a fluence of 4 joules/m^2) or by EMS (up to a concentration of 50 mM). The results are consistent with the theory that caffeine partially inhibits an error-prone postreplication repair mechanism without changing the mutation frequency. (26 refs)

78-2687 **The Association Between Mutagenicity and Adduct Formation of 1,2,7,8-Diepoxyoctane and 1,2,5,6-Diepoxycyclooctane.** (Eng) Huang, S. L. (Lab. Envi
ronmental Mutagenesis, Natl. Inst. Environmental Healt
Sciences, NIH, Research Triangle Park, NC, 27709); Rader
D. N.; Lee, C. Y. *Chem Biol Interact* 20(3): 333-340; 1978

The mutagenicity of 1,2,5,6-diepoxycyclooctane (DECO
and 1,2,7,8-diepoxyoctane (DEO) was investigated in diploi
Chinese hamster lung cells. 6-Thioguanine (6-TG) resistanc
was the marker for mutagenicity testing: DEO was genetica
ly active, but DECO was totally inactive. The frequencies c
DEO-induced mutations to 6-TG resistance were dose-de
pendent. DEO readily formed adducts with radiolabeled nu
cleotides, as demonstrated by thin-layer chromatography an
the shift of the UV absorption max in DEO/nucleotide mix
tures. There was no formation of adducts between radionu
cleotides and DECO. The difference between the chemica
and genetic activities of the two compounds was attribute
to their molecular conformations and the resulting differen
tial flexibilities and adduct-forming abilities. The associatio
between mutagenicity and adduct formation was conclusive
(25 refs)

78-2688 **Squamous Lesions in Lungs of Rats Exposed t
Tobacco-Smoke-Condensate Fractions by Re
peated Intratracheal Instillation.** (Eng) Simons, P. J. (Hazle
ton Labs. Europe Ltd., Otley Road, Harrogate HG3 1PY
England); Lee, P. N.; Roe, F. J. *Br J Cancer* 37(6): 965-97
1978.

Twice-weekly intratracheal instillations in specific-path
gen-free Wistar rats of up to 24 mg of the tobacco smok
condensate caffeine-complexing fraction [Fraction (R
P)G] suspended in either infusine (I) or buffered saline/gel
tin (BS/G) gave rise to foci of squamous metaplasia of alve
lar epithelium (SqM) and squamous neoplasms (SqN). Fra
tion (R + P) G, which is almost as tumorigenic for mous
skin as the nearly 30 times larger mass of condensate fro
which it is derived, could be given in this way for up to
wk without excessive mortality or any marked effect on th
rate of body-wt gain. By contrast, similar treatment wit
Fraction N(QG), a fraction having very low tumorigenic a
tivity for mouse skin, induced no SqN and barely an exce
of SqM over that induced by either vehicle alone. The effec
of Fraction (R + P)G on the incidence of SqM and SqN we
both time- and dose-related, the effect on SqM incidence b
ing evident after 10 wk of treatment. No SqN seen we
unequivocally malignant; however, because of the design
the experiment, only 5 rats exposed to Fraction (R + P)
were observed >60 wk after the start of the experiment. Ot
er changes in the lung, including aggregates of alveolar ma
rophages laden with golden-brown pigment and foci of cubo
dal/columnar metaplasia of alveolar epithelium, we
frequently seen in response to both fractions. Fraction (R
P)G administered in I was more effective in causing SqM an
SqN than the same fraction administered in BS/G. The in
plications of the findings are discussed, particularly the pos
bility that the intratracheal instillation technique might

seful as a rapid bioassay for comparing the tumorigenicity f different cigarette smoke condensates. (13 refs)

8-2689 **The Mutagenicity of Cigarette Smoke Condensate in *Saccharomyces cerevisiae* (Meeting bstract).** (Eng) DeMarini, D. M. (Illinois State Univ., ormal, IL, 61561). *Mutat Res* 53(1): 84; 1978. (no efs)

8-2690 **Chemical Studies on Tobacco Smoke-LIV Determination of Hydroxybenzyl Alcohols and Hydroxyphenyl Ethanols in Tobacco and Tobacco Smoke.** Eng) Hecht, S. S. (Naylor Dana Inst. Disease Prevention, merican Health Foundation, Valhalla, NY, 10595); Carmella, S.; Hoffmann, D. *J Anal Toxicol* 2(2): 56-59; 1978.

ince 2-(p-hydroxyphenyl)-ethanol (PHPE) and m-hydroxybenzyl alcohol (MHBA) produced hyperplasia in mouse skin nd thus could contribute to the tumor-promoting and cocarcinogenic activity of cigarette smoke condensate, a method was developed for their quantitation in tobacco and tobacco moke. The method consists of solvent partitions followed by preparative thin-layer chromatography and gas-liquid hromatography of the silylated hydroxyphenyl alcohol fraction. The major source of PHPE in mainstream smoke was ransferred from tobacco, but MHBA was formed, at least partially, from cellulose. (14 refs)

78-2691 **Fractionation of Cigarette Smoke Condensate for Chemical and Biological Testing.** (Eng) Walters, D. B. (Natl. Inst. Environmental Health Services, Environmental Biology and Chemistry Branch, P.O. Box 12233, Research Triangle Park, NC, 27709); Chamberlain, W. J.; Akin, F. J.; Snook, M. E.; Chortyk, O. T. *Anal Chim Acta* 99(1): 143-150; 1978.

Gel filtration and silicic acid column chromatography were used to separate the three fractions that account for most of the tumorigenicity of cigarette smoke condensate (CSC): the ether-soluble weak-acid fraction (F-8); the polycyclic aromatic hydrocarbon (PAH) fraction (F-55); and a polar, neutral lipid fraction (F25 + 26). Biological activity was concentrated in subfractions containing significantly smaller percentages of initial CSC wt, and various groups of compounds were concentrated into individual subfractions, facilitating chemical and biological analyses. F-8 was separated into four subfractions containing fatty acids, phenols, polyphenols, phenolic acids, and other high-mol-wt materials. F-55 was separated into two subfractions, one of which contained essentially pure PAH's (0.01% by wt of CSC). F-25 + 26 was separated into three subfractions containing glycolipids, sterol glucosides, and other high-mol-wt materials. Mouse-skin-painting experiments showed that F-8 had significant tumorigenic activity (48.5% tumor bearers). A subfraction of F-8, in which catechol was concentrated and which comprised 3.27% CSC, was also tumorigenic (30.7% tumor bearers), as was a combination of this subfraction, the active PAH subfraction, and a neutral lipid subfraction. The results indicate that catechol may be a potent cocarcinogen and that the PAH's in CSC interact with other components to exert a tumorigenic effect. (12 refs)

78-2692 **In Vitro Morphological Transformation of Cryopreserved Hamster Embryo Cells with Tobacco Tar.** (Eng) Takayama, S. (Dept. Experimental Pathology, Cancer Inst., Kami-Ikebukuro 1-327-1, Toshima-ku, Tokyo 170, Japan); Katoh, Y.; Hirakawa, T.; Tanaka, M. *Gann* 69(1): 85-90; 1978.

The in vitro transformation of cryopreserved line 7602 hamster embryo cells by tobacco tar and its hexane (NA-1), benzene (NA-2), ethyl acetate (NA-3), and methanol (NA-4) acidic or neutral fractions and its ether (B-1) and methanol (B-2) basic fractions was investigated. Line 7602 was chosen over six other hamster embryo cell lines, as it had the best response to transformation by 3-methylcholanthrene. Neither the tobacco tar nor any of its fractions were cytotoxic at doses of 0.5 to 5.0 μg/ml. A significant number of transformed colonies were induced by NA-4 at levels of 1 and 5 μg/ml. The tobacco tar and fractions NA-2, NA-3, and B-2 also induced a significant number of transformed colonies. The tar and all four fractions have been reported to be potent mutagens in bacterial systems. Fractions NA-1 and B-1 did not show any transforming or mutagenic activity. These findings indicate that a close correlation exists between in vitro morphological transformation and bacterial mutagenicity. (16 refs)

78-2693 **Protection of Hamster Lung Cultures by L-Cysteine or Vitamin C Against Carcinogenic Effects of Fresh Smoke from Tobacco or Marihuana Cigarettes.** (Eng) Leuchtenberger, C. (Dept. Cytochemistry, Swiss Inst. Experimental Cancer Res., Rue du Bugnon 21, 1011 Lausanne, Switzerland); Leuchtenberger, R. *Br J Exp Pathol* 58(6): 625-634; 1977.

Hamster lung cultures, when grown in normal medium (NM) and exposed to fresh smoke from tobacco or marihuana cigarettes for 1-8 wk, displayed cytotoxic alterations, loss of lysosomes, and abnormalities of cell division and chromosome DNA content. Cultures grown in NM in the presence of L-cysteine showed an increase of lysosomes and significantly less abnormalities in cell division and DNA content of chromosomes. Cultures grown in NM and exposed to smoke for 9-24 wk showed rapid irregular growth of fibroblasts and/or malignant transformation, but cultures grown in L-cysteine-containing NM and exposed to smoke closely resembled nonexposed controls. Addition of vitamin C to NM had an effect similar to that of L-cysteine. Cultures grown in NM that had undergone malignant transformation continued their abnormal growth during the 2-yr test period. When these transformed cultures were grown for 3 mo or longer in the presence of L-cysteine or vitamin C, growth became more normal, and the cultures closely resembled those of nonexposed controls. These results suggest that, in hamster lung cultures, addition of L-cysteine or vitamin C protects against or reverses abnormal growth and malignant transformation occurring after repeated exposure to smoke. The increase of lysosomes after L-cysteine or vitamin C was added to the media suggests the possible importance of lysosomes in protecting the cultures against the enhancement of carcinogenesis by smoke. (25 refs)

78-2694 **Tobacco-specific Nitrosamines: Formation from Nicotine In Vitro and During Tobacco Curing and Carcinogenicity in Strain A Mice.** (Eng) Hecht, S. S. (Div. Environmental Carcinogenesis, Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY, 10595); Chen, C. B.; Hirota, N.; Ornaf, R. M.; Tso, T. C.; Hoffmann, D. *J Natl Cancer Inst* 60(4): 819-824; 1978.

The formation of tobacco-specific nitrosamines from nicotine, the major tobacco alkaloid, was examined. Detached leaf tobacco was fed either labeled nicotine or nornicotine and air-cured. The cured leaf was then examined for labeled N′-nitrosonornicotine (NNN). The yield of NNN was 0.007% from nornicotine and 0.009% from nicotine. Because the ratio of nicotine to nornicotine in conventional nicotine-type tobacco is 20-100:1, nicotine is considered to be the major precursor of NNN in tobacco. The formation of other nitrosamines from nicotine in vitro was then investigated. Reaction of nicotine with $NaNO_2$ gave rise to NNN, 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and 4-(N-methyl-N-nitrosamino)-4-(3-pyridyl)butanal (NNA). An analysis of market products revealed 0.6-2.4 μg NNK/g chewing tobacco and snuff. The three compounds were then tested for carcinogenicity in female A/J mice. The animals were given ip injections of 22 mg of the compounds three times weekly for 22 injections. NNK in trioctanoin induced lung adenomas in 20/23 animals, compared with 16/21 for NNN in saline, 12/23 for NNN in trioctanoin, and 9/25 for NNA in saline. Furthermore, two cases of undifferentiated carcinoma of the salivary glands occurred in the NNN group. Since the hazardous compounds were formed during curing, methods should be developed to reduce their concentrations in both tobacco and smoke. (32 refs)

78-2695 **Nitrate and Gastric Cancer (Meeting Abstract).** (Eng) Tannenbaum, S. R. (Massachusetts Inst. Technology, Cambridge, MA). *Digestion* 16(3): 279; 1977. (no refs)

78-2696 **Hypothesis for the Aetiology of Gastric Cancer (Meeting Abstract).** (Eng) Ruddell, W. S. (Dept. Gastroenterology, Central Middlesex Hosp., London, England); Hill, M. J.; Walters, C. L. *Digestion* 16(3): 272; 1977. (no refs)

78-2697 **Nitrite Studies in Oesophageal Cancer.** (Eng) Lowenfels, A. B. (Westchester County Medical Center, Valhalla, NY); Tuyns, A. J.; Walker, E. A.; Roussel, A. *Gut* 19(3): 199-201; 1978.

Salivary and gastric nitrite levels were studied in 20 patients with esophageal cancer and 15 healthy controls. A basal saliva sample was obtained prior to the ingestion of 100 ml of beet juice containing 160 mg nitrate; a second saliva sample were obtained 90 min later. Basal nitrite levels were significantly higher in controls (115 μM) than in cancer patients (48 μM). Following administration of the beet juice, the levels increased significantly in both groups (913 and 674 μM, respectively), and the difference between the groups lost its significance. The mean pH of the gastric juice of seven patients with a gastronomy was 3.5, and the mean gastric juice nitrite concentration was 1.3 μM. Since both groups had similar ability to convert nitrate to nitrite in the saliva, it is suggested that this factor is less important for the in vivo formation of nitrite than the amount of nitrate in the diet. (14 refs)

78-2698 **Organotropic Effect of N-Bis(2-hydroxypropyl)nitrosamine: Production of Lung and Liver Tumors by Its Oral Administration in Mice.** (Eng) Konishi, Y. (Dept. Oncological Pathology, Cancer Center, Nara Medical Univ., 840 Shijo-cho, Kashihara, Nara-ken 634, Japan); Kondo, H.; Inui, S.; Denda, A.; Ikeda, T.; Kojima, K. *Gann* 69(1): 77-84; 1978.

The organotropic effect of N-bis(2-hydroxypropyl)nitrosamine (DHPN), a postulated metabolite of dipropylnitrosamine, was studied in male dd mice. The mice were divided into five groups: Group 1 served as controls, and Groups 2-5 received 100, 250, 500, or 1,000 ppm DHPN, respectively, in the drinking water. The animals were sacrificed 16 wk after the beginning of the experiment. Each mouse in Groups 2-5 ingested 6.3, 13.9, 22.4, and 34.2 mg DHPN/day, respectively. Lung tumors were noted in 10/11 Group 2 animals (10 adenomas, 1 adenocarcinoma), 9/11 Group 3 animals (adenomas), 8/11 Group 4 animals (8 adenomas, 2 adenocarcinomas), and 8/14 Group 5 animals (8 adenomas, 1 adenocarcinoma). Liver tumors were noted in 8 Group 2 animals (hemangiomas), 8 Group 3 animals (hemangiomas), 8 Group 4 animals (8 hemangiomas and 8 hemangioendotheliomas), and 14 Group 5 animals (3 hemangiomas, 3 hemangioendotheliomas, 8 hemangioendothelial sarcomas). No lung or liver tumors were found in Group 1 animals. Furthermore, no pancreatic tumors, which have been found in hamsters, were found. These results are similar to those previously reported for rats and guinea pigs. (27 refs)

78-2699 **Bladder Tumors Induced in Dogs by N-butyl -N- (4-hydroxybutyl)-nitrosamine — Scanning Electron Microscopic Observation (Meeting Abstract).** (Jpn) Hirao, Y. (Nara Medical Univ., Kashihara, Nara pref., 634, Japan); Kondoh, T.; Yamada, K.; Babatani, K.; Ohara, S.; Aoyama, H.; Watanabe, H.; Hashimoto, M.; Okajima, E. *Jpn J Urol* 68(9): 864-865; 1977. (no refs)

78-2700 **Effects of Sex Hormones on Oncogenesis in Rat Urinary Bladder by N-Butyl-N-(4-hydroxybutyl)-nitrosamine.** (Eng) Kono, N. (Dept. Urology, Tokyo Electric Power Hosp., 9-2, Shinanomachi, Shinjuku-ku, Tokyo, Japan); Tanahashi, T.; Suzawa, N.; Azuma, C. *Int J Clin Pharmacol Biopharm* 15(3): 101-105; 1977.

The effects of sex hormones on N-butyl-N-(4-hydroxybutyl)-nitrosamine (BBN)-induced carcinogenesis in the bladders of male Wistar-Imamichi rats were evaluated. Eight-week-old rats were divided into five treatment groups: (1) 30 ml drinking water supplemented with 0.02 mg/head/day of BBN for 8 wk; (2-4) BBN + testosterone propionate, estradiol benzoate, or estriol tripropionate, the hormones given in a dose of 3 mg/kg/wk im for 20 wk; (5) untreated. The incidence of hyperplasia in the respective groups was 2/25, 2/23, 5/23, 3/24, and 0/25. The corresponding incidence of bladder tumors was 6/25, 9/23, 2/23, 2/24, and 0/25. There were statistically significant differences in bladder tumor incidence between Groups 2 and 3 and between Groups 2 and 4. It is concluded that external sex hormones can influence experimental oncogenesis in the male rat bladder. (11 refs)

78-2701 **Promoting Effect of DL-Tryptophan and Saccharin in Urinary Bladder Carcinogenesis in the Rat (Meeting Abstract).** (Eng) Cohen, S. M. (Dept. Pathology, St. Vincent Hosp., Worcester, MA, 01504); Arai, M.; Friedell, G. H. *Proc Am Assoc Cancer Res* 19: 4; 1978. (no refs)

78-2702 **Induction of Transitional Cell Carcinoma of the Urinary Bladder in Rats by Feeding N-[4-(5-Nitro-2-furyl)-2-thiazolyl]formamide. Histological and Ultrastructural Findings.** (Eng) Adolphs, H. D. (Dept. Urology, Univ. Bonn, D-5300 Bonn-Venusberg, W. Germany); Thiele, J.; Kiel, H.; Steffens, L. *Urol Res* 6(1): 19-27; 1978.

Female Wistar rats were fed a diet containing 0.188% N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT) for 8 mo, at an average FANFT consumption of 0.195 g/wk. One animal was sacrificed each month for investigation by light and electron microscopy, and the remaining animals were killed after 12 mo. There was a 100% incidence of bladder tumors in the rats after 8 mo on the FANFT-containing diet. The first definite alterations of the mucosa were apparent after 5-6 mo of FANFT feeding. Histological changes of the urothelium consisted of various degrees of hyperplasia and dysplasia. An infiltrating transitional cell carcinoma first appeared after 8 mo. Electron microscopy showed microvillous transformation of the luminal plasma membrane and the appearance of a thick fluffy cell coat (glycocalyx). These changes are explained by an altered function of the Golgi complex that occurs during malignancy and leads to a loss of the specific discoid vesicles of the urothelial cell. The electron microscopic findings showed that the transitional cell carcinoma induced by FANFT is identical to the papillary carcinoma of man, indicating the usefulness of this model for studying new methods of therapy in papillary carcinoma of the urinary bladder. (44 refs)

78-2703 **Differences in Binding of Nitrosamines to Cytochrome P 450. Possible Relevance for Their Activation and Inactivation (Meeting Abstract).** (Eng) Appel, K. E. (Dept. Molecular Toxicology, German Cancer Res. Center, D-6900 Heidelberg, W. Germany); Kunz, H. W. *Naunyn Schmiedebergs Arch Pharmacol* 302(Suppl): R20; 1978. (no refs)

78-2704 **Cytochrome P-450 and the Activation of Aflatoxin B$_1$ in *Saccharomyces cerevisiae* (Meeting Abstract).** (Eng) Callen, D. F. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709); Philpot, R. M.; Ong, T. M. *Mutat Res* 53(1): 85; 1978. (no refs)

78-2705 **Aryl Hydrocarbon Hydroxylase Activity and Microsomal Cytochrome Content of Human Fetal Tissues.** (Eng) Rifkind, A. B. (Dept. Pharmacology, Cornell Univ. Medical Coll., New York, NY, 10021); Tseng, L.; Hirsch, M. B.; Lauersen, N. H. *Cancer Res* 38(6): 1572-1577; 1978.

The suitability of prostaglandin (PG) abortuses for the study of mixed function oxidase activity was examined. Aryl hydrocarbon hydroxylase (AHH) was used as an index of mixed-function oxidation and as an indirect indicator of tissue capacity to activate polycyclic hydrocarbons and microsomal cytochrome content. Tissues were obtained from human fetuses aborted by (a) ia PG F$_2\alpha$, (b) im PG 15-methyl-F$_2\alpha$, (c) vaginal suppositories containing PG E$_2$ (20 mg every 2 hr until abortion), (d) intravaginal Silastic devices containing PG 15 (S)-15-methyl-$F_2\alpha$ (11 mg), and (e) hysterotomy performed for medical reasons. Cytochrome P-450 concentrations and AHH activity were approx 4 times higher in adrenal glands than in liver. AHH levels in testes, ovaries, vagina, and uterus were greater than or equal to those in the liver, but levels in lung, kidney, and intestines were lower than those in the liver. Mean hepatic AHH and hepatic and adrenal cytochromes P-450 and b_5 were not significantly different in PG and hysterotomy abortuses. Mean adrenal AHH was significantly lower in PG abortuses (95.1 vs 228.4 picomoles phenol/mg protein/hr for hysterotomy), but the ranges in both groups were overlapping. Neither maternal cigarette smoking habit nor fetal sex affected hepatic or adrenal AHH activity or microsomal cytochrome concentrations or difference spectra. Hepatic and adrenal AHH activities were concentrated in microsomes and were correlated with microsomal P-450 content. These findings are consistent with P-450 mediation of AHH in human fetal tissues. The data demonstrate the utility of PG abortuses for studies of fetal tissue mixed-function oxidase activity and point to the endocrine glands and target tissues in addition to the liver as potential sites for activating chemical carcinogens and cytotoxins in the human fetus. (35 refs)

78-2706 High Mutagenicity of N-(α-Acyloxy)alkyl-N-alkylnitrosamines in *S. typhimurium*: Model Compounds for Metabolically Activated N,N-Dialkylnitrosamines. (Eng) Camus, A. M. (Unit Chemical Carcinogenesis, International Agency Res. Cancer, 69008 Lyon, France); Wiessler, M.; Malaveille, C.; Bartsch, H. *Mutat Res* 49(2): 187-194; 1978.

Several N,N-dialkylnitrosamines monosubstituted at the α-carbon with an acetoxy group were tested for their mutagenic activity in *Salmonella typhimurium* TA1530 in the presence or absence of an S-9 rat liver mix. In the absence of S-9 enzymes, the mono-α-acetoxy derivatives of N-ethyl-N-methylnitrosamine and N,N-diethylnitrosamine gave nonlinear dose-response curves, but those of N,N-di-n-propylnitrosamine and N,N-di-n butylnitrosamine increased the number of his+ revertants up to a concentration of approx 100 nanomoles (nmol)/plate. At 50 nmol/plate, N-(α-acetoxy)ethyl-N-methylnitrosamine induced 73 his+ revertants/nmol, but the ethyl, propyl, and butyl compounds produced 4, 19, and 26 his+ revertants/nmol, respectively. Dose-response curves for the mutagenicity of N-(acyloxy)-methyl-N-alkylnitrosamine in the absence of S-9 were of the same shape when the alkyl group consisted of a methyl, ethyl, n-propyl, or n-butyl residue. Thus, the mutagenicity of these compounds was inversely related to their half-lives in aqueous media. N-(Acetoxy)methyl-N-tert-butylnitrosamine and a β-acetoxy derivative of N,N-diethylnitrosamine were not mutagenic either in the presence or in the absence of S-9. Thus, α-carbon hydroxylation is one mechanism involved in the metabolic activation of N,N-dialkylnitrosamines. (2
refs)

**78-2707 Effect of Choline Administration on the Toxic
ty of N-Nitrosodimethylamine in Female Rat**
(Eng) Pani, P. (Istituto di Patologia Generale, Via Porcell
09100 Cagliari, Italy); Columbano, A.; Dessi, S.; Porcu, M
Ledda, G. M.; Diaz, G. *Toxicol Appl Pharmacol* 42(3): 61
616; 1977.

The effect of choline administration on the acute toxicity
N-nitrosodimethylamine (DMN) was studied in female rat
A 0.5% aqueous soln of choline was given po at a dos
of 500 mg/kg bid for 3 wk. DMN was injected ip
doses of 40 or 120 mg/kg, and the animals were kil
ed 24 hr or 90 min after intoxication, respectivel
Hepatic necrosis, evaluated by the release of SGP
activity in the serum, was significantly enhanced b
the choline administration. This was confirmed b
morphological observations and by morphometric analyse
of hepatic nuclei. Polysomal disaggregation induced b
DMN was also augmented by choline administration. Cho
line also increased the activity of DMN-demethylas
The results indicate that choline increases DMN toxicit
in female rats, probably through an enhancement
microsomal activities. (14 refs.)

**78-2708 Carcinogenicity and Acute Toxicity of Dime
thylnitrosamine in Rainbow Trout (*Salm*
gairdneri).** (Eng) Grieco, M. P. (R. T. French Co., On
Mustard St., Rochester, NY, 14609); Hendricks,
D.; Scanlan, R. A.; Sinnhuber, R. O.; Pierce, D.
J Natl Cancer Inst 60(5): 1127-1131; 1978.

A chronic dose-response study was made of the ca
cinogenicity of dimethylnitrosamine (DMN) in rainbow trou
(*Salmo gairdneri*), and the results were compared with thos
of a similar study in Porton rats. Trout were also subjecte
to acute exposures to determine the LD50. At the age of
wk, the trout were fed diets containing 0, 3, 50, 200, 400, an
800 ppm DMN for 52 wk, followed by a control, DMN-fr
diet for an additional 26 wk. Thirty trout were sampled
26 and 52 wk, the remainder at 78 wk, to determine tum
incidence. With the six dietary levels, the av total DMN do
per trout was 0, 0.72, 47.8, 45.6, 96.4, and 195.9 mg. At
wk, the respective tumor incidences were 0/60, 0/60, 2/
4/60, 20/60, and 44/60; at 78 wk, these incidences were 0/7
1/75, 14/79, 15/75, 36/73, and 57/63. Thus, the carcinoge
ic response was dose-related. Rats and trout were appr
equal in their sensitivity to DMN carcinogenesis. The LD
after ip injection of DMN was 1,770 mg/kg body wt in t
trout. Relative to the range of 15-50 mg/kg reported for se
eral mammalian species, the trout were resistant to the acu
toxicity of DMN. (11 refs)

78-2709 **Levamisole Is Without Effect in Modifying Chemically Induced Renal Carcinogenesis.** (Eng) Hard, G. C. (Dept. Pathology, Univ. Melbourne Medical Centre, Grattan St., Parkville, Victoria, Australia 3052). *Cancer Lett* 3(5/6): 221-226; 1977.

The effect of levamisole (2,3,5,6-tetrahydro-6-phenylimidazole [2,1-b] thiazole monohydrochloride) on renal tumors induced in female Porton albino Wistar rats by dimethylnitrosamine (DMN: 45 mg/kg ip) was investigated. Levamisole (10 mg/kg) was administered in six sc injections at 3- to 4-day intervals 1-3 wk or 9-12 wk after DMN treatment. Levamisole had no modulating effect on the number of tumor-bearing rats, the incidence of different types of renal tumor, tumor multiplicity, survival time, or tumor size. There was a small number of metastases in both the DMN-only and DMN + levamisole groups. (27 refs)

78-2710 **Effect of a Single Injection of Different Doses of Diethylnitrosamine on the Hepatic Nuclear Fractions in Rats of Various Lines.** (Ukr) Grinchishin, V. P. (Inst. Oncological Problems, Kiev, USSR); Bikoriz, A. I.; Shumilina, V. V. *Dop Akad Nauk Ukr RSR [B]* (12): 1117-1121; 1977.

Diethylnitrosamine (DEN)-induced changes in the hepatic nuclear fraction were studied in random-bred albino rats and Wistar rats. The animals received a single ip injection of DEN: 100 or 250 mg/kg to random-bred rats, 100 mg/kg to Wistar rats. They were sacrified at various time intervals after injection, and the proliferative activity in the hepatic cells was assessed. The ratio of diploid to tetraploid cells in Wistar rats was max (approx 7.5) 12 hr after DEN injection, and it remained increased for at least 3 days. In random-bred rats inoculated with 250 mg/kg, the ratio was increased on day 1 after injection (2.6, compared with 1.92 in rats inoculated with 100 mg/kg and 1.78 in controls), but random-bred rats inoculated with 100 mg/kg showed an increase in the ratio on day 2 after injection (3.6). (2 refs)

78-2711 **In Vitro Demonstration of Mallory Body Formation in Liver Cells from Rats Fed Diethylnitrosamine.** (Eng) Borenfreund, E. (Lab. Cell Biochemistry, Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Bendich, A. *Lab Invest* 38(3): 295-303; 1978.

Epithelioid liver cells were established in culture from rats sacrificed 10-18 wk after administration of the hepatocarcinogen diethylnitrosamine in their drinking water (4 mg/kg/day). After 3 mo in vitro, more than half the cells propagated from rats bearing hepatocellular carcinomas had bean-shaped, acentrically displaced nuclei with large juxtanuclear homogeneous-appearing areas resembling the hyalin or Mallory bodies in the livers of chronic alcoholics. These abnormalities were not seen in the livers of origin, but they were retained in the carcinomas that formed after cultured cells containing such inclusions were injected ip into young Wistar rats or id into nude (thymusless) Swiss mice. Transmission electron microscopy showed that the hyaline bodies in the cultured tumor cells consisted of a disorganized meshwork of filaments. Examination by incubating tumor cells with cytochalasin B and by using antiactin and anti-DNase antisera as indirect immunofluorescence probes also revealed a disturbance in the cytoskeleton. Such an alteration could influence cell movement, metabolism, and transport and could, therefore, be pivotal in the transformation of cells. (46 refs)

78-2712 **Gamma-Glutamyl Transpeptidase: A Positive Marker for Cultured Rat Liver Cells Derived from Putative Premalignant and Malignant Lesions.** (Eng) Laishes, B. A. (McArdle Lab. for Cancer Res., Univ. Wisconsin, Madison, WI, 53706); Ogawa, K.; Roberts, E.; Farber, E. *J Natl Cancer Inst* 60(5): 1009-1016; 1978.

Use of the histochemical localization of γ-glutamyl transpeptidase (γ-GT) as a specific positive marker for rat hepatocytes (H) was assessed in cells derived from: (1) premalignant foci induced by diethylnitrosamine (DEN) and 2-acetylaminofluorene (2-AAF); (2) an early-passage, tumorigenic liver cell line (T50) from a 2-AAF-treated rat; and (3) an established malignant hepatoma cell (HTC) line. High γ-GT levels were located in it from macroscopic, carcinogen-induced lesions at every stage of hepatocarcinogenesis tested. H from normal-looking liver surrounding carcinogen-induced, early lesions were γ-GT-negative. In vitro, γ-GT activity was often localized as spots or bands in the H, and γ-GT-positive (γ-GT+) bile ducts were easily seen. After continuous exposure of rats to 2-AAF or DEN, γ-GT-positive H appeared in vitro only when they were derived from livers containing γ-GT-positive micro- or macroscopic lesions. With early lesions, the relative proportions of γ-GT+ H were greater when the cells were derived from individual lesions rather than from total liver, but they increased in total liver isolates when the lesions increased in size. Grey-white hyperplastic nodules yielded primary cultures containing 29.0%-44.0% γ-GT+ H, with no selective attachment of γ-GT+ H to the cell culture vessels. No γ-GT + H was observed in primary cultures derived from (1) normal adult F344 rats, (2) rats subjected to two-thirds partial hepatectomy, or (3) rats subjected to acute doses of DEN or dimethylnitrosamine. γ-GT activity could not be induced in normal H by exposing them to various carcinogens in vitro, and no γ-GT+ proliferating cells could be isolated from any of the stages of hepatocarcinogenesis except obvious hepatocellular carcinoma. (26 refs)

78-2713 The Carcinogenic Effect of Dimethylnitrosamine in Laboratory Bred European Hamsters (*Cricetus cricetus*). (Eng) Richter-Reichhelm, H. B. (Abteilung fur Experimentelle Pathologie, Medizinische Hochschule Hannover, Karl-Wiechert-Allee 9, 3000 Hannover 61, W. Germany); Green, U.; Ketkar, M. B.; Mohr, U. *Cancer Lett* 4(1): 1-4; 1978.

The carcinogenic effect of dimethylnitrosamine (DMN: injected sc once weekly for life at 0.025 or 0.05 the LD_{50}) was studied in laboratory-bred European hamsters, and the data were compared to findings for wild European hamsters. No significant difference could be established between the two dose levels as to survival time and tumor incidence. The first tumor, in the liver, appeared 22 wk after the start of DMN treatment. An almost 100% incidence had developed by the 45th-50th weeks. No differences as to the latency period for these tumors or their localization could be determined between the two treatment groups. Malignant hemangioendotheliomas of the liver and kidney, hepatocellular carcinomas, and cholangiocarcinomas were induced. In contrast to the results obtained with wild European hamsters, a large number of the laboratory-bred females developed ovarian granulosa cell tumors. There was also a greater number of hepatic tumors in laboratory-bred animals. These findings show that differences in maintenance and environment may influence the carcinogenic effect of a given compound. (7 refs)

78-2714 Procarcinogenic Compounds: Problem of Activation in Rapid Testing. (Rus) Belitsky, G. A. (Cancer Res. Center, Moscow, USSR); Khovanova, E. M.; Logvinenko, E. G. *Vestn Akad Med Nauk SSSR* (10): 80-82; 1977.

A new rapid method for screening chemical pro-carcinogens involves the detection of induced somatic mutations in *Drosophila melanogaster* from the cross (female Y/Yx male Wsn^3/Y). These compounds can be activated easily in these organisms to induce a high level of somatic variegation. Several N-nitroso derivatives and their noncarcinogenic analogs were added to the medium. All compounds induced somatic mutations, but the mutagenic activity of the carcinogens was significantly higher than that of their noncarcinogenic analogs. High mutagenic activity was detected not only for the direct carcinogen N-methyl-N′-nitro-N-nitrosoguanidine, but also for the procarcinogens N-nitroso-N,N-dimethylamine and N-nitroso-N,N-diethylamine. (7 refs.)

78-2715 Loss of Two-Stage-Control of Cell Proliferation in Rat Liver After a Single Dose of the Carcinogen Diethylnitrosamine (Meeting Abstract). (Eng) Hermann, R. S. (Institut fur Toxikologie und Pharmakologie, Philipps Universitat Marburg/Lahn, Pilgrimstein 2, D 355 Marburg, W. Germany). *Naunyn Schmiedebergs Arch Pharmaco* 302(Suppl): R21; 1978. (1 ref)

78-2716 Metabolism of Dimethylnitramine by NZR Rat Liver (Meeting Abstract). (Eng) Goodall, C. M. (Cancer Res. Unit, Dept. Surgery, Univ. Otago, Otago, New Zealand); Bryson, W. G.; Kennedy, T. H. *NZ Med J* 86(595): 246; 1977. (no refs)

78-2717 Metabolism and Subcellular Effects of Dimethylnitrosamine Incubated in Vitro with Isolated Liver Cells (Meeting Abstract). (Eng) Schwarz, M. (Dept. Molecular Toxicology, German Cancer Res. Center, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany); Rickart, R. *Naunyn Schmiedebergs Arch Pharmaco* 302(Suppl): R21; 1978. (no refs)

78-2718 Detection of DNA Damage Induced In Vivo Following Exposure of Rats to Carcinogens. (Eng) Petzold, G. L. (Pathology and Toxicology Res. Unit, Upjohn Co., Kalamazoo, MI, 49001); Swenberg, J. A. *Cancer Res* 38(6): 1589-1594; 1978.

A modified alkaline elution assay was used to detect DNA damage in various rat tissues following in vivo exposure of the animals to several classes of carcinogens. Newborn rats were inoculated ip with ^{3}H-thymidine (total, 150 μCi over 3 wk) DNA and then exposed to the carcinogens 1-3 wk later. DNA damage was assessed in the liver, lung, thymus, brain, kidney, stomach, duodenum, colon, bone marrow, and mammary gland. Dimethylnitrosamine (DMN), diethylnitrosamine, 4-nitroquinoline 1-oxide (4-NQO), 2-acetylaminofluorene, N-hydroxyacetylaminofluorene, aflatoxin B_1, 1,2-dimethylhydrazine, azoxymethane, benzidine-HCl, and 7,12-dimethylbenz(a)anthracene induced the greatest extent of DNA damage in target organs for carcinogenicity. Exposure to direct-acting alkylating agents such as N-methyl N′-nitro-N-nitrosoguanidine, N-methyl-N-nitrosourea, N ethyl-N-nitrosourea, methyl methanesulfonate, and ethy methanesulfonate caused significant DNA damage i target and non-target tissues. Dose-related increase in the DNA elution patterns were observed. DNA re pair time, as measured by the return to a normal elu tion pattern, varied from as little as 24 hr for 4-NQ to as long as 7 days for DMN. It is concluded that thi assay is a rapid and reliable method for assessing th ability of a compound to induce DNA damage. (3 refs)

78-2719 Saccharide Alterations in Rat Kidney Associated with Malignant Transformation by Injectio

of Dimethylnitrosamine. (Eng) Whyte, A. (Dept. Pathology, Univ. Cambridge, Tennis Court Road, Cambridge CB2 1QP, England); Loke, Y. W. *Br J Cancer* 37(5): 689-700; 1978.

Cell-surface glycoprotein alterations and saccharide distributions were analyzed in normal rat kidney and renal tumors induced in diet-primed Wistar albino rats by injection of dimethylnitrosamine (DMN: 60 mg/kg ip). (Rats were fed a diet of sucrose and water for the 7 days before DMN injection.) Control and tumor tissue was excised at different periods up to 9 mo after DMN injection and maintained in short-term organ culture in the presence of ^{3}H- or ^{14}C-fucose. The plasma membranes were then isolated, and the isotopic profiles of normal kidney and renal tumor membrane proteins were established, using polyacrylamide gel electrophoresis in dodecyl sulfate. The levels of several fucose-containing glycoproteins of the plasma membranes were altered upon neoplastic transformation: four increased and three decreased. The probable identity of two of these proteins was determined: α-fetoprotein increased whereas neutral endopeptidase decreased in the tumor membranes. Fluorescein-labeled lectin binding by the kidney tissue also was altered upon transformation. The most marked changes were an increase in sialic acid (neuraminidase-sensitive) and galactosamine (*Ricinus communis* agglutinin Type I) in the nuclei of some neoplastic cells and some hyperplastic tubule cells. The nuclear fluorescence of the cells of the DMN-induced renal tumor apparently did not involve the nucleoplasm itself, but was restricted to the nuclear membrane. It may be that alterations of the glycosylated molecules of the nuclear membrane are a common feature of neoplasia. (40 refs)

78-2720 **Lethal and Mutagenic Effects of 4-Nitroquinoline-1-oxide in UV-Sensitive Strains of *Neurospora crassa* (Meeting Abstract).** (Eng) Inoue, H. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709); Ong, T. M.; de Serres, F. J. *Mutat Res* 53(1): 82-83; 1978. (no refs)

78-2721 **Differences in the Repair Activity in the Cells of Inbred Mice after Exposure to Chemical Carcinogen 4-Nitroquinoline-1-Oxide.** (Rus) Zasukhina, G. D. (Inst. General Genetics, Moscow, USSR); Sinel'shchikova, T. A.; L'vova, G. N. *Dokl Akad Nauk SSSR* 238(4): 959-962; 1978.

Repair of 4-nitroquinoline -1- oxide-induced DNA breaks was assayed to evaluate the sensitivity of CBA, C57BL/6, and 101/H murine fibroblasts to carcinogen induced damage. There was a max decrease in the mol wt of DNA in the 101/H fibroblasts and a minimal decrease in the CBA fibroblasts. The intensity of DNA degradation was dose-dependent, and the effectiveness of DNA repair was exposure time-dependent. (9 refs)

78-2722 **In Vitro Studies of Chemical Mutagens and Carcinogens: Stability Properties in Cell Culture Medium (Meeting Abstract).** (Eng) Jensen, E. M. (EG and G/Mason Res. Inst., Rockville, MD, 20852); LaPolla, R. J.; Kirby, P. E.; Haworth, S. R. *Mutat Res* 53(1): 90; 1978. (no refs)

78-2723 **Differential Killing Between Repair-deficient and Repair-proficient Human Cell Lines After Exposure to Carcinogens: Application as a Screening Test (Meeting Abstract).** (Eng) Burrell, A. D. (General Products Div., IBM Corporation, San Jose, CA, 95193); Andersen, J. J.; Kay, E. S. *Mutat Res* 53(1): 94-95; 1978. (no refs)

78-2724 **Ultrastructural Hepatic Changes in Rats after Oral Administration of Ethoxyquin (EMQ).** (Eng) Nafstad, I. (Dept. Pharmacology and Toxicology, Veterinary Coll. Norway, Postboks 8146, Oslo-Dep, Oslo 1, Norway); Skaare, J. U. *Toxicol Lett* 1(5/6): 295-299; 1978.

Ultrastructural changes were determined in the livers of male albino rats after they were administered a single po dose of 500 mg/kg ethoxyquin (EMQ). The rats were sacrificed 3 days later. There was a proliferation of smooth endoplasmic reticulum in the parenchymal cells. Mitochondrial alterations were of minor severity, and they consisted of deformation and derangement of the cristae. Irregular dilation of the perinuclear space was a common occurrence. Alterations in the lining cells appeared to be more severe than those in the parenchymal cells. Dilation of the endoplasmic reticulum and distorted mitochondria were the most striking observations. In some areas, the lining cells were desquamated and fragmented, the cell fragments being intermingled with blood cells in the sinusoids. In another group of rats that had received 150 mg EMQ/liter drinking water throughout an experimental period, no ultrastructural changes were noted. (5 refs)

78-2725 Carcinogenic Effects of Niridazole (an Antischistosomal Drug) on the Kidneys of MRC Rats (Meeting Abstract). (Eng) Bulay, O. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NB 68105); Shubik, P. *Proc Am Assoc Cancer Res* 19: 180; 1978. (no refs)

78-2726 **Production of Adenocarcinomas of the Small Intestines of Wistar Rats Fed Panfuran-S Containing 3-Di(hydroxymethyl)amino -6-(5-nitro -2-**

furylethenyl)-1,2,4-triazine. (Eng) Konishi, Y. (Dept. Oncological Pathology, Cancer Center, Nara Medical Univ., 810 Kashihara, Nara 634, Japan); Denda, A.; Takahashi, S.; Inui, S.; Aoki, Y. *J Natl Cancer Inst* 60(6): 1339-1343; 1978.

When fed to outbred Wistar rats, the antimicrobial agent panfuran-S (PS) produced a high incidence of invasive and metastatic adenocarcinomas of the duodenum and jejunum. Depressed growth and cancer incidence were dependent on the amount of 3-di(hydroxymethyl)amino-6-(5-nitro-2-furylethenyl)-1,2,4-triazine (DHNT) in the contained PS. Duodenal adenocarcinomas were present in 2/18 rats that received 1,750 ppm DHNT in the diet and in 11/19 rats that received 3,500 ppm DHNT. Jejunal adenocarcinomas were present in 10/18 and 16/19 rats that received 1,750 and 3,500 ppm DHNT, respectively, for 35-37 wk. The only other tumors observed were squamous cell papillomas of the forestomach. Histologically, most of the adenocarcinomas appeared tubular or papillary, and they were similar to adenocarcinomas in the human small intestine and also to human intestinal type adenocarcinomas of the stomach. This system provided a useful experimental model for studying the pathogenesis of small intestinal carcinoma. (26 refs)

78-2727 **Nitrofurazone-induced DNA Damage to Tissues of Mice.** (Eng) Olive, P. L. (Radiobiology Section, Johns Hopkins Oncology Center, 601 N. Broadway, Baltimore, MD, 21205). *Chem Biol Interact* 20(3): 323-331; 1978.

Cytotoxicity and DNA damage by nitrofurans has previously been correlated with metabolic reduction of these drugs in vitro. In the present study, nitrofurazone (NF) increased the rate of disappearance of stable ^{3}H-thymidine-labeled DNA from tissues of Swiss albino mice fed 0.1% NF in the diet. A significant loss of labeled DNA occurred within 25 days of the start of the diet in all tissues observed, and the loss was related to the rate of metabolic reduction of NF. A similar correlation was found when another end point for DNA damage was used; NF reduced by mouse tissue slices caused DNA single-strand breaks in cultured mouse L cells incubated in vitro with the tissues. Again, the ability of each tissue to produce toxic NF metabolites determined the amount of DNA damage to the L cells. (23 refs)

See also:

*(Rev.): 78-2402, 78-2403, 78-2404, 78-2405, 78-2406
78-2408, 78-2409, 78-2410, 78-2411, 78-2412
78-2413, 78-2414, 78-2415, 78-2416, 78-2417
78-2418, 78-2420, 78-2421, 78-2422, 78-2423
78-2424, 78-2425, 78-2426, 78-2427, 78-2428
78-2429, 78-2430, 78-2432, 78-2433, 78-2435
78-2437, 78-2438, 78-2440, 78-2447, 78-2449
78-2452, 78-2458, 78-2461, 78-2463, 78-2464
*(Phys.): 78-2737, 78-2738, 78-2741, 78-2746
78-2748, 78-2749, 78-2754.
*(Viral): 78-2768, 78-2813, 78-2834, 78-2874, 78-2891
*(Immun.): 78-2910, 78-2911, 78-2912, 78-2913
78-2914, 78-2916.
*(Path.): 78-2948, 78-2951, 78-2953, 78-2955
78-2956, 78-2957.
*(Epid.-Biom.): 78-2966, 78-2967, 78-2969, 78-2975

78-2728 **Some Effects of Irradiation of Mice in Utero with Tritiated Compounds.** (Eng) Lambert, B. E. (Radiobiol. Dept., Med. Coll. St. Bartholomew's Hosp., London, England); Phipps, M. L. *Curr Top Radiat Res* 12(1): 197-211; 1977.

SAS/4 female mice were exposed in utero to tritiated thymidine (^{3}H-TdR: infused continuously into the mother). For comparison, tritiated water (HTO: via maternal drinking water) was included in the study as a source of internal homogeneous irradiation. In both cases the labeling patterns and subsequent loss of tritium over an extended period were studied. The technique of infusion in unrestrained mice and its application in the production of fully ^{3}H-TdR labeled offspring is described in detail. In HTO-exposed mice, rapid fetal growth did not unduly influence tritium incorporation in utero, and the av organ dose from organically bound tritium was small relative to the dose from tritiated water. The fully labeled mice are being used to study a number of early and late effects, in particular, gonad cell effects and carcinogenesis, following this form of internal irradiation. Some preliminary results are presented. (10 refs)

78-2729 **Comparative Incorporation of Tritium from Tritiated Water Versus Tritiated Thymidine, Uridine or Leucine.** (Eng) Mewissen, D. J. (Dept. Radiology, Univ. Chicago, Chicago, IL); Furedi, M.; Ugarte, A.; Rust, J. H. *Curr Top Radiat Res* 12(1): 225-254; 1977.

Newborn, juvenile, and young adult C57BL mice were inoculated sc or ip with 1 or 10 μCi/g tritiated water (HTO), ^{3}H-thymidine, ^{3}H-leucine, or ^{3}H-uridine. The animals were sacrificed 1, 8, 15, 22, or 29 days posttreatment, and ^{3}H incorporation was assessed in the aqueous fraction and in the organic component of tissues from organs such as the thymus, spleen, lung, liver, brain, and kidney. Following HTO administration, free water ^{3}H activity decreased exponentially with time in all animals. Organically bound (OB) activity was consistently higher in juvenile vs newborn animals and in male vs female adults. After administration of the tritiated precursors, ^{3}H was transferred to the FW pool at a higher rate than the one that occurred following the administration of HTO, which suggested a steady rate of exchange between the OB compartment and the FW pool. RSA values (the ratio of the specific activity of OB ^{3}H to that of aqueous ^{3}H, corrected for relative abundance of hydrogen in either pool) ranged from 0.5 to $<$ 10 after HTO administration, but they decreased a thousandfold after 28 days. Realistic estimates of the internal dose to a radiosensitive nucleus must take into account the age-dependent incorporation of ^{3}H from HTO, as well as the variation between organs. Implications of the results for carcinogenesis from internal irradiation are discussed. (50 refs)

78-2730 **Distribution of Tritiated Compounds (Tritiated Thymidine and Tritiated Water) in the Mother-Fetus System and its Consequences for the Radiotoxic Effect of Tritium.** (Eng) Schreml, W. (Dept. Clinical Phys., Univ. Ulm, D-7900 Ulm-Donau, W. Germany); Fliedner, T. M.; Ohnander, J.; Zipfel, L. *Curr Top Radiat Res* 12(1): 255-277; 1977.

Tritiated thymidine (^{3}H-TdR) and tritiated water (HTO) incorporation and distribution were measured in newborn Wistar rats exposed to the compounds by continuous infusion into pregnant rats from day 9 until term. The tritium levels administered were 0.8 and 1.6 μCi/g/day ^{3}H-TdR and 8 and 16 μCi/g/day HTO. In HTO-exposed animals, tritium activity was distributed homogeneously but ^{3}H-TdR led to the accumulation of DNA-bound and homogeneously distributed tritium. The incorporated activity and the specific activity of DNA from the ovaries, which exhibited an approx 50% reduction in total oocyte number, were used to estimate the dose absorbed by ovarian cell nuclei in both systems. From the absorbed dose, a factor of 3.7 was calculated for the internal relative biological effectiveness of DNA-bound tritium, compared with homogeneously distributed ^{3}H, under the restrictive assumption that the static system at birth reflects the situation during the time of dynamic development of the ovaries, when the toxic effect occurs. The influence of these dynamic factors of changing nuclear size and tritium incorporation during the sensitive period was weighed against the possibility that continuous ^{3}H-TdR infusion during pregnancy might represent a model in which DNA-bound tritium shows a higher effectiveness than homogeneously distributed tritium. (31 refs)

78-2731 **The Genetic and Late Somatic Effects of Chronic Tritium Ingestion in Mice.** (Eng) Carsten, A. L. (Medical Res. Center, Brookhaven Natl. Lab., Upton, Long Island, NY, 11973); Commerford, S. L.; Cronkite, E. P. *Curr Top Radiat Res* 12(1-4): 212-224; 1977.

The long-term somatic and genetic effects of chronic tritium (^{3}H) ingestion were measured in Hale-Stoner-Brookhaven mice. Second-generation animals, who with their parents were maintained on drinking water containing 3 μCi/ml HTO, were bred for dominant lethal testing. The increase in dominant lethal mutations in treated animals compared with tap water controls, was significant at the 1% level. The stem cell content of the bone marrow was measured throughout the lifetime of the animals. A slight reduction in colony-forming units in the bone marrow of treated animals was evident after approx 12 wk, increasing in severity throughout the lifetime of the animals. The distribution of ^{3}H in tissue water protein fractions was determined, and the relative contribution of this ^{3}H to tissue dose was calculated. The differential incorporation into protein fractions was not significant. (13 refs)

78-2732 **Chromosome Aberrations in Human Lymphocytes Exposed to Tritiated Water In Vitro.** (Eng) Bocian, E. (Inst. Nuclear Res., Dorodna 16, 03-195 Warsaw, Poland); Ziemba-Zak, B.; Rosiek, O.; Sablinski, J. *Curr Top Radiat Res* 12(1): 168-181; 1977.

The induction of chromosome aberrations in human peripheral blood lymphocytes by tritiated water (HTO) or 180 kV x-rays was studied in vitro. The lymphocytes were exposed to 1.70-14.36 mCi/ml HTO for 2 hr or to 0.063-0.51 mCi/ml for 53 hr. Chromosomes and chromatid aberrations were scored during the first mitotic division after stimulation with phytohemagglutinin. For analysis of the dose-response relationship, the data were fitted by the least-square method to different models. After acute exposure to tritium β-rays and x-rays, the dicentrics + centric rings and terminal + interstitial deletions gave the best fit to the linear-quadratic function. However, data for these types of aberrations after 53 hr exposure to HTO gave an equally good fit to the linear and linear-quadratic functions. The best description of the dose-response relationship for chromatid aberrations was given by the linear model. The relative biological effectiveness of tritium β-rays compared to 180 kV x-rays was 1.17 ± 0.02. (25 refs)

78-2733 **Tritium Retention in Rat After Administration of Various Doses of Tritiated Water.** (Eng) Radwan, I. (Central Lab. Radiological Protection, Warsaw, Poland); Pietrzak-Flis, Z.; Jaworowski, Z. *Curr Top Radiat Res* 12(1): 278-290; 1977.

Four groups of hooded inbred rats were inoculated sc with tritiated water (HTO) at the following doses: Group I (controls), 1.8 μCi/g; Group II, 25 μCi/g; Group 3, 50 μCi/g; Group 4, 115 μCi/g. Samples of liver, lung, skin, muscle, and intestinal tissue were collected at 1-, 3-, and 5-day intervals over a 21-day period. Free-water and tissue-bound HTO concentrations were determined, and the biological half-life (Tb) of tritium was calculated by the least-squares method. The Tb of free-water tritium varied from 3.5 days in kidney to 4.8 days in muscle in Group I animals; the values were slightly lower in the higher-dose groups. The retention of tissue-bound tritium varied in each group, with Tb values ranging from 10.3 days in the large intestine to 85.0 days in the muscle of control rats. The values decreased significantly at higher HTO doses. This result is the opposite of the effect observed after external x-ray irradiation at the same dose level. (7 refs)

78-2734 **The Early Retention of Monomeric ^{237}Pu(IV)- and ^{239}Pu(IV)-Citrate in C57BL/Do and BALB/cJ Mice.** (Eng) Smith, J. M. (Radiobiology Lab., Univ. Utah Coll. Medicine, Salt Lake City, UT, 84112); Taylor, G. N.; Mays, C. W.; Atherton, D. R. *Radiat Res* 73(3): 502-509; 1978.

The early retention (6 hr to 28 days postinjection) of plutonium(IV) citrate was followed in C57BL/Do and BALB/cJ mice. As a tracer, the photon-emitting nuclide, ^{237}Pu, was mixed with α-emitting ^{239}Pu, so that Pu retention could be determined by standard NaI(Tl) crystal scintillation spectroscopy. Mice were inoculated ip with 0.3-0.5 μCi ^{239}Pu/kg containing 0.5-1.0 μCi ^{237}Pu/kg. The BALB/cJ mice showed essentially the same early retention of Pu as the C57BL/Do mice. By 12 hr, the females had excreted more than twice as much of the injected Pu as the males. Since these and previous studies establish that the sex difference for Pu retention in mice is reproducible and exists in at least two strains, further experiments were conducted. Six male and six female C57BL/Do were gonadectomized (GD) 3-4 wk prior to injection, and 10 male BALB/Cj mice were GD 2 wk prior to injection. Retention of Pu by GD females did not differ significantly from that of intact females, but Pu retention by GD males resembled that of the females. When ^{237}Pu(IV)- and ^{239}Pu(IV)-citrate was given iv to intact male and female BALB/cJ mice, the sex difference in Pu retention was less pronounced. Physiological factors, other than specific protein binding, may be responsible for the sex difference. However, if the transport of Pu out of the peritoneal cavity and its ultimate distribution and retention are determined in some way by cellular activity or by specific protein binding, either path may be under the influence of sex hormones. (17 refs)

78-2735 **Blood Lymphocyte Chromosome Aberrations in Plutonium Workers (Meeting Abstract).** (Eng Brandom, W. (Univ. Denver, Denver, CO, 80210); Bloom A.; Archer, P.; Bistline, R. *Mutat Res* 53(1): 79; 1978. (no refs)

78-2736 **Gynecologic Morbidity in Women Exposed Occupationally to Radioactive Substances.** (Rus Muratova, R. M. (All-Union Res. Inst. Social Hygiene and Organization of Health Care, Moscow, USSR); Chusova, V N. *Med Radiol (Mosk)* (7): 52-55; 1978.

Gynecologic morbidity was studied in a group of 100 women who had been exposed occupationally to radioactive substances (isotopes of radium, thorium). Patients wer divided into two groups: Group 1 included 23 women who received a total of 100-400 roentgens-equivalent-man (rem) of external irradiation, and Group 2 included 77 women who received total of 30-100 rem. The overall incidence of gynecologic diseases in Groups 1 and 2 was 73.8% and 67.1%, respectively; 12.6% of the women in Group 1 and 7.7% in Group 2 had uterine myomas. Several years prior to the development of myoma, most of the patients had transient hypoestrogenemia (8.97-24.13 μg/day). (14 refs)

78-2737 **Interactions Between Polonium-210 α-Radiation, Benzo(a)pyrene, and 0.9% NaCl Solution Instillations in the Induction of Experimental Lung Cancer.** (Eng) Little, J. B. (Dept. Physiology, Harvard Sch. Public Health, Boston, MA, 02115); McGandy, R. B.; Kennedy, A. R. *Cancer Res* 38(7): 1929-1935; 1978.

Benzo(a)pyrene (BP) and ^{210}Po were administered both simultaneously and sequentially by intratracheal instillations to Syrian golden hamsters in experiments designed to identify any synergistic interactions (SI) between these carcinogens. There was significant SI when BP exposure followed 5 mo after ^{210}Po exposure and the effects were additive after simultaneous administration. The implications of these findings in terms of the interaction between α-radiation and cigarette smoke in human populations are discussed. (28 refs)

78-2738 **Changing Levels of UV Light and Carcinogen-Induced Unscheduled DNA Synthesis in Mouse Oocytes During Meiotic Maturation.** (Eng) Brazill, J. L. (Dept. Zoology, Univ. Toronto, Ontario M5S 1A1, Canada); Masui, Y. *Exp Cell Res* 112(1): 121-125; 1978.

Mouse oocytes were exposed in vitro to three DNA-damaging agents at the germinal vesicle (GV), metaphase I (MI), or metaphase II (M II) stage of meiotic maturation and then labelled with ^{3}H-thymidine for 2.0 hr. The levels of unscheduled DNA synthesis induced in the oocytes at the different stages of maturation by the agents were ranked in the following order: GV > M-I = M-II when exposed to UV light; GV > M-I > M-II when exposed to 4-nitroquinoline-1-oxide (4-NQO); GV = M-I = M-II when exposed to methyl methanesulfonate (MMS). When the oocytes were exposed to UV light or 4-NQO at the M-II stage, the unscheduled DNA synthesis levels in the polar bodies were considerably lower than those in the oocytes. However, MMS exposure at the M-II stage resulted in the same levels of unscheduled DNA synthesis in the polar bodies and the oocytes. Factors that may account for both the variable levels of unscheduled DNA synthesis at the different stages, as well as the differential response of the oocytes and the polar bodies, are discussed. (15 refs)

78-2739 **Ultraviolet Light-induced Sister Chromatid Exchanges in Xeroderma Pigmentosum and in Cockayne's Syndrome Lymphocyte Cell Lines.** (Eng) Cheng, W. S. (Medicine Branch, NCI, NIH, Bethesda, MD, 20014); Tarone, R. E.; Andrews, A. D.; Whang-Peng, J. S.; Robbins, J. H. *Cancer Res* 38(6): 1601-1609; 1978.

Sister chromatid exchanges (SCE) were studied in lymphocyte cell lines derived from 11 patients with xeroderma pigmentosum (XP; representing all 6 forms of XP) and 2 patients with Cockayne's syndrome (CS). XP patients have DNA repair defects and develop numerous tumors on sun-exposed skin; CS patients do not develop such tumors, and the cause of the sensitivity of their cells to UV light is not known. Reciprocal SCE were evaluated by fluorescence microscopy following staining of the chromosomes with the bisbenzimidazole dye 33258 Hoechst after the cells had passed through two phases of DNA synthesis in the presence of 5-bromodeoxyuridine. In the absence of UV irradiation, XP and CS cells had the same frequency of spontaneous SCE as did normal cells. UV irradiation resulted in significant increases in the SCE of normal, XP, and CS cells. The number of UV-induced SCE in an XP variant's cells was not greater than the number in normal cells. The number of UV-induced SCE in XP complementation group B and E cells and in the CS cells was higher than that in normal cells, but the difference was not significant. All XP complementation group A, C, and D cells tested had significantly greater numbers of UV-induced SCE than did normal cells. There were significant differences in the number of UV-induced SCE among cells from unrelated XP group C patients, but cells from three XP group C siblings had almost identical UV-induced responses. In normal and XP cells, there was a significantly greater number of spontaneous and UV-induced SCE in the longer chromosomes than in the shorter chromosomes. (47 refs)

78-2740 **The Effect of 8-Methoxypsoralen Plus UV-Light on the Frequency of Viral Transformation by Polyoma Virus In Vitro (Meeting Abstract).** (Eng) Morhenn, V. B. (Dept. Dermatology, Stanford Univ. Sch. Medicine, Stanford, CA); Kaye, J. A. *J Invest Dermatol* 70(4): 215; 1978. (no refs)

78-2741 **Ultraviolet Carcinogenesis in Mice and the Effect of Oral 8-Methoxypsoralen (8-MOP) (Meeting Abstract).** (Eng) Pathak, M. A. (Dept. Dermatology, Harvard Medical Sch., Boston, MA); Molica, S. J. *Clin Res* 26(3): 300A; 1978. (no refs)

78-2742 **Prolonged UV-Erythema and Light-induced Cancer (Meeting Abstract).** (Eng) Klostermann,

G. (Dept. Dermatology, City Hosp., Mannheim, W. Germany); Bohnert, E.; Jung, E. G. *Arch Dermatol Res* 261(1): 87-88; 1978. (1 ref)

78-2743 **Mechanisms of the Carcinogenic Effect of UV Radiation.** (Ita) Feo, F. (No affiliation given). *Minerva Med* 69(12): 770-773; 1978.

Epidemiological evidence shows that there is a carcinogenic effect of UV radiation on exposed skin areas. According to a recent hypothesis, malignant transformation is initiated during the repair of the UV-damaged DNA and not by the DNA damage itself. (no refs)

78-2744 **Evaluating Individual Differences in Unscheduled DNA Synthesis in Normal Human Fibroblasts (Meeting Abstract).** (Eng) DeLozier, C. D. (Methodist Hosp., Indianapolis, IN, 46205); Antley, R. M. *Mutat Res* 53(1): 109; 1978. (no refs)

78-2745 **Sister Chromatid Exchanges in Lymphocytes from Psoriasis Patients Treated with 8-Methoxypsoralen and Longwave Ultraviolet Light.** (Eng) Lambert, B. (Dept. Clinical Genetics, Karolinska sjukhuset, S-104 01 Stockholm 60, Sweden); Morad, M.; Bredberg, A.; Swanbeck, G.; Thyresson-Hok, M. *Acta Derm Venereol (Stockh)* 58(1): 13-16; 1978.

The frequency of sister chromatid exchanges (SCE) was determined in the peripheral blood lymphocytes of 14 psoriasis patients (7 men, 7 women aged 14-63 yr) before, during, and after treatment. Each patient received 0.6 mg/kg 8-methoxypsoralen (8-MOP) po and, 2.0-2.5 hr later, longwave UV light (UVA) radiation using black light fluorescent lamps with a spectral max at 360 nanometers. Irradiation usually lasted 20 min, during which time a total dose of approx 3 joules/cm^2 was delivered. The treatment was given 5 days per week for 3-30 wk, depending on the healing progress. The frequency of SCE was approx the same before and after treatment. In another part of the experiment, in vitro UVA irradiation of cells from patients who had taken 8-MOP 2.0-2.5 hr before blood sampling increased SCE frequency: a dose of 1.8 joules/cm^2 increased the av number of SCE/cell by 24% and 3.6 joules/cm^2 increased it by 54%, compared with nonirradiated controls. There was no increase after systemic 8-MOP tretament alone or after UVA irradiation alone. Thus, po 8-MOP and UVA increase the number of SCE in peripheral lymphocytes in vitro, but clinical UVA skin irradiation in vivo does not produce this effect. It is possible that the accumulated UVA dose in circulating lymphocytes is not large enough to produce SCE or that the DNA damage caused by this treatment is repaired during the treatment intervals. (17 refs)

78-2746 **Elucidation of the Nature of Genetic Damage Formed in the Presence of the Sunscreening Agent, Para-amino Benzoic Acid, During Irradiation with Near Ultraviolet Light (Meeting Abstract).** (Eng) Hodges, N. D. (Sch. Pharmacy, Univ. Bath, Avon, England); Moss, S. H.; Davies, D. J. *J Pharm Pharmacol* 29(Suppl): 72P; 1977. (1 ref)

78-2747 **Repair in the Cells Infected with Viruses.** (Eng) Dubinin, N. P. (Inst. General Genetics, USSR Academy Sciences, Moscow, USSR); Andzaparidze, O. G.; Zasukhina, G. D. *Stud Biophys* 66(2): 121-127; 1977.

Three cellular systems differing in DNA repair activity were investigated: human diploid cells and cells infected by leukosis virus; commercial and leukosis-free chick embryo cells; and primary and established Syrian hamster kidney (SHK) cells. At 24 hr post-UV irradiation, no excision of thymine dimers in the DNA was observed in the virus-infected human cells, commercial chick embryo cells latently infected with avian leukosis virus, or the two established SHK lines. Nitrosoguanidine significantly enhanced the number of virus-induced chromosome aberrations in virus-infected diploid cells, and, in contrast to γ irradiation, it induced unrepairable DNA damage. In the infected chick embryo cells, DNA repair was observed after γ irradiation but not after ethyleneimine treatment. This chemical also increased the number of chromosome abberations. Repression of repair capacity and an enhancement of chromosome aberrations may be an initial mechanism in the malignant transformation of a normal cell. (no refs)

78-2748 **Induction and Kinetics of In Vitro Transformation of Syrian Hamster Cells (Meeting Abstract).** (Eng) DiPaolo, J. A. (Biology Branch, NCI, Bethesda, MD, 20014). *In Vitro* 14(4): 385; 1978. (no refs)

78-2749 **Comparing the Cytotoxic and Mutagenic Effect on Human Cells of Low Doses of Physical and Chemical Carcinogens (Meeting Abstract).** (Eng) Maher, V. M. (Michigan State Univ., East Lansing, MI, 48824); McCormick, J. J. *Mutat Res* 53(1): 120-121; 1978. (no refs)

78-2750 **Frequent Appearance of Radiation-induced Transformation in Junctional Areas of Colonies.** (Eng) Okada, Y. S. (Div. Physiology and Pathology, Natl. Inst. Radiological Sciences, Anagawa 4-9-1, Chiba 280, Japan); Watanabe, I. *Gann* 69(1): 91-93; 1978.

Transformation induction was studied in mouse embryo 10T-1/2 cells that had been x-irradiated with 600 R. Following irradiation, at least three different types of morphologically altered foci were noted: Type 1 consisted of tightly packed cells, but there was no other deviation from normal morphology; Type 2 appeared as a focus of cells piled into opaque multilayers; Type 3 consisted of densely piled, multilayered cells criss-crossed by arrays of densely stained cells. Since Types 2 and 3 have been shown to form malignant tumors in syngeneic mice, they were studied further. The morphology of these cells in the interior of a colony was almost uniform. Phenotypic changes were noted only when two or three colonies contacted each other. The phenotypic changes included the appearance of morphologically altered cells, the formation of giant cells, and the development of transformed cells. These altered cells were also characterized by a change in cytoplasmic affinity to May-Grunwald and Giemsa staining. The transformation frequency was $< 10^{-2}$; the frequency of induction of giant cells was apparently higher than that of transformed foci. The phenotypically changed cells appeared to be induced by overlapping of the cells of two developing colonies and by interaction between the cells. Contact between colonies is suggested to be an important factor in radiation-induced transformation. (6 refs)

78-2751 **Induction of Colonic Adenocarcinoma in the Rat by X-Irradiation.** (Eng) Denman, D. L. (Radiation Res. Lab., 14 Medical Labs., Univ. Iowa, Iowa City, IA, 52242); Kirchner, F. R.; Osborne, J. W. *Cancer Res* 38(7): 1899-1905; 1978.

A procedure for the in situ irradiation of the rat descending colon was developed to study how the level of x-ray exposure affects the incidence of colonic neoplasms. Only the descending colons of the rats were irradiated with a collimated x-ray beam. Ninety male Holtzman rats were divided into six exposure groups of 15 animals each. In the first group, the colon was exposed to 2,500 R: for each successive group, the exposure was increased by 1,000-R increments through 6,500 R. The sixth group was sham-irradiated. Animals in each of the irradiated groups developed mucoid adenocarcinomas, with the highest incidence (47%) being observed in the 4,500-R group. Histopathologically, the tumors were well-differentiated adenocarcinomas that closely resembled human colon adenocarcinomas. One of the animals with multiple primary tumors developed pulmonary metastases. During histological studies of the irradiated colons from animals not displaying grossly visible tumors, many areas of pathological bone formation were noted. These foci of heterotopic ossification were present in many of the irradiated colons regardless of whether cancer had developed. No reports of radiation-induced bone formation in the colon could be found in the literature. (16 refs)

78-2752 **Radiation Induced Ovarian Pathogenesis in the Progeny of X-irradiated Female Mice.** (Eng) Palayoor, T. (Embryology Div., Cancer Res. Inst., Bombay 400 012, India); Batra, V. K.; Batra, B. K. *Indian J Exp Biol* 15(3): 163-167; 1978.

Female C57BL ICRC mice were given a whole-body dose of 300 R x-rays and mated with unexposed litter mates. The descendants resulting from several generations of inbreeding had decreased gonadotropic activity. Offspring from the 16th generation onward were exposed at weaning to 400 R γ-rays and autopsied 2.5-3.5 mo later. Adenomatous growths observed in the ovaries of mice sacrificed 3.5 mo after irradiation were not considered tumors, but their resemblance in structure and possible histogenesis to neoplastic developments in irradiated mouse ovaries reported by others was obvious. No adenomatous growths were observed in the ovaries of control animals similarly irradiated, but initial stages of change were noted. The reduction of the latency period can be considered a significant alteration in the ovarian response of the treated progeny to ionizing radiation. This reduction may reflect the inherent qualities of ovarian cells to react to the hormonal stimulus available (in this case, decreased pituitary gonadotropic potency) more intensely than the cellular components of the control ovaries. (28 refs)

78-2753 **Transmission of Radiation-induced Elevations in Abnormally-Shaped Murine Sperm (Meeting Abstract).** (Eng) Hugenholtz, A. P. (Dept. Medical Biophysics, Univ. Toronto, Toronto, Ontario, Canada); Bruce, W. R. *Mutat Res* 53(1): 122-123; 1978. (1 ref)

78-2754 **Comparisons of X-ray and Ethylmethanesulfonate Mutagenesis of Three Human Lymphoblast Cell Cultures (Meeting Abstract).** (Eng) Slesinski, R. S. (Chemical Hygiene Fellowship, Carnegie-Mellon Univ., Pittsburgh, PA, 15213); Kotek, J. J. *Mutat Res* 53(1): 119-120; 1978. (no refs)

78-2755 **Myeloid Leukaemia in X-ray Irradiated CBA Mice.** (Eng) Major, I. R. (Radiobiology Unit, Medical Res. Council, Harwell, Didcot, Oxon, England); Mole, R. H. *Nature* 272(5652): 455-456; 1978.

The development of myeloid leukemia (ML) was studied in male CBA mice given a single, brief, whole-body x-ray exposure. The dose-response relationship was highly curvilinear, and the optimum dose for ML induction was in the region 150-300 rads. Larger doses resulted in a substantial and significant reduction in leukemia frequency. In a further study, ML frequency in mice exposed to 300 rads was markedly reduced (or postponed) by a further 600-rad dose given 1, 4, 16, or 32 wk later. The results support the hypothesis that the dose-dependent reduction of leukemia in this strain is the result of competition between two concomitant radiation effects: malignant transformation and cellular inactivation (loss of the ability to undergo sequential cell division). (10 refs)

78-2756 **Malignant Transformation in Cultured Hamster Embryo Cells Produced by X-Rays, 430-keV Monoenergetic Neutrons, and Heavy Ions.** (Eng) Borek, C. (Dept. Radiology, Coll. Physicians and Surgeons, Columbia Univ., New York, NY, 10032); Hall, E. J.; Rossi, H. H. *Cancer Res* 38(9): 2997-3005; 1978.

The efficiency of x-rays (1-600 rads), 430-kiloelectron volt (keV) monoenergetic neutrons (0.1-150 rads), and argon ions (429 million electron volts/atomic mass unit) in producing tumors in short-term cultures of golden hamster embryo cells was determined. The transformation frequency per surviving cell for neutrons reached a higher max value than that for x-rays. When the data were expressed as the number of transformations per initial cells at risk, the two dose-response curves rose to essentially the same peak value. Thus the max carcinogenic potential of the two types of radiation is the same. The relative biological effectiveness of neutrons relative to x-rays was not greatly different whether killing or transformation was the end point scored. Studies of the biological properties of the malignant transformed cells indicated no qualitative difference in those transformed by neutrons compared with those transformed by x-rays. Argon ions were much more effective in producing transformations than x-rays, and they were similar in effectiveness to 430-keV neutrons. (36 refs.)

78-2757 **The Effect of Different Numbers of Untransformed Cells on Focus Formation of C3H 10T1/2 Cells Transformed by Alpha Particles (Meeting Abstract).** (Eng) Lloyd, E. L. (Argonne Natl. Lab., Argonne, IL, 60439); Gemmell, A. *In Vitro* 14(4): 383; 1978. (no refs)

78-2758 **Diagnostic Radiation - A Possible Factor in Malignancy in Patients with Chronic Inflammatory Bowel Disease (Meeting Abstract).** (Eng) Faintuch, J. (Dept. Medicine, Misericordia Hosp., Winnipeg, Canada); Rogers, A. G.; Sourkes, A. M.; Cormack, D. V. *Gastroenterology* 74(5, part 2): 1033; 1978. (no refs)

78-2759 **Gastric Stump Cancer: Statistical Evaluation.** (Eng) Cheli, R. (Divisione de Gastroenterologia, Ospedali Civili di Genova, Ospedale Generale Regionale, Viale Benedetto XV, 16132 Genoa, Italy); Molinari, F.; Santi, L.; Ciancamerla, G.; Puntoni, R. *Rend Gastroenterol* 9(3): 169-172; 1978.

The incidence of gastric stump cancer was determined in 180 patients who underwent resection of a gastric ulcer and 337 patients who underwent resection of a duodenal ulcer. Seven of the former and nine of the latter group died of stump cancer; the interval between surgery and death was 13-16 yr. Comparison with a control group indicated that the risk of gastric cancer was significantly higher in the ulcer patients. The reason for this higher risk is not known. (23 refs)

78-2760 **Malignant Astrocytoma Following Radiotherapy of a Craniopharyngioma. Case Report.** (Eng) Sogg, R. L. (Div. Ophthalmology, Stanford Univ. Medical Center, Stanford, CA, 94305); Donaldson, S. S.; Yorke, C. H. *J Neurosurg* 48(4): 622-627; 1978.

A malignant astrocytoma developed in the right temporal lobe of a 14-yr-old girl 5 yr after she had received 6,007 rads to the suprasellar region for a craniopharyngioma. Superimposition of isodose curves from the original irradiation on the site of the glioma showed that it had received 80%-95% of the original 6,007 rads. There was no microscopic connection between the craniopharyngioma and the glioma. (16 refs)

78-2761 **Development of Highly Differentiated Thyroid Carcinomas.** (Rus) Valdina, E. A. (Dept. Pulmonary and Mediastinal Tumors, N. N. Petrov Scientific Res. Inst. Oncology, Leningrad, USSR); Puchkov, Yu. G. *Vopr Onkol* 23(11): 23-29; 1977.

To verify the hypothesis that highly differentiated carcinomas can undergo anaplastic transformation (especially after irradiation), the case histories of 83 patients subjected to repeated surgery for highly differentiated carcinoma of the thyroid are reviewed. The interval between the first and second surgery ranged from 2 to 15 yr, and 30 patients had received radiotherapy before or after the first surgery. Although the morphological diagnosis of the tumor was reconsidered in 51 patients, none of them showed signs of anaplasia. In a group of 11 patients in whom the interval between surgeries ranged from 8 to 15 yr, both the recurrent tumor and the regional and hematogenic metastases retained the features of a highly differentiated carcinoma. (15 refs)

78-2762 **Radiation Associated Anaplastic Carcinoma of the Thyroid (Meeting Abstract).** (Eng) Shimaoka, K. (Roswell Park Memorial Inst., Buffalo, NY, 14263); Getaz, E. P.; Rao, U. *Proc Am Assoc Cancer Res* 19: 219; 1978. (no refs)

78-2763 **The Effect of Different Mineral Dusts on the Mechanism of Phagocytosis: A Scanning Electron Microscope Study.** (Eng) Miller, K. (Dept. Immunology, Natl. Res. Inst. Occupational Diseases, South African Medical Res. Council, Johannesburg 2000, S. Africa); Handfield, R. I.; Kagan, E. *Environ Res* 15(1): 139-154; 1978.

A scanning electron microscopic study was made of the ability of male Wistar rat alveolar macrophages to phagocytize crocidolite asbestos fibers (≥ 20 μm or ≤ 5 μm in length) and quartz particles (1-3 μm in diameter). Both short and long asbestos fibers were firmly attached to the macrophages after 4 min. Attachment of the short fibers occurred without any extensive spreading of pseudopodia or development of filopodia. After 20 min, phagocytosis of all the fibers was complete. With long fibers, many macrophages developed large flattened pseudopodia, and the fibers were attached, along their long axis, to the pseudopodia by small cytoplasmic tendrils. Phagocytosis of one or more fibers did not prevent the cell from further active phagocytosis. After 30 min of incubation, long fibers were still present in the culture, and macrophages were still attempting to phagocytose them. Quartz particles, however, were phagocytosed within 2-4 min. The macrophages developed long tenuous filopodia to which the quartz particles were attached. These differences in phagocytosis could be the result of the different physicochemical properties of the fibers. The recognition and attachment of the fibers could have some bearing on the role of the macrophage in the pathogenesis of dust disease. (11 refs)

78-2764 **Ferruginous Bodies in Sputa of Former Asbestos Workers.** (Eng) Farley, M. L. (Tyler Asbestos Workers Program, Texas Chest Foundation, East Texas Chest Hosp., Tyler, TX); Greenberg, S. D.; Shuford, E. H.; Hurst, G. A.; Spivey, C. G.; Christianson, C. S. *Acta Cytol (Baltimore)* 21(5): 693-700; 1977.

The occurrence of ferruginous bodies in the sputa of 628 former asbestos workers and 138 controls was determined, and the findings were related to age, cigarette smoking history, cellular atypia, time since last occupational exposure, and length of occupational exposure. No ferruginous bodies were detected in sputum specimens from controls, but they were detected in 224 former workers. One worker with only 1 day of exposure 10 yr previously had one body in 1/4 sputum specimens. Generally, the number of bodies was related to the age of the worker and length of exposure. However, study of the significance of each of these factors indicated that only length of exposure and number of ferruginous bodies were related by an increasing logarithmic function. No significant association was found between these bodies and age, smoking history, degree of cellular epithelial atypia, or time since last exposure. It is concluded that ferruginous bodies in the sputa are evidence of probable significant occupational exposure to asbestos dust and that routine cytopathology procedures are sufficient for their detection. (30 refs)

78-2765 **Morphological Changes in Sc Connective Tissue of Rats after Implantation of Polymers Used in Stomatology.** (Rus) Zabezhinsky, M. A. (Lab. Chemical Carcinogens, N. N. Petrov Scientific Res. Inst. Oncology, Leningrad, USSR); Pliss, G. B.; Eliseev, V. V.; Bobin, E. Yu.; Shteingart, M. Z. *Vopr Onkol* 24(1): 94-95; 1978.

To evaluate the possible carcinogenic effect of the polymers used in stomatology, the polymer AKR-15 (Ethakril) or its analog were implanted sc in random-bred albino rats. The polymers were in the form of a plate or a powder (500 mg). Implantation of plastic plates resulted in the development of tumoral encapsulated nodes in 10/26 rats implanted with AKR-15 and 7/14 rats implanted with its analog (av latent periods were 16.5 and 15 mo, respectively). Rats implanted with powder were tumor-free. (2 refs)

See also:
*(Rev.): 78-2407, 78-2413, 78-2414, 78-2438, 78-2439, 78-2440, 78-2441, 78-2458.
*(Chem.): 78-2504, 78-2563, 78-2624, 78-2659, 78-2663, 78-2686.
*(Viral): 78-2788.
*(Path.): 78-2948.
*(Epid.-Biom.): 78-2965.

78-2766 **Reverse Transcriptase of RNA Tumor Viruses. V. In Vitro Proteolysis of Reverse Transcriptase from Avian Myeloblastosis Virus and Isolation of a Polypeptide Manifesting Only RNase H Activity.** (Eng) Lai, M. T. (Tumor Virology Lab., Salk Inst., San Diego, CA, 92112); Verma, I. M. *J Virol* 25(2): 652-663; 1978.

Purified avian myeloblastosis virus reverse transcriptase contains two structurally related subunits. The larger subunit, β (mol wt, 95,000), was converted in vitro by chymotrypsin into a polypeptide with a mol wt of 63,000. This polypeptide was indistinguishable from the smaller subunit, α (mol wt 65,000), in its chromatographic behavior on a phosphocellulose column and in its tryptic peptide composition. A 32,000-mol-wt polypeptide (fragment B) generated during proteolysis was composed of tryptic peptides unique to β, and it appeared to be derived from the portion of the subunit that was cleaved during the conversion of the latter into α. Upon continued proteolysis, a 24,000-mol-wt peptide (fragment A) was generated. This polypeptide manifested only RNase H activity and shared common amino acid sequences with the β and α subunits. Fragments A and B did not share any amino acid sequence homology. (45 refs)

78-2767 **Anti-tumor Virus Activity of Copper-binding Drugs.** (Eng) Levinson, W. (Dept. Microbiology, Univ. California at San Francisco, San Francisco, CA 94143); Mikelens, P.; Jackson, J.; Kaska, W. *Adv Exp Med Biol* 91: 161-178; 1977.

Several Cu-binding ligands can inhibit the RNA-dependent DNA polymerase activity of Rous sarcoma virus (RSV) and inactivate the ability of this virus to transform chicken embryo cells. Compounds effective as free ligands and as Cu complexes in inhibiting both RNA-dependent DNA polymerase activity and transforming ability include N-methylisatin β-thiosemicarbazone, 2-pyridine thiosemicarbazone, and 8-hydroquinoline and its 5-chloro and 7-iodo derivatives. Kethoxal bis(thiosemicarbazone), thiosemicarbazide, and dithizone were less active as free ligands than as preformed complexes and isoquinoline 1-formyl thiosemicarbazone was active as a free ligand. Isonicotinic acid hydrazide was active only in complex form. O-Phenanthroline and aurintricarboxylic acid inhibited the polymerase but failed to inactivate transforming ability except at high concentrations. Some Cu-binding ligands had no effect on either parameter. Thus, there is no simple relationship between the antiviral activity of Cu-binding ligands and their nucleic acid-binding activity. However, the biological importance of thiosemicarbazone-Cu complex binding to nucleic acids is supported by the observation that treatment of intact RSV virions with the complex causes 70S RNA to sediment abnormally in centrifugation analyses. (46 refs.)

78-2768 **Polyadenylated RNA Molecules and Polyribosomes in Tumours of Chemical and Viral Origin.** (Eng) Shaposhnikov, J. D. (Lab. Biochemistry, Prof. N. N. Petrov Res. Inst. Oncology, 188646 Leningrad, USSR); Ratovitski, E. A. *Int J Cancer* 21(3): 395-399; 1978.

The distribution of polyadenosine [poly(A)]-enriched messenger RNA in the nuclei, mitochondria, and free and membrane-bound polyribosomes from normal C3HA mouse and Syrian hamster livers and normal chicken fibroblasts was compared with that in the corresponding subcellular fractions in the transplantable, chemically induced MD hepatoma and in non-virus-producer hamster and virus-producer avian (chicken) Rous sarcomas. The amount of poly(A)-RNA was higher in the nuclei, mitochondria, and membrane-bound polysomes of the tumor cells than in normal cells. In the free polysomes of the hepatoma and the hamster Rous sarcoma, the poly(A)-RNA content was not changed significantly, but the free polysomes of avian Rous sarcoma contained less poly(A)-RNA than normal chicken fibroblasts. Differences between free and membrane-bound polysomes can be related to changes in the amount of ribosomal RNA. A study of the comparative distribution of free, membrane-bound, and mitochondria-associated polysomes in normal and transformed cells indicated that the relative content of free polysomes was increased in transformed cells. Therefore, the amount of polysome associated with various membranous structures was decreased. The amount of mitochondria-associated polysomes per milligram of mitochondrial protein was reduced in the hepatoma, almost the same in the hamster sarcoma, and significantly increased in the chicken sarcoma compared to their normal cell counterparts. It is concluded that in carcinogen-induced, virus-producing, and non-virus-producing tumors, similar changes occur in the subcellular distribution of polyribosomes and poly(A)-containing RNA. (32 refs)

78-2769 **The Size and Genetic Composition of Virus-specific RNAs in the Cytoplasm of Cells Producing Avian Sarcoma-Leukosis Viruses.** (Eng) Weiss, S. R. (Dept. Microbiology, Univ. California, San Francisco, CA 94143); Varmus, H. E.; Bishop, J. M. *Cell* 12(4): 983-992; 1977.

The major species of stable virus-specific RNA's were identified and characterized in chicken embryo fibroblasts productively infected with both nondefective and defective strains of avian sarcoma virus (ASV). Radioactive DNA's (cDNA) complementary to defined portions of the ASV genome were used to characterize the virus-specific RNA molecules. The cytoplasm of the cells producing nondefective ASV contained three species of polyadenylated virus-specific RNA. These species may serve as messengers for the synthesis of viral proteins. The largest species (3.3 x 10^6 daltons) encodes all four viral genes and is probably identical with the viral genome. A second species (1.8 x 10^6 daltons) encodes the $3'$ half of the viral genome, including *env*, *src*, and a genetically silent region known as "c". The three species of virus-specific RNA contain nucleotide sequences at least partially homologous to a sequence of 101 nucleotides found at the extreme $5'$ end of the ASV genome. The size and genetic composition of virus-specific RNA's in cells producing defective deletion mutants reflected the nature of the deletion. It is hypothesized that 3/4 viral genes (*gag*, *env*, and *src*) are translated from distinct messenger RNA's that contain the expressed gene at the $5'$ end. The remaining gene, *pol*, may be expressed by the synthesis and subsequent processing of a polyprotein that contains the products of both *gag* and *pol*. (49 refs.)

78-2770 **Uninfected Avian Cells Contain RNA Related to the Transforming Gene of Avian Sarcoma Viruses.** (Eng) Spector, D. H. (Dept. Microbiology, Univ. California, San Francisco, CA, 94143); Smith, K.; Padgett, T.; McCombe, P.; Roulland-Dussoix, D.; Moscovici, C.; Varmus, H. E.; Bishop, J. M. *Cell* 13(2): 371-379; 1978.

The DNA of uninfected avian cells contains nucleotide sequences (*sarc*) related to the transforming gene (*src*) of avian sarcoma virus. In the present study, *sarc* was transcribed into RNA in a variety of normal and neoplastic avian cells and tissues, including cultured embryonic fibroblasts, normal embryos, normal adult tissues, methylcholanthrene-induced fibrosarcomas, and cells explanted from these tumors into continuous culture. Since RNA was not transcribed from the genes for globin and ovalbumin in embryonic avian fibroblasts, the presence of *sarc* RNA in normal cells cannot be attributed to a low constitutive level of transcription from all cellular genes. *Sarc* RNA levels were similar in normal and neoplastic cells, in embryos at all stages of development, and in both logarithmically growing and fully quiescent fibroblasts. Synthesis of *sarc* RNA in chicken cells was coordinated with the expression of endogenous retrovirus genes. This confirms other evidence that *sarc* is not linked to an endogenous viral genome. Although *sarc* is part of messenger RNA in normal and neoplastic avian cells, it is not implicated in normal cellular growth, embryogenesis, or chemical carcinogenesis. (28 refs)

78-2771 **Intracellular Precursors to the Major Glycoprotein of Avian Oncoviruses in Chicken Embryo Fibroblasts.** (Eng) Buchhagen, D. L. (Sidney Farber Cancer Inst., Boston, MA, 02115); Hanafusa, H. *J Virol* 25(3): 845-851; 1978.

The identification of a 96,000-dalton glycoprotein, p(96), is reported. It was present in cell extracts from gs-chf-chicken embryo fibroblasts infected with Rous-associated virus-2 subgroup B (RAV-2) and the Schmidt-Ruppin strain of Rous sarcoma virus subgroup A (SR-RSV-A), as well as from uninfected gsLchf+ (HE) cell extracts. It was not found in cell extracts from uninfected gs-chf- or gs+chf+ (HH) cells or from gs-chf-cells infected with envelope-deficient Bryan high-titer Rous sarcoma virus. Immunoprecipitation, kinetic, and biochemical data indicated that this polyprotein was a precursor to gp85. A second polyprotein of 80,000 daltons, p(80), was also present in the RAV-2- and SR-RSV-A-infected gs-chf- cells. This second polyprotein contained less carbohydrate than p(96), and kinetic and biochemical data indicated that p(80) may be an immature form of p(96). (22 refs)

78-2772 **Glucose-Specific Cytochalasin B Binding Is Increased in Chick Embryo Fibroblasts Transformed by Rous Sarcoma Virus (Meeting Abstract).** (Eng) Salter, D. W. (Dept. Microbiology, Univ. Illinois, Urbana, IL, 61801); Weber, M. J. *Fed Proc* 37(6): 1597; 1978. (no refs)

78-2773 **Cytoplasmic Vacuoles of Rous Virus Transformed Cells Are Organelles Involved in Cation Uptake.** (Eng) Bader, J. P. (NCI, NIH, Bethesda, MD, 20014); Brown, N. R. *J Gen Virol* 38(3): 471-481; 1978.

Cytoplasmic vacuoles induced during the transformation of chick embryo cells by Bryan strain of Rous sarcoma virus (RSV-BH) were studied using neutral red (NR), a cationic dye. Both the rate of uptake and the accumulation of NR were greater in RSV-BH-transformed cells than in nontransformed cells. However, uptake was greater in vacuolated than in nonvacuolated cells, whether or not they were transformed. The NR was incorporated into preexisting vacuoles in the absence of cytoplasmic staining, suggesting the existence of direct channels from the cell surface to the vacuoles. Other low-mol-wt cationic dyes could also be incorporated into vacuoles, but those with branched structures or cationic wts > 330 were excluded; no anionic dyes were incorporated. In experiments with the mutant RSV-BH-Ta, which induces temperature-dependent vacuolization, vacuoles developed at different rates and at various morphologies with different cations, following a shift to the vacuole-permissive temperature. Vacuoles that had formed in the presence of K^+, Rb^+, tris+, and choline+ failed to disappear when cells were incubated at a temperature sufficient to revert vacuoles formed in Na+-containing medium. No short-term effects of Cl^- replacements (Br^-, I^-, or SO_4^{2-}) on vacuolization or reversal were observed. These findings suggest that the vacuoles are organelles involved in cation uptake. In transformed cells, the increased Na+ content (most common cation) in the vacuoles could affect the extent or specificity of transcription and be responsible for the failure of these cells to respond to regulatory mechanisms. (11 refs)

78-2774 **Transformation of Chick Embryo Neuroretina by Rous Sarcoma Virus (Meeting Abstract).** (Eng) Munson, R. (Washington Univ. Medical Sch., St. Louis, MO, 63100); Glaser, L. *Fed Proc* 37(6): 1698; 1978. (no refs)

78-2775 **Autoradiographic Studies of Proviral DNA Synthesis in Enucleated Chick Embryo Fibroblasts Infected with Avian Sarcoma Virus.** (Jpn) Tanaka, T. (Dept. Biochemistry, Cancer Inst. Okayama Univ., Shikadacho, Okayama 700, Japan). *Okayama Igaku Zasshi* 89(9/10): 1177-1186; 1977.

After infection of susceptible cells with RNA tumor viruses, viral RNA is reversibly transcribed into proviral DNA. To determine the site of proviral DNA synthesis, chick embryo fibroblasts (CEF) were enucleated by centrifugation in the presence of cytochalasin B, and the enucleated CEF (cytoplasts) were infected with the B77 strain of avian sarcoma virus (B77-ASV). ^{3}H-thymidine incorporation into cytoplast DNA was investigated by autoradiography. Photopositive grains were observed in cytoplasts infected with B77-ASV, but not in mock-infected cytoplasts. The photopositive grains in the B77-ASV-infected cytoplasts disappeared almost completely after DNase I treatment. N-Demethylrifampicin, a specific inhibitor of reverse transcriptase, inhibited the appearance of photopositive grains. When ultrathin sections of the infected cytoplasts were studied by electron microscope autoradiography, the photopositive grains appeared in the cytoplasm without relation to mitochondria. These results indicate that proviral DNA synthesis is initiated in the cytoplasm of B77-ASV-infected CEF without the direct participation of the nucleus. (24 refs)

78-2776 **Identification of a Polypeptide Encoded by the Avian Sarcoma Virus *src* Gene.** (Eng) Purchio, A. F. (Dept. Microbiology and Immunology, Univ. California Sch. Medicine, Los Angeles, CA, 90024); Erikson, E.; Brugge, J. S.; Erikson, R. L. *Proc Natl Acad Sci USA* 75(3): 1567-1571; 1978.

The use of two procedures to detect a polypeptide encoded by the avian sarcoma virus (ASV) *src* gene is reported. First, antiserum from rabbits bearing ASV-induced fibrosarcomas was used to immunoprecipitate a transformation-specific antigen from ASV-transformed chick embryo fibroblasts. This antigen had an apparent mol wt of 60,000 daltons. Second, the 3′ one-third of the ASV genome, selected by oligo(deoxythymidine)cellulose chromatography and sucrose gradient sedimentation, was translated in a messenger RNA-dependent reticulocyte cell-free lysate. This RNA species programmed the synthesis of a polypeptide that comigrated with the transformation-specific antigen of mol wt 60,000 daltons immunoprecipitated from transformed cells. The methionine-containing tryptic peptides from the 60,000-dalton polypeptides obtained by both methods were identical upon two-dimensional fractionation. (32 refs)

78-2777 **Reversion to the Normal Phenotype by Protein Synthesis Inhibitors Acting on Rat Kidney Cells Transformed by a Temperature-Sensitive Rous Sarcoma Virus Mutant (Meeting Abstract).** (Eng) Carroll, R. (Univ. California at San Diego, La Jolla, CA, 92093); Ash, J. F.; Singer, S. J.; Vogt, P. K. *Fed Proc* 37(6): 1433; 1978. (1 ref)

78-2778 **Characteristics of Cellular RNA Related to the Transforming Gene of Avian Sarcoma Viruses.** (Eng) Spector, D. H. (Dept. Microbiology, Univ. California, San Francisco, CA, 94143); Baker, B.; Varmus, H. E.; Bishop, M. J. *Cell* 13(2): 381-386; 1978.

Nucleotide sequences (*sarc*) related to the transforming gene of avian sarcoma viruses are present in the DNA and transcribed into RNA in uninfected avian cells. Cytoplasmic RNA containing the *sarc* sequences has a sedimentation coefficient of 30S, is linked to polyadenylic acid, and is present in polyribosomes in a form that can be released by EDTA treatment. Thus, the *sarc* sequences (complexity = 5 x 10^5 daltons) are part of a larger transcriptional unit (approx 1.8 x 10^6 daltons), and the *sarc* RNA appears to serve an as yet unknown messenger function. In addition, the *sarc* sequences in chicken cells are not linked to RNA transcribed from the provirus of the endogenous virus RAV-O, since RAV-O cytoplasmic RNA had sedimentation coefficients of 32S and 21S. Comparison of *sarc*-containing RNA in quail embryo fibroblasts and in quail cells derived from a methylcholanthrene-induced fibrosarcoma revealed no differences in the size, polyadenylation, or concentration of the RNA in nuclei, cytoplasm, and polyribosomes. Therefore, phenotypic differences between these cells are unlikely to be a consequence of altered expression of the *sarc* sequences. (17 refs)

78-2779 **Purification and Characterization of Particles Containing RNA-Dependent DNA Polymerase, in the Allantoic Fluid of Uninfected Leukosis Virus-Free Chicken Eggs.** (Eng) Bauer, G. (Max-Planck-Institut fur Biochemie, 8033 Martensried, 6300 Giessen, W. Germany); Friis, R. R.; Jilek, G.; Hofschneider, P. H. *Biochim Biophys Acta* 518(1): 125-137; 1978.

Particles containing particle-associated RNA-dependent DNA polymerase were isolated from the allantoic fluid of embryonated chicken eggs and purified and examined. The particles were obtained from commercially available leukosis virus-free eggs. They did not have the protein composition or sedimentation constant of known avian RNA tumor viruses and they did not exhibit helper activity, interfering properties, or infectivity. Endogenous DNA synthesis was not detected in the particles. The particle-bound enzyme accepted homopolymeric and heteropolymeric RNA as templates and it could synthesize DNA complementary to natural heteropolymeric RNA. These findings indicate that the RNA-dependent DNA polymerase test for leukosis virus can no longer be taken as specific unless the enzyme tested is purified and characterized. (23 refs)

78-2780 **Glial-Mesenchymal Tropism of In Vivo Avian Sarcoma Virus Neuro-oncogenesis in Rats.** (Eng) Copeland, D. D. (Dept. Pathology, Duke Univ. Medical Center, Durham, NC, 27710); Bigner, D. D. *Acta Neuropathol (Berl)* 41(1): 23-25; 1978.

To determine whether avian sarcoma virus (ASV) inoculation during neurogenesis would induce neuronal tumors, the fetuses of 22 pregnant F-344 rats received 5 or 2.5 x 10^5 focus-forming units of the Bratislava-77 strain of ASV between days 17 and 20 of gestation. Inoculations were made into the right cerebral hemisphere of the fetus. Seventeen females delivered 112 live pups, but only 61 could be recovered for autopsy because of hemorrhagic-cyst disease. There were a total of 183 tumors, and most of these were poorly differentiated gliomas (58) or polymorphic astrocytomas (56). No tumor composed of neurons or neuronal precursors was identified. Thus, glial-mesenchymal tropism of in vivo ASV neurooncogenesis is independent of the stage of neurocytodifferentiation at inoculation. (20 refs)

78-2781 **Cell Surface Changes Occurring as Part of the Process of Transformation (Meeting Abstract).** (Eng) Hawkes, S. (Univ. California, Berkeley, CA, 94720); Parry, G. *Fed Proc* 37(6): 1562; 1978. (no refs)

78-2782 **In Vitro Transformation with Avian Myelocytomatosis Virus Strain CMII: Characterization of the Virus and Its Target Cells.** (Eng) Graf, T. (Biologisch-Medizinische Abteilung, Max-Planck-Institut fur Virusforschung, D-7400 Tubingen, W. Germany); Royer-Pokora, B.; Meyer-Glauner, W.; Claviez, M.; Gotz, E.; Beug, H. *Virology* 83(1): 96-109; 1977.

The ability of avian myelocytomatosis virus strain CMII to transform cells from normal SPAFAS chickens was determined. When cells from the bone marrow, yolk sac, and spleen of these chickens were infected with liver extracts and plasma from CMII-infected chickens, transformation foci were noted in 4-6 days. CMII-transformed bone marrow cells could be propagated for up to 15 passages. No differences in the properties of transformed hematopoietic cells from the various organs were noted. The cells closely resembled hematopoietic cells transformed by strain MC29 myelocytomatosis virus, but they differed from avian myeloblastosis virus-transformed cells. The cells were phagocytic, became adherent under certain culture conditions, and required colony-stimulating factor for colony formation in semisolid medium; these properties are characteristic of cells of the granulocyte/macrophage line of differentiation. In contrast to avian erythroblastosis virus, CMII effectively transformed macrophage cultures, suggesting that the target cell belongs to the corresponding differentiation lineage. However, cell-separation experiments showed that the target cells were not identical to normal granulocyte/macrophage colony-forming cells. CMII also induced a morphological transformation of chicken embryo fibroblasts. CMII was found to consist of a mixture of a transforming component and an associated nontransforming virus of subgroup B or D. The transforming component is defective for replication and could be complemented by subgroup B, C, or D helper viruses. (25 refs)

78-2783 **Sites of Integration of Infectious DNA of Avian Reticuloendotheliosis Viruses in Different Avian Cellular DNAs.** (Eng) Battula, N. (NCI, Bethesda, MD, 20014); Temin, H. M. *Cell* 13(2): 387-398; 1978.

The integration pattern of the infectious DNA of spleen necrosis virus (SNV) and chick syncytial virus (CSV), two avian reticuloendotheliosis viruses whose DNA is not inactivated by digestion with the restriction endonuclease Eco RI, was determined. High-mol-wt DNA from infected chicken, turkey, and pheasant cells was digested with Eco RI, electrophoresed through agarose gels, and assayed for infectivity. The integration of SNV and CSV infectious DNA was similar in the three species of avian cells infected at high or low multiplicities of infection. There were multiple sites of integration in acutely infected cells with concomitant cell death. There was a single site of integration in chronically infected cells with no cell death. Compared with chronically infected cells, acutely infected cells contained more integrated infectious viral DNA molecules per cell. These results are consistent with hypotheses that cell death in the acute phase of infection is a result of the integration of infectious viral DNA at multiple sites and that cells survive only when infectious viral DNA is integrated exclusively at a single site. (32 refs)

78-2784 **Effect of Sensitised Leucocytes on Plaque Formation by Marek's Disease Virus.** (Eng) Ross, L. J. (Houghton Poultry Res. Station, Houghton, Huntingdon, Cambs., PE17 2DA, England). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 179-182; 1978.

Evidence was obtained for the presence in White Leghorn chickens vaccinated against Marek's disease virus (MDV) of circulating T lymphocytes sensitized to virus-specific antigens. The sensitized lymphocytes (SL) inhibited the formation of plaques by syngeneic lymphoid and kidney cells previously infected with MDV. The plaque inhibition occurred as a result of direct interaction between effector cells and cell-surface virus-specific antigens on the target cells, since the SL were removed by absorption to infected but not uninfected monolayers. The capacity of the SL to inhibit plaques was reduced significantly by anti-T serum but not by anti-B or anti-chick immunoglobulin sera. Absolute compatibility between effector and tar was not essential for plaque inhibition, as evidenced by the ability of chick SL to inhibit the formation of infective centers by MDV-infected duck embryo fibroblasts. (7 refs)

78-2785 **Murine and Human Melanomas Containing a High Molecular Weight RNA Associated with an RNA-instructed DNA Polymerase.** (Eng) Hehlmann, R. (Medizinische Poliklinik, Univ. Munich, Pettenkoferstrasse 8a, 8 Munich 2, W. Germany); Balda, B. R.; Spiegelman, S. *Int J Dermatol* 17(2): 114-122; 1978.

The presence of a 70S RNA associated with a reverse transcriptase was detected in murine, hamster, and human melanomas. B16 melanoma isolated from C57BL mice was found to contain 70S RNA-associated reverse transcriptase that back-hybridized to complementary DNA. Mouse melanoma DNA was then hybridized to human and hamster melanoma viral-enriched cytoplasmic RNA. Up to 5.7% of the mouse melanoma DNA hybridized to human melanoma RNA and >7% hybridized to hamster melanoma RNA. These results suggest the presence of melanoma-specific RNA sequences in human and hamster melanomas. The simultaneous detection technique was then applied to 14 human malignant melanomas. Thirteen tumors showed evidence of particles containing a reverse transcriptase and a high-mol-wt RNA. Two specimens from normal skin gave negative responses, but skin near a melanoma gave a positive response. Density determination of the high-mol-wt RNA and reverse transcriptase in human melanoma particles indicated that most of the particles banded at 1.16 g/cm^3, characteristic of RNA tumor viruses. Back-hybridization experiments indicated that labeled tumor DNA combined with the melanoma RNA. Thus, particles possessing characteristics of RNA tumor viruses are found in mouse melanoma B16 and in human melanomas. (28 refs)

78-2786 **Analysis of a 5′ Leader Sequence on Murine Leukemia Virus 21S RNA: Heteroduplex Mapping with Long Reverse Transcriptase Products. Heteroduplex Mapping with Long Reverse Transcriptase Products.** (Eng) Rothenberg, E. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Donoghue, D. J.; Baltimore, D. *Cell* 13(3): 435-451; 1978.

The 5′ terminal sequences of the glycoprotein messenger RNA (mRNA) of murine leukemia virus were investigated to see if the mRNA contained viral sequences from both the 5′ and 3′ ends of the genome. A 5′ probe was made by purifying a discrete 50-nucleotide-long reverse transcript attached to its transfer RNA primer. This probe hybridized to RNA of the size of glycoprotein mRNA--21S, polyadenosine-containing RNA; this indicated that the mRNA could have a 5′ leader sequence. The 5′-specific sequences were studied by electron microscopic examination of hybrids between 21S RNA and the two longest discrete complementary DNA species synthesized in the endogenous reverse transcriptase reaction. One 8.8-kilobase-long species was only made in the absence of actinomycin D; however, it did not contain any self-complementary sequences and it therefore appeared to be a complete transcript of the viral genome. An 8.2-kilobase species was synthesized regardless of the presence of actinomycin D; it terminates 500-600 nucleotides internal to the 5′ end of the template RNA. The structures observed in heteroduplexes of 21S RNA and these DNA's indicated the presence of a leader sequence approx 500 nucleotides long at the 5′ end of the 21S RNA. Sequences comprising this leader segment mapped at the 5′ end of the genome RNA; the remainder of the 21S RNA consisted of sequences from the 3′ portion of the genome. Analysis of heteroduplexes with 8.2-kilobase DNA suggested that actinomycin D could block the reverse transcription of most of the sequence in the genome RNA that appeared as a leader in the 21S RNA. (40 refs)

78-2787 **T1 Oligonucleotide Maps of N-, B-, and B — > NB-tropic Murine Leukemia Viruses Derived from BALB/c.** (Eng) Faller, D. V. (Dept. Biology, Center Cancer Res., Massachusetts Inst. Technology, Cambridge, MA 02139); Hopkins, N. *J Virol* 26(1): 143-152; 1978.

The large RNase T1-resistant oligonucleotides of N-, B-, and NB-tropic murine leukemia viruses derived from BALB/c mice were mapped relative to the 3′ end of the viral RNA. The maps indicated that (1) the large T1 oligonucleotides shared by the N-, B-, and NB-tropic viruses probably occupy the same relative positions on their genomes, (2) the 14 T1 oligonucleotides that differ between the N- and B-tropic viruses are derived from regions scattered along the genomes, and (3) an oligonucleotide that is present in five NB-tropic viruses but not in their B-tropic virus progenitors lies toward the 5′ end of the NB-tropic virus oligonucleotide map. Thus, the determinants of N-tropism reside in the 5′ end of the oligonucleotide map of the N-tropic virus isolate. Conversion from N- to B-tropism might require an alteration in the viral genome at a position corresponding to this portion of the map. (28 refs)

78-2788 **Production and Analysis of a Viral Complex Derived from a Radiation-induced Leukemia Virus of the C57BL Mouse.** (Fre) Mamoun, R. (Unite de Recherches de Radiobiologie Experimentale et de Cancerologie, INSERM Unite 117, 229 cours de l'Argonne, 33076 Bordeaux, France); Guillemain, B.; Astier, T.; Portail, J. P.; Legrand, E.; Duplan, J. F. *Int J Cancer* 22(1): 98-105; 1978.

A radiation leukemia virus (RadLV-Rs) isolated from a C57BL mouse with radiation-induced leukemia was shown to induce lymphoreticular cell neoplasms of the spleen and lymph nodes rather than thymomas. Several cell lines established in culture from the spleen and lymph node tumors were originally composed of round virus-producing cells, but their ability to produce virus disappeared progressively upon serial passage. Simultaneously, the cells grew as a monolayer of polygonal epitheliallike cells. In several clones derived from one of the tumor cell lines, the presence of round cells correlated positively with viral release. Besides these morphological differences, the karyotype of the virus-producing clones was strongly modified. Cells of clone 13-3C (producing a virus called 13C) contained 43 chromosomes, a large number of which were metacentric. In contrast, clone 13-2D, which had the same origin as 13-3C but did not produce virus, had 70 acrocentric chromosomes. Virus 13C had the same pathogenic effects as the original RaLV-Rs; on the basis of its biochemical and morphological properties, it was classified as a C-type retrovirus, and it also induced Gross-type cell-surface antigens. Unlike most murine leukemia viruses (MuLV), this virus did not show any helper activity for the murine sarcoma virus (MSV) integrated in S+L-mouse cells. However, syncytia formation was readily observed after cocultivation of 13-3C and XC cells. The 13C virus was shown to be a complex composed of an ecotropic MuLV that preferentially infects B-type mouse cells (MuLV-B) and a xenotropic virus (MuLV-X). The MuLV-B had no helper activity for integrated MSV and did not induce XC syncytia formation. The MuLV-X was a potent helper for MSV in S+L-mink cells, but it did not induce XC syncytia formation. (37 refs)

78-2789 **Loss of Immunosuppressive Potential in a Highly Leukemogenic Clone of Murine RNA Tumor Virus, 334C (Meeting Abstract).** (Eng) Robinson, M. K. (Roswell Park Memorial Inst., Buffalo, NY, 14263); Evans, M. J.; Manly, K. F. *Fed Proc* 37(6): 1570; 1978. (no refs)

78-2790 **Relationships of gp70 of MuLV Envelopes to gp70 Components of Mouse Lymphocyte Plasma Membranes.** (Eng) Tung, J. S. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); O'Donnell, P. V.; Fleissner, E.; Boyse, E. A. *J Exp Med* 147(4): 1280-1284; 1978.

To investigate the relationship of viral gp70's to plasma-membrane gp70's, peptide maps of tryptic digests of gp70 bands obtained by sodium dodecyl sulfate-polyacrylamide gel electrophoresis of immunoprecipitated cell lysates were compared with maps of plasma-membrane gp70's from cultured cells infected by ecotropic or xenotropic murine leukemia viruses. Maps of gp70's from cells infected with ecotropic and xenotropic viruses were distinguishable from one another, and in general they resembled the gp70 maps prepared directly from ecotropic and xenotropic virions, respectively. Maps of gp70's immunoprecipitated from thymocytes of five mouse strains and from two A-strain T-cell leukemias also showed two distinguishable and generally corresponding patterns, indicating that viral gp70's and plasma-membrane gp70's inherited independently of virus production are closely related or identical molecules. The gp70 maps of thymocytes from B6, B6-G(IX)+, 129, and A mice resembled the gp70 map of cultured cells infected with xenotropic virus, whereas thymocytes from AKR mice and the two A-strain leukemias yielded gp70 maps conforming more to the pattern of cultured cells infected with ecotropic virus. The data indicate that the G(IX)-gp70 and O-gp70 subspecies of gp70 expressed in the cell populations studied are coded by xenotropic viral genomes and that X-gp70 is coded by ecotropic viral genomes. (14 refs)

78-2791 **Gene Order of the Mouse Mammary Tumor Virus Glycoproteins.** (Eng) Schochetman, G. (Viral Oncology Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501); Oroszlan, S.; Arthur, L.; Fine, D. *Virology* 83(1): 72-83; 1977.

Immunochemical and tryptic peptide mapping techniques were used to determine the derivation and gene order of the mouse mammary tumor virus (MMTV) envelope glycoproteins gp52 and gp36. According to these techniques, gp52, gp36, and p27 are distinct components. The following evidence indicated that gp52 and gp36 are derived from a common glycosylated precursor polypeptide of 75,000 daltons (gpPr75) representing the MMTV *env* gene: (1) monospecific antisera directed against both gp52 and gp36 each precipitated gPr75; (2) during pulse-chase experiments, a decrease in the amount of gPr75 was always accompanied by an increase in the amount of gp52 and gp36; (3) polypeptide gPr75 incorporated ^{3}H-glucosamine; and (4) kinetic analysis of MMTV protein synthesis demonstrated that gp52 and gp36 were synthesized sequentially, not simultaneously. The gene order of gp52 and gp36 was deduced from three different types of experiments: measurement of the differential inhibition by NaCl hypertonic shock on initiation of gp52 and gp36 synthesis; measurement of the kinetics of appearance of various MMTV proteins following the synchronized reinitiation of polypeptide synthesis resulting from NaCl hypertonic shock; and analysis of the polypeptides released from polyribosomes after a series of variable-length short pulses with ^{3}H-amino acids. The results indicate that the gene order for gPr75 within the MMTV genome is H_2N-gp52-gp36-COOH and 5′-gp52-gp36-3′. (18 refs)

78-2792 **In Vitro Transcription of Mouse Mammary Tumor Virus RNA into DNA.** (Eng) Taylor, J. M. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA, 19111); Illmensee, R.; Young, H. A. *J Virol* 25(2): 693-694; 1978.

Polyacrylamide gel electrophoresis of [^{32}P]-deoxy-ATP-labeled nucleic acids extracted from detergent-labeled virions showed that the RNA-directed DNA synthesis of B-type mouse mammary tumor virus is similar to that of the avian leukosis-sarcoma and murine leukemia viruses. The synthesis can be initiated on an RNA primer molecule approx the size of a transfer RNA, and a unique linkage (rA-dC) exists between this primer and the DNA product. (6 refs)

78-2793 **Expression of Antigens and Antibody to Mouse Mammary Tumor Virus (MMTV) in Low Mammary Tumor Strain Mice Carrying Endogenous MMTV (Meeting Abstract).** (Eng) Fine, D. L. (Frederick Cancer Res. Center, Frederick, MD, 21501); Arthur, L. O.; Smith, G. H. *Proc Am Assoc Cancer Res* 19: 111; 1978. (no refs)

78-2794 **Type-specific and Group-specific Antigenic Determinants on the Major External and Internal Proteins of Mouse Mammary Tumor Viruses (MMTVs) (Meeting Abstract).** (Eng) Teramoto, Y. A. (NCI, Bethesda, MD, 20014); Schlom, J. *Proc Am Assoc Cancer Res* 19: 14; 1978. (no refs)

78-2795 **Cell Transformation by the Mouse Mammary Tumor Virus as Evaluated by Agar-Base Techniques (Meeting Abstract).** (Eng) Lasfargues, E. Y. (Inst. Medical Res., Camden, NJ, 08103); Lasfargues, J. C.; Robinson, C. S. *In Vitro* 14(4): 361; 1978. (1 ref)

78-2796 **Cellular Immunity to Mouse Mammary Tumor Virus (MTV) in Normal and Tumor-bearing C3H Mice (Meeting Abstract).** (Eng) Tagliabue, A. (Lab Immunodiagnosis, NIH, Bethesda, MD, 20014); Herberman, R.; McCoy, J. *Proc Am Assoc Cancer Res* 19: 56; 1978. (no refs)

78-2797 **Characterization of Natural and Induced Immune Responses to Mouse Mammary Tumor Virus (MMTV) in Mice (Meeting Abstract).** (Eng) Arthur, L. O. (Frederick Cancer Res. Center, Frederick, MD, 21501); Fine, D. L.; Gardner, M. B. *Proc Am Assoc Cancer Res* 19: 111; 1978. (no refs)

78-2798 **Mouse Mammary Tumor Virus and Murine Leukemia Virus Cell Surface Antigens on Mammary Epithelial Tumor Cells (Meeting Abstract).** (Eng) Schochetman, G. (Viral Oncology Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501); Fine, D. L.; Massey, R. *Proc Am Assoc Cancer Res* 19: 110; 1978. (no refs)

78-2799 **Replication of the Mouse Mammary Tumor Virus in Human Cells (Meeting Abstract).** (Eng) Lasfargues, E. Y. (Inst. Medical Res., Camden, NJ, 08103); Coutinho, W. G. *Proc Am Assoc Cancer Res* 19: 188; 1978. (no refs)

78-2800 **High Leukemia Incidence in a Recombinant Inbred Strain (Meeting Abstract).** (Eng) Taylor, B. A. (Jackson Lab., Bar Harbor, ME, 04609); Meier, H.; Bedigian, H. G. *Proc Am Assoc Cancer Res* 19: 176; 1978. (no refs)

78-2801 **Induction of Malignant Brain Tumors in Rats by Inoculation of a Mouse Erythroblastosis Virus (Meeting Abstract).** (Eng) Hekmatpanah, J. (Chicago, IL); Quigley, J.; Weber, D. *Neurology (Minneap)* 28(4): 405; 1978. (no refs)

78-2802 **Effect of Pseudotype on Abelson Virus and Kirsten Sarcoma Virus-induced Leukemia.**

(Eng) Scher, C. D. (Sidney Farber Cancer Inst., Boston, MA, 02115). *J Exp Med* 147(4): 1044-1053; 1978.

To study the effect of virus pseudotype on leukemogenesis, a newborn Swiss or DBA/2 mice were inoculated with Abelson murine leukemia virus (A-MuLV) or Kirsten sarcoma virus (KiSV) rescued from nonproducer cells with several N- or NB-tropic helper viruses. The Friend, Moloney, and Rauscher (FMR) group of MuLV pseudotypes of KiSV induced an erythroid leukemia efficiently, whereas an endogenous helper pseudotype (N35-MuLV) did not. The FMR pseudotypes of A-MuLV induced Abelson lymphoid leukemia, but N35-MuLV and a Kirsten leukemia virus (Ki-MuLV) pseudotype did not. Pseudotypes of A-MuLV were also used to infect bone marrow cells of Fv-1nn mice in vitro. The FMR pseudotypes transformed bone marrow cells at a 40- to 100-fold higher frequency than did the N35-MuLV and Ki-MuLV pseudotypes. Mixing experiments demonstrated that the addition of an effective helper, such as M-MuLV, did not enhance lymphoid transformation by ineffective A-MuLV(N35-MuLV). Isolation of an A-MuLV-transformed clone of nonproducer lymphoid cell indicated that the A-MuLV component of A-MuLV(M-MuLV) was responsible for hematopoietic cell transformation. The data indicate that the pseudotype of A-MuLV determines its ability to transform hematopoietic cells. (24 refs)

78-2803 **The Effect of Helper Virus on Abelson Virus-induced Transformation of Lymphoid Cells.** (Eng) Rosenberg, N. (Dept. Pathology, Tufts Univ. Sch. Medicine, Boston, MA, 02111); Baltimore, D. *J Exp Med* 147(4): 1126-1141; 1978.

The oncogenic activity and transforming ability of Abelson murine leukemia virus (A-MuLV) prepared with several different helper viruses were tested to determine the role of helper virus in A-MuLV-induced transformation. Transforming ability was determined in vitro using bone marrow cells from 4- to 6-wk-old mice, and oncogenicity was determined by injection of virus into NIH/Swiss, BALB/cAN, CD-1/Swiss, and C57BR/cdJ mice. All of the A-MuLV stocks transformed NIH/3T3 or BALB/3T3 fibroblasts efficiently, indicating that all of the murine retroviruses tested were equivalently effective helpers. However, only A-MuLV stocks prepared with highly oncogenic helper viruses were able to transform hematopoietic cells efficiently in vitro and in vivo. A-MuLV prepared with weakly oncogenic helpers was highly inefficient as a transforming agent in vivo and in vitro. Inefficient helpers were also unable to establish a stable infection in lymphoid nonproducer cells. The results suggest that the helper virus may play a more active role in lymphoid cell transformation than in fibroblast transformation. (30 refs)

78-2804 **Existence of a Reverse Transcriptase-P30 Complex in AKR Mice with a High Incidence of Spontaneous Lymphocytic Leukemia (Meeting Abstract).** (Eng) Bandyopadhyay, A. K. (NIH, NCI, Baltimore Cancer Res. Center, Baltimore, MD, 21211); Mardiney, M. R.; Levy, C. C. *Proc Am Assoc Cancer Res* 19: 220; 1978. (no refs)

78-2805 **Oncogenicity of AKR Endogenous Leukemia Viruses.** (Eng) Nowinski, R. C. (Fred Hutchinson Cancer Res. Center, Seattle, WA, 98104); Hays, E. F. *J Virol* 27(1): 13-18; 1978.

On the basis of infectious properties in tissue culture, four biologically distinct types of endogenous murine leukemia virus (MuLV) have been isolated from AKR mice. They include: (1) ecotropic XC^+ MuLV, which occurs in high titer in normal tissues and serum of AKR mice throughout their life-span; (2) ecotropic XC^- MuLV, produced in high titer by leukemia cells; (3) xenotropic MuLV, readily demonstrated only in aged mice; and (4) polytropic MuLV, which arises in the thymuses of aged mice as a result of genetic recombination between ecotropic and xenotropic MuLV. All four types can be isolated from the thymuses of leukemic mice. However, leukemia cell lines in culture tend to produce a predominance of type 2 virus, suggesting that the other types may be produced by nonleukemic cells in the thymus. Isolates of types 2 and 4 virus had high oncogenic activity in AKR mice: 100% developed leukemias within 90 days of inoculation. In contrast, types 1 and 3 did not demonstrate oncogenic activity. The results show that there are fundamental differences between XC^- and XC^+ ecotropic MuLV and that ecotropic XC^- and polytropic MuLV are involved in the spontaneous leukemogenesis of AKR mice. It is hypothesized that the accelerated induction of leukemia in these mice is the result of in vivo interaction between viruses and that XC^- MuLV, because of its inefficient replication properties, preferentially recombines with xenotropic MuLV to yield an efficiently replicating polytropic MuLV that is highly oncogenic. (13 refs)

78-2806 **High-Molecular-Weight RNAs of AKR, NZB, and Wild Mouse Viruses and Avian Reticuloendotheliosis Virus All Have Similar Dimer Structures.** (Eng) Bender, W. (Div. Biology, Dept. Chemistry, California Inst. Technology, Pasadena, CA, 91125); Chien, Y. H.; Chattopadhyay, S.; Vogt, P. K.; Gardner, M. B.; Davidson, N. *J Virol* 25(3): 888-896; 1978.

Tumor viral 50S-70S RNA's from the AKR-L1 strain of ecotropic murine leukemia virus, NZB murine xenotropic virus, an ecotropic and an amphotropic virus isolated from a wild mouse, and avian reticuloendotheliosis virus (REV) were studied to determine the degree of homology of their dimer structures. The RNA's of AKR, NZB, the ecotropic wild mouse virus, and REV each showed a single band on agarose gel corresponding to estimated molecular lengths of 9.6, 9.5, 9.3, and 9.2 kilobases, respectively. Amphotropic wild mouse virus RNA was not present in sufficient quantities for electrophoresis. All five 50S-70S RNA's had similar 5′-5′ dimer structures. These observations support the hypothesis that the dimer linkage is a structural feature common to all C-type mammalian viruses. All of the mammalian viral RNA's, but not those of REV, demonstrated symmetrically placed loops in each subunit of the dimer. Thermal stability measurements with REV RNA indicated that at 55 C, 90% of the RNA still migrated as 50S-70S, but that at 65 C, the RNA was 90% dissociated to 35S. With NZB, heating to 80 C did not affect migration, but heating to 100 C disrupted all migration. The RNA's of AKR and the wild mouse viruses were not available for thermal stability measurements. (30 refs)

78-2807 **Properties of Mouse Leukemia Viruses. XV. Electron Microscopic Studies on the Organization of Friend Leukemia Virus and Other Mammalian C-Type Viruses.** (Eng) Frank, H. (Max-Planck-Institute fur Virusforschung, Spemannstrasse 35, D-7400 Tubingen 1, W. Germany); Schwarz, H.; Graf, T.; Schafer, W. *Z Naturforsch C* 33(1/2): 124-138; 1978.

The structural organization of various mammalian C-type viruses was investigated. Investigations of Friend leukemia virus (FLV) revealed that transition from the native to the collapsed form occurs mainly during preparation for electron microscopy. A short pretreatment of the virus with the detergent Nonidet P40 prior to negative staining with uranyl acetate preserved the native structure in a high percentage of virus particles from standard preparations. This technique was then used to study the internal organization of the virus. A thin inner coat was detected in close apposition to the viral membrane between the viral envelope and core. Removal of the unit membrane by more intensive detergent treatment revealed the existence of material extending between the viral surface knobs and the viral interior. The icosahedral core shell had an opening at one side and this corresponded to an opening in the helically arranged ribonucleoprotein strand representing the innermost structure. A comparative study of the structures of representative mammalian C-type viruses revealed that they were similar to that of Friend virus; differences in stability, however, were observed. It is thus suggested that C-type viruses contain knobs known to represent the virus glycoprotein and that these knobs are anchored in the viral membrane. The inner coat is either inside this viral membrane or is part of it. Between the envelope and the core lies the material that apparently links the knobs to the core. The core shell contains an opening representing the site of the shell synthesized last during budding. It is suggested that the ribonucleoprotein represents a wound-up thread forming a helical structure. Its opening is closely apposed to the opening in the core shell. (32 refs)

78-2808 **Identification of a Gene (Fv-3) Which Regulates Susceptibility to Immunosuppression by Friend Virus Complex (FV) (Meeting Abstract).** (Eng) Kumar, V. (Dept. Pathology, Boston Univ. Sch. Medicine, Boston, MA, 02118); Bennett, M. *Fed Proc* 37(6): 1570; 1978. (no refs)

78-2809 **Variations in Viral Gene Expression in Friend Virus-transformed Cell Lines Congenic with Respect to the *H-2* Locus.** (Eng) Freedman, H. A. (Dept. Pathology, State Univ. New York, Downstate Medical Center, Brooklyn, NY, 11203); Lilly, F.; Strand, M.; August, J. T. *Cell* 13(1): 33-40; 1978.

The effect of *H-2* genotype on the genetic expression of Friend oncovirus was studied using Friend virus (FV)-transformed cells differing genetically only at the *H-2* locus. Seven lines and three sublines were derived from solid tumors obtained by sc transplantation of fragments from the enlarged spleens of FV-infected mice to syngeneic recipients. Upon serial passage, the *H-2b*, *H-2g*, and *H-2kb* cell lines were stable for the production of infectious FV, whereas the *H-2a* and *H-2k* cell lines stopped releasing infectious virus after a relatively short time. Synthesis of viral proteins by these cell lines was independent of the synthesis of infectious virus in that some viral proteins continued to be synthesized by *H-2a* and *H-2k* in the absence of infectious virus. The temporal sequence of change suggests that the loss of protein expression may have occurred sequentially: reverse transcriptase was reduced first, followed by the structural proteins p30 and p15, and then the envelope glycoprotein gp69/71. While p30 and p15 were coordinately expressed, p30 and gp69/71 were not, indicating that these proteins are regulated by independent genetic loci. Loss of viral genes did not occur, because cell lines that had previously ceased to synthesize viral proteins could be chemically induced to produce infectious FV. These data suggest that for these *H-2*-congenic cell lines, there is an association of the *H-2Db* allele and the long-term production and release in vitro of both infectious virus and viral proteins. (27 refs)

78-2810 **Studies on the Nature of the Cell Surface Antigen Reacting with Cytolytic T Lymphocytes in Murine Oncornavirus-induced Tumors.** (Eng) Gomard, E. (Laboratoire d'Immunologie et de Virologie des Tumeurs, INSERM U 152, Hopital Cochin, 27 Rue du Faubourg Saint Jacques, F-75674 Paris Cedex 14, France); Levy, J. P.; Plata, F.; Henin, Y.; Duprez, V.; Bismuth, A.; Reme, T. *Eur J Immunol* 8(4): 228-236; 1978.

In the murine sarcoma virus (MSV) system, the activity of the tumor cell surface antigen that reacts with cytolytic T lymphocytes (CTL) is under histocompatibility (H-2) antigen restriction. This was shown in systematic experiments, including chromium-release tests lasting 3-4 and 16-20 hr. With the 16- to 20-hr tests, however, quantitative methods were necessary to demonstrate H-2 restriction. These results support the hypothesis that H-2 molecules may be determinants in the structure and/or function of the CTL reactive antigen. Under syngeneic conditions (ie, using H-2-identical immune lymphocytes, stimulators, and target cells), the pattern of specificities recognized by anti-MSV or anti-Friend leukemia virus T cells on 20 different lymphomas suggested that the main reactive antigen is an 'FMR-like' (Friend, Moloney, Rauscher) substance. Identical conclusions were drawn from competition experiments. The CTL-mediated reaction was blocked by preincubation of target cells with goat antisera against the viral glycoprotein gp70, which suggested an association between the FMR antigen and gp70. However, this might have been due to nonspecific reactions, as two other gp70 antisera and antisera against the viral components gp45, pr60, p30, p15, p12, and p10 lacked blocking activity. The CTL/tumor cell interaction was not inhibited by virion-associated antigens added to the medium. Lysostrip and cocapping experiments failed to reveal an association between H-2 and gp70. The nature of the viral protein bearing the 'FMR-like' substance, therefore, remains to be established. (48 refs)

78-2811 **Virulence of Clones of Friend Leukemia Virus Complex Expressing Different Immunosuppressive Properties (Meeting Abstract).** (Eng) Eckner, R. J. (Boston Univ. Sch. Medicine, Boston, MA, 02118); Scherzer, L. J. *Fed Proc* 37(6): 1570; 1978. (no refs)

78-2812 **Rfv-1 and Rfv-2, Two H-2-associated Genes that Influence Recovery from Friend Leukemia Virus-induced Splenomegaly.** (Eng) Chesebro, B. (Dept. Health, Education and Welfare, Rocky Mountain Lab., Natl. Inst. Allergy and Infectious Diseases, Hamilton, MT, 59840); Wehrly, K. *J Immunol* 120(4): 1081-1085; 1978.

The influence of genes of the mouse major histocompatibility complex (*H*-2) on recovery from Friend murine leukemia virus (FV)-induced leukemia was studied using the congeneic F_1 hybrid strains differing only at genes of the *H*-2 complex and *Tla* region. Two genes influenced recovery from FV-induced leukemia: *Rfv*-1 and *Rfv*-2. *Rfv*-1 was located to the left of the *Tla* region within the *H*-2 complex, specifically, in the *H-2G* or *H-2D* regions. *Rfv*-2 was located in the *H*-2 *K* or *H-2I* regions or in the *Tla* region. Since the *Rfv*-2 effect was not seen in *H-2D(Rfv-1)b/b* mice, which show a high recovery rate from FV leukemia, this gene may have a weaker influence than *Rfv*-1 on recovery from leukemia. The *H*-2 effect on recovery appeared to function in a gene-dose fashion. This effect may act on the kinetics of induction in virus-specific cytotoxic T cells. (29 refs)

78-2813 **Transformation-dependent Secretion of a Low Molecular Weight Protein by Murine Fibroblasts.** (Eng) Gottesman, M. M. (Lab. Molecular Biology, NCI, NIH, Bethesda, MD, 20014). *Proc Natl Acad Sci USA* 75(6): 2767-2771; 1978.

The major excreted proteins from transformed and untransformed murine cells were characterized by a pulse-chase protocol that radiolabels proteins secreted by cells in culture. A radiolabeled 35,000-mol wt polypeptide was released into the medium in relatively large amounts (0.1%-1% of total protein synthesized) by Kirsten virus-transformed NIH Swiss 3T3 cells but in much smaller amounts (≤ 0.02% of total protein synthesized) by untransformed 3T3 cells. In experiments performed to establish whether secretion of this major excreted polypeptide (MEP) is related to transformation, it was found that BALB/c cell strains transformed spontaneously or by RNA tumor viruses (Kirsten, Moloney sarcoma viruses), a DNA tumor virus (simian virus 40), or by methylcholanthrene all excreted relatively large amounts of MEP. In contrast, the parental cell line secreted barely detectable levels of this polypeptide. MEP appearance seemed to correlate well with anchorage independence in the transformed cells. Indirect immunofluorescence showed that MEP was localized within the cytoplasm of the transformed cells and that it is not a cell-surface protein. The presence of MEP may serve as a biochemical marker of transformation. A biological role for this protein has not been assigned. (21 refs)

78-2814 **High Molecular Weight Viral Reverse Transcriptase in Moloney Sarcoma Virus Transformed Cells.** (Eng) Rokutanda, M. (Dept. Biochemistry, Tokyo Metropolitan Inst. Medical Science, Tokyo 113, Japan); Maeda, Y.; Watanabe, N. *Biochem Biophys Res Commun* 80(4): 729-734; 1978.

Viral reverse transcriptase (RT) was isolated and purified from Moloney sarcoma virus-transformed rat cells by ion-exchange chromatography and density gradient centrifugation. The time course of the mol wt shift of the viral RT from the cytoplasmic form to the virion form was examined by harvesting the virus after 20 min, 2 hr, and 4 hr incubation and determining the mol wt of the enzyme from glycerol gradient centrifugation. The cytoplasmic RT and the RT from the virions harvested at 20 min had a mol wt of 105,000. A small amount of a 70,000-mol wt enzyme was also included in the 20-min sample. At 2 hr incubation, the enzyme consisted mainly of a 70,000-mol wt protein and a small amount of a 105,000-mol wt protein. At 4 hr incubation, the mol wt of the viral RT was 70,000. It is suggested that the viral RT was present in the cytoplasm as the high-mol-wt form and that maturation of the enzyme (to the low-mol-wt form) occurs in the virus particles immediately after budding of the particles from the cell surface. (8 refs)

78-2815 **Large T1 Oligonucleotides of Moloney Leukemia Virus Missing in an *env* Gene Recombinant, HIX, Are Present on an Intracellular 21S Moloney Viral RNA Species.** (Eng) Faller, D. V. (Biology Dept., Massachusetts Inst. Technology, Cambridge, MA); Rommelaere, J.; Hopkins, N. *Proc Natl Acad Sci USA* 75(6): 2964-2968; 1978.

HIX, a dual tropic virus whose ecotropic parent is Moloney leukemia virus, has an envelope glycoprotein different from that of Moloney virus. HIX and Moloney viruses share most of the large T1 oligonucleotides derived from their genomes, but each possesses a set of distinctive oligonucleotides that lie clustered in corresponding regions in the 3′ halves of their oligonucleotide maps. These regions presumably contain envelope glycoprotein coding sequences. The C-type viral envelope glycoprotein is believed to be translated from a 21S RNA. Thus, at least part of the region of the Moloney virus genome that is altered relative to HIX was expected to be present on such a species. To test this prediction, an intracellular 21S Moloney viral RNA species was purified and its large T1 oligonucleotides were analyzed by two-dimensional polyacrylamide gel electrophoresis. This RNA contains one T1 oligonucleotide that is probably derived from the 5′ end of the Moloney virus genome, the Moloney virus T1 oligonucleotides that are missing in HIX, and those that lie to their 3′ side on the Moloney virus T1 oligonucleotide map. (25 refs)

78-2816 **Oncornavirus-induced Autoaggression and Lymphomagenesis: The Obverse of Altered-Self?** (Eng) Proffitt, M. P. (Dept. Immunology, Cleveland Clinic Foundation, Cleveland, OH, 44106); Vessey, A. R.; Del Villano, B. C. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 84-87; 1978.

Normal C3H/He mouse embyro cells at low passage levels ($P < 25$) were consistently killed by thymocytes from adult C3H/HeJ mice infected with Moloney murine leukemia virus (M-MuLV) since birth. However, the target cells lost their susceptibility to killing at high passage levels ($P > 30$). This resistance to the M-MuLV-carrier thymocytes correlated with the spontaneous expression of the viral envelope glycoprotein gp70 and of infectious ecotropic MuLV by the target cells. Possible factors involved in these phenomena are discussed. (12 refs)

78-2817 **Selective Amplification of Murine Sarcoma Virus Proviral DNA in Productively Infected Mammalian Cells.** (Eng) Frankel, A. E. (NCI, Bethesda, MD, 20014); Fischinger, P. J. *In: Advances in Comparative Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 171-173; 1978.

The mechanisms by which helper viruses augment the morphologic transformation of various mammalian cell lines by Moloney murine sarcoma virus (M-MSV) were studied. Complementary DNA (cDNA)-cellular DNA hybridizations were performed using MSV-murine leukemia virus cDNA for cat and dog systems and MSV-specific cDNA for human and mouse systems. Human sarcoma positive, leukemia negative cells (S+L- cells) and cat, dog, or mouse cells transformed by M-MSV contained 1-2 copies of M-MSV proviral DNA. In each system, the number of MSV proviral DNA copies increased from three- to tenfold after superinfection with helper viruses, including ecotropic mouse leukemia virus, feline leukemia and feline endogenous xenotropic viruses, and simian sarcoma-associated virus. In cells that did not contain leukemia-related sequences, helper virus infection resulted in the appearance of only a few copies of the helper leukemia proviral DNA. The amplification of M-MSV DNA by helper virus superinfection was accompanied by increases in MSV RNA and viral proteins and in the degree of morphological transformation. (1 ref)

78-2818 **Amino-Terminal Amino Acid Sequence and Carboxyl-Terminal Analysis of Rauscher Murine Leukemia Virus Glycoproteins.** (Eng) Henderson, L. E. (Viral Oncology Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501); Copeland, T. D.; Smythers, G. W.; Marquardt, H.; Oroszlan, S. *Virology* 85(1): 319-322; 1978.

The results of amino acid analyses, NH_2-terminal sequence determinations, and carboxypeptidase digestions of Rauscher murine leukemia virus glycoproteins gp80 and gp45 are presented. The two gp had the following identical NH_2-terminal amino acid sequences: Ala-Ala-Pro-Gly-Ser-Ser-Pro His-Gln-Val-Tyr-X-Ile-Thr-X-Glu-Val- (X, unidentified) The COOH-terminal amino acid is tyrosine for gp7 and leucine for gp45. Computer-analyzed amino aci compositional data indicated that the protein component in gp45 is smaller (approx 36,000) than that n gp70. These data provide compelling evidence that gp45, which also contains less carbohydrate, is derived from gp70 through proteolysis that may or may not be accompanied by glycolytic cleavage(s). The alternative that gp45 may be generated from the nonglycosylated or partially glycosylated precursor cannot be excluded completely. The results suggest that the two gp are not encoded separately in he viral genome. (25 refs)

8-2819 **Impaired Processing of Precursor Polypeptides of Temperature-sensitive Mutants of Rauscher Murine Leukemia Virus.** (Eng) van de Ven, W. J. (Dept. Biochemistry, Univ. Nijmegen, Nijmegen, Netherlands); van Zaane, D.; Onnekink, C.; Bloemers, H. P. *J Virol* 25(2): 553-561; 1978.

The synthesis and processing of virus-specific polypeptides n NIH/3T3 cells infected at the permissive temperature (31 C) with temperature-sensitive (*ts*) mutants of Rauscher murine leukemia virus (R-MLV) were studied in pulse-chase experiments at the permissive and nonpermissive (39 C) temperatures. The viral polypeptides were identified by sodium dodecyl sulfate-polyacrylamide gel electrophoresis after immunoprecipitation with polyvalent and monospecific anti-R-MLV sera. In cells infected with *ts* mutants defective in early replication steps (*ts*17 and *ts*20) or *ts* mutants defective in postintegration steps (*ts*25 and *ts*26), the processing of the primary *gag* gene product was impaired at 39 C. In all four mutants, *gag*-pr75 was converted into *gag*-pr65; however, *gag*-pr65 accumulated at 39 C, and the main internal virion polypeptide p30 was not formed. Therefore, he proteolytic cleavage was blocked beyond *gag*-pr65. Concomitantly, the formation of the *env* gene-related polypeptide p12(E) of all four mutants was blocked at the restrictive temperature. In contrast, cells inected with the late mutant *ts*28, which produced nfectious virions at 39 C, showed a normal turnover of the *gag* and *env* precursor polypeptides. (27 refs)

78-2820 **Virion Precipitation Test Based on the Determination of Reverse Transcriptase Activity: Analysis of Oncornavirus Surface Antigens.** (Rus) Zakharova, L. G. (D. I. Ivanovsky Inst. Virology, Moscow, USSR); Al'tshtein, A. D. *Vopr Virusol* (1): 19-24; 1978.

A new method for assessing oncornavirus surface antigens s described. The concentrates of a series of 11 oncornaviruses Rauscher murine leukemia virus, Gross murine leukemia virus, endogenous virus from NIH Swiss mice, exogenous eline leukemia virus, Rous sarcoma virus, murine mammary umor virus, etc.) showing marked reverse transcriptase acivity were incubated with immune goat sera against the test viruses. The precipitate was centrifuged, and the supernatant was tested in the reverse transcriptase reaction. Incubation with immune sera resulted in the precipitation of 80%-100% reverse transcriptase activity (compared with 22%-42% precipitation after incubation with normal sera). Immune sera precipitated only homologous virus. (5 refs)

78-2821 **Studies on Aminoglycolipid Contents and Their Synthesis in Hamster Cell Lines. Polyoma-transformed BHK Cell Lines with Low β- and High α-N-Acetylgalactosaminyltransferase Activities.** (Eng) Yokosawa, N. (Biochemical Lab., Cancer Inst., Hokkaido Univ. Sch. Medicine, Sapporo 060, Japan); Kijimoto-Ochiai, S.; Makita, A. *Biochim Biophys Acta* 528(1): 138-147; 1978.

The aminoglycolipid contents of baby hamster kidney (BHK) cells and polyoma (Py)-transformed BHK cells were investigated and compared with those of NIL-2K hamster cells. The amount of globoside in the BHK cells was larger than that in the BHK-Py cells, but thin-layer chromatography indicated that both lines contained little Forssman hapten. Hemolysis inhibition studies indicated that Forssman hapten was synthesized in BHK and BHK-Py cells, but that the amount was small in comparison with NIL-2K and NIL-Py cells. When glycolipid β- and α-N-acetylgalactosaminyltransferases were assayed with enzyme preparations, BHK-Py cells showed little β-transferase (globoside synthesis) activity and elevated α-transferase (Forssman hapten synthesis) activity compared with the parent BHK cells. This suggested that the low β-transferase activity results in low levels of both the aminoglycolipids. The properties of the α-transferase of both lines were similar. There was no inhibitor of this enzyme in BHK cells and no activator in BHK-Py cells. (26 refs)

78-2822 **Increased Trypsin Sensitivity of Cell Surface Macromolecules after Malignant Transformation.** (Eng) Latner, A. L. (Cancer Res. Unit, Univ. Dept. Clinical Biochemistry, Royal Victoria Infirmary, Newcastle upon Tyne, England); Turner, G. A. *Exp Cell Biol* 46(5): 298-309; 1978.

The effect of standard trypsin treatment (STT: 400 μg/ml for 3 hr) on the electrophoretic mobility (EPM) of baby hamster kidney (BHK21) cells and their polyoma virus-transformed counterparts (Py6 cells) was investigated. In a previous study, these conditions had resulted in the release of material from the Py6 cells, but not the BHK21 cells. STT increased the mean EPM of the transformed cells significantly, from -2.07 to -2.41 μm·sec^{-1}· V^{-1}·cm. This change was not observed when the cells were incubated in the absence of trypsin, and STT of the BHK21 cells did not change their mean EPM. Increasing the incubation time to 6 hr had no significant effect, but decreasing this time to 1 hr gave a mean EPM that was the same as that of untreated cells. Treatment of Py6 cells with 200 μg/ml trypsin still increased their EPM significantly, but to a significantly lower degree than that produced by STT. Active enzyme was required to produce the change. No evidence was obtained for cytolysis, cytotoxicity, or uptake of the enzyme by the treated Py6 cells. Measurement of the EPM of the Py6 cells at different pH levels before and after treatment suggested that the enzyme was removing cationic groupings from the cell surface. The increase in EPM of Py6 cells after STT may be the result of a direct enzyme attack on molecules that contribute to the EPM of the cells. The trypsin-sensitive positive component of the cell-surface charge, present only on the transformed cells, may be important in the expression of the malignant characteristics of the cancer cell. (40 refs)

78-2823 **Endogenous Guinea Pig Retrovirus: Its Expression in Leukemic Guinea Pig Cells.** (Eng) Davis, A. R. (Dept. Microbiology and Immunology, Univ. California Sch. Medicine, Los Angeles, CA, 90024); Nayak, D. P.; Lofgren, J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 176-178; 1978.

Molecular hybridization of viral complementary DNA to cellular RNA was used to study the expression of guinea pig retrovirus ([5-bromodeoxyuridine (BUdR)-induced GPV]) in L_2C leukemic lymphoblasts. Although the leukemic cells contained fewer copies of viral RNA per cell than the BUdR-induced cells, the results indicated the essentially complete expression of the BUdR-induced GPV genes in the leukemic cells. Both BUdR-induced cells and L_2C lymphoblasts contained 14S, 22S, 35S, and 70S RNA species of total and cytoplasmic virus-specific RNA. In addition, BUdR-induced GPV-specific messenger RNA (mRNA) was present in polyribosomes and leukemic lymphoblasts: 14S, 20S, and 35S mRNA species, the same as those observed in BUdR-induced cells, were identified. (8 refs)

78-2824 **The Frequency of Occurrence of Feline Leukaemia Virus Subgroups in Cats.** (Eng) Jarrett, O. (Dept. Veterinary Pathology, Veterinary Sch., Univ. Glasgow, Glasgow G61 1QH, Scotland); Hardy, W. D.; Golder, M. C.; Hay, D. *Int J Cancer* 21(3): 334-337; 1978.

Feline leukemia viruses (FeLV) were isolated from cats in Glasgow and New York with lymphosarcoma and from apparently healthy carrier cats, and the subgroup composition of each isolate was determined. The majority of isolates consisted of either FeLV-A alone or FeLV-A and B; FeLV-C was rare, and it occurred in mixtures that included FeLV-A. Of healthy cats, 65% had FeLV-A alone and 33% had FeLV-AB, but of leukemic cats, 42% had FeLV-A and 58% had FeLV-AB. This same pattern was noted in both Glasgow and New York. No correlation was noted between a particular virus subgroup and any of the common types of lymphosarcoma or leukemia in cats. FeLV-C was found only in one cat with leukemia and two others with aplastic anemia. The viruses isolated from the two latter were mixtures of subgroups A and C and A, B, and C respectively. Isolates were also studied in cats from 25 British multiple-cat households (MCH). In 15 MCH, FeLV-A alone was isolated; in 10, isolates containing either FeLV-A or FeLV-AB were found. Of the MCH in which FeLV-AB was found, half yielded FeLV-A and half yielded FeLV-AB. Overall, the proportion of FeLV-infected cats in MCH was 42%. In MCH in which only FeLV-A was isolated, 28% were FeLV-positive, but in MCH with FeLV-AB, 53% of the cats were infected. (9 refs)

78-2825 **Dissemination of Bovine Oncornavirus among Cattle from Leukemic Herds.** (Rus) Valikhov, A. F. (All-Union Inst. Experimental Veterinary Medicine, Moscow, USSR); Burba, L. G.; Nakhmanson, V. M.; Zinevich, L. A.; Kozin, A. F.; Golovchenko, A. P.; Guliukin, M. I.; Ezovitov, N. B.; Borodin, A. G. *Veterinariia* (3): 40-43; 1978.

Cattle from herds with a high incidence of leukemia (up to 57%) were investigated for antibodies to oncornavirus. Antibodies were found in 281/769 animals in one herd and in 290/2,412 in another. Most of the positive animals were 3-5 yr old. However, only 20% of the oncornavirus-infected animals had leukemia; most of these animals were 3-4 yr old. The hematological parameters of most of the virus-infected animals were normal. (no refs)

78-2826 **Infection of Human Cell Cultures with Bovine Visna Virus.** (Eng) Georgiades, J. A. (Rega Inst. Medical Res., Univ. Leuven, Minderbroedersstraat 10, B-3000 Leuven, Belgium); Billiau, A.; Vanderschueren, B. *J Gen Virol* 38(2): 375-381; 1978.

The possibility that bovine visna virus (BVV) could infect and transform human cells was investigated using fibroblastoid cell cultures derived from the bone marrow of five humans with various forms of leukemia. Cell cultures were inoculated with BVV by mixing freshly trypsinized cells with an undiluted cell-free BVV preparation. After 2 mo of subcultivation, foci of altered cells suggestive of malignant transformation were noted. These foci were not present in noninoculated cultures. Both control and inoculated cultures had limited life-spans and could not be developed into continuous transformed cell lines. BVV genome functions could be demonstrated in the inoculated cells by immunofluorescence using a reference BVV serum, by the presence in the supernatant culture fluid of reverse transcriptase with a preference for Mg^{2+} ions, and by electron microscopic detection of cell-associated virus particles in one infected culture. However, infectious BVV could not be rescued. Compared with leukemic bone marrow cultures, normal human embryonic kidney and spleen tissue could not be infected with BVV. These findings indicate that BVV may play a role in either malignant or slow virus diseases in man and that exposure of genetically predisposed individuals could result in latent infection. Furthermore, the Mg^{2+} preference of the reverse transcriptase indicates that BVV differs from bovine syncytium virus and is closely related to Mason-Pfizer monkey virus and sheep visna virus. (20 refs)

78-2827 **Malignant Conversion of Shope Papillomas and Associated Changes of Serum Ceruloplasmin in Domestic Rabbits.** (Eng) Seto, A. (Dept. Microbiology, Faculty Medicine, Kyoto Univ., Kyoto 606, Japan); Tokuda, H.; Ito, Y. *Proc Soc Exp Biol Med* 157(4): 694-696; 1978.

Quantitative and qualitative changes of serum ceruloplasmin associated with the malignant conversion of Shope papillomas to squamous cell carcinomas were determined. The papillomas were induced in neonatal domestic rabbits by id injection of Shope papilloma virus, and they were converted into squamous cell carcinomas after a lapse of a year or more. Serum copper level before death due to malignancy was elevated two to six times compared with the level at 4 mo of age. Disk electrophoresis analysis of the serum showed that ceruloplasmin activity was manifested as single and double bands before and after malignant conversion, respectively. In one rabbit, the serum copper level was elevated twice at parturition and four times at malignant conversion compared with that of normal serum. At parturition, only a single band of ceruloplasmin appeared upon disk electrophoresis. The elevated level dropped to normal (68 μg/deciliter) at 1 mo after total tumor resection. The presence of two bands of ceruloplasmin activity in the malignant sera could be due to two distinct isozymes of ceruloplasmin. If this speculation is confirmed, the presence of ceruloplasmin isozymes could be used as a marker for the malignant conversion of benign tumors. (11 refs)

78-2828 **Characterization of Two Types of Human Papillomaviruses in Lesions of Epidermodysplasia Verruciformis.** (Eng) Orth, G. (Unite de Recherches sur l'Etiologie Virale des Cancers humains, Inst. Gustave-Roussy, Villejuif, France); Jablonska, S.; Favre, M.; Croissant, O.; Jarzabek-Chorzelska, M.; Rzesa, G. *Proc Natl Acad Sci USA* 75(3): 1537-1541; 1978.

Human papillomaviruses (HPV's) found in the lesions of 11 patients (5 men, 6 women, aged 7-45 yr) with epidermodysplasia verruciformis (EV) were compared to HPV type 1 (HPV-1) and HPV type 2 (HPV-2) previously characterized in plantar and common warts, respectively. The patients had had EV for 1-42 yr. Complementary RNA's (cRNA's) to HPV-1, HPV-2, and to the prototype viruses from two EV patients (1 and 2) were used in cRNA-DNA filter hybridization experiments. No sequence homology was detected between HPV-1 or HPV-2 DNA's and DNA's obtained from the 11 EV HPV isolates. Furthermore, with the cRNA's from patients 1 and 2, EV DNA fell into two groups showing little, if any, sequence homology. A lower extent of annealing was observed for the DNA's of some isolates showing a genetic heterogeneity within each of the two groups. Almost no antigenic cross-reaction was detected by immunodiffusion or indirect immunofluorescence tests, either between EV HPV's and HPV-1 or HPV-2, or between the HPV's of patients 1 and 2. Viruses related to the HPV of patient 1 were preferentially associated with flat wartlike EV lesions, and they were found in lesions from five patients bearing multiple flat warts. Viruses related to the HPV of patient 2 were found in the morphologically distinct lesions (red spots) present in some EV patients. It is suggested that these two types of HPV be labeled HPV-3 and HPV-4, respectively. Malignant conversion was noted in 3 patients with HPV-4, 1 with both HPV-3 and HPV-4, and 1 with HPV-3. (23 refs)

78-2829 **Characterization of a DNA-Protein Complex and Capsomere Subunits Derived from Polyoma Virus by Treatment with Ethyleneglycol-bis-N,N′-tetraacetic Acid and Dithiothreitol.** (Eng) Brady, J. N. (Div. Biology, Kansas State Univ., Manhattan, KS, 66506); Winston, V. D.; Consigli, R. A. *J Virol* 27(1): 193-204; 1978.

Polyoma virus was purified from lysates of infected mouse embryo and kidney cells. Treatment of the virions with ethyleneglycolbis-N,N′-tetraacetic acid (EGTA) and dithiothreitol (DTT) at pH 8.5 resulted in their dissociation into a DNA-protein complex and individual structural capsomere subunits. The sedimentation value of the DNA-protein complex in sucrose gradients was approx 48S, and it had a density of 1.45 g/cm³ in equilibrium CsCl gradients. Alkaline sucrose analysis of the DNA in the complex demonstrated that approx 75% of it was component 1. The proteins associated with the DNA were dissociated by treatment with NaCl or the anionic detergent Sarkosyl. VP1 and the histone proteins VP4-7 were the major proteins associated with the DNA. Treatment of the DNA-protein complex with alkaline pH resulted in the specific removal of VP1. Electron microscopy of the 48S DNA-protein complex demonstrated that it was a tightly coiled structure slightly larger than the intact virion. Treatment of the complex with NaCl or pH 10.5 buffer resulted in the loss of protein and subsequent loosening of the DNA-protein complex with the DNA becoming visible. The capsomere subunits released as a result of the EGTA-DTT treatment sedimented as 18S, 12S, and 5S subunits in sucrose gradients. Electrophoretic analysis of the isolated capsomeres demonstrated that VP1, VP2, and VP3 were present in each species, although the ratios of the proteins varied. In addition to the structural proteins, histones VP4-7 were found to be predominantly associated with the 5S capsomere subunit. (31 refs)

78-2830 **In Vitro Recombinant DNA Technology for Mapping Animal Virus Mutations (Meeting Abstract).** (Eng) Miller, L. K. (Dept. Bacteriology and Biochemistry, Univ. Idaho, Moscow, ID, 83843). *Miami Winter Symp* 14: 265; 1977. (1 ref)

78-2831 **Restriction to the Cell Nucleus of Virus-induced Host RNA's in a Rat Kidney Tumor Induced by Polyoma Virus.** (Eng) Shearer, R. W. (Issaquah Group Health and Environmental Res., 1595 N.W. Gilman Blvd., Issaquah, WA, 98027). *J Natl Cancer Inst* 60(6): 1383-1385; 1978.

A hybridization competition assay showed that the RNA transcripts of repetitive DNA of WF rat kidney, which were derepressed during transformation by polyoma virus, were not transported to the cytoplasm in the tumor cells. Transcripts of repetitive DNA found in the normal cell nucleus were all transported to the cytoplasm in the tumor cells, including those restricted to the nuclei in the normal cells. The results are contrasted with those of chemical carcinogenesis in which there is no derepression of cellular gene families. (9 refs)

78-2832 **Elevation of Alkaline Endodeoxyribonucleases in Nuclei of Polyoma Virus-infected Mouse Embryo Fibroblasts (Meeting Abstract).** (Eng) Goodwin, R. G. (Dept. Biochemistry, Univ. California, Riverside, CA, 92521); Sei, K. S.; Karu, A. E. *Fed Proc* 37(6): 1413; 1978. (no refs)

78-2933 **Electron Microscopic Evidence for Splicing of SV40 Late mRNAs.** (Eng) Bratosin, S. (Dept. Genetics, Weizmann Inst. Science, Rehovot, Israel); Horowitz, M.; Laub, O.; Aloni, Y. *Cell* 13(4): 783-790; 1978.

RNA displacement loops (R loops) were formed when poly-(A)-containing simian virus 40 (SV40) cytoplasmic RNA was hybridized with linear double-stranded SV40 DNA. Analysis of the R loops by electron microscopy confirmed the conclusions of biochemical studies that the leader sequences at the 5′ ends of the 16S and 19S late messenger RNA's (mRNA's) are not coded immediately adjacent to the main portions c the mRNA's. These data are consistent with either segmentation of the leaders or heterogeneity of their lengths. Measurements of the R-loop structures yielded the locations, on th physical map of SV40 DNA, of the bodies and leaders of th 16S and 19S late mRNA's plus the lengths of the bodie leaders, and the corresponding intervening DNA sequence (27 refs)

78-2834 **Exogenous DNA Transcription in Cells wi Their Native DNA Inhibited. III. Ribosom Changes Induced by Exogenous DNA.** (Eng) Lopez-Burill S. (Dept. Biology and Biochemistry Cancer, Instituto N cional de Oncologia, Ciudad Universitaria, Madrid-3, Spain Valladares, Y.; Alvarez-Rodriguez, Y.; Alvarez-Noves, *Rev Esp Oncol* 24(2): 287-294; 1978.

DNA transcription was studied in TC-SV40 cells (simia virus 40-induced hamster sarcoma established in vitro) i which native DNA replication had been blocked by incubation of the cells with 5′-bromodeoxyuridine (BUdR DNA isolated from Ehrlich ascites cancer (EAC) ce was injected into TC-SV40 cells. The cells were label by incubating them in the presence of ^{14}C-prote hydrolysate. Polyribosomes were isolated from e ponentially growing cells, and their optical densit radioisotopic activity, and ultrastructure were studie Logarithmically growing TC-SV40 cells and EAC ce showed a characteristic distribution of ribosom When the BUdR-blocked TC-SV40 cells were inoc lated with EAC cell DNA, there was an increase monoribosomes, an almost complete disappearance of dimers, and a diminution of protein synthesis. Thus, the ribos mal distribution of the TC-SV40 cells was changed to a pa tern typical of that for EAC cells. (6 refs)

78-2835 **Transfection of Permissive Monkey Cells wi Restriction Endonuclease-derived Fragments SV40 DNA. II. Rescue of SV40 Virions by Cell Fusion a Transfection (Meeting Abstract).** (Eng) Moyer, M. (Trini Univ., San Antonio, TX); Moyer, R.; Gerodetti, M.; Lip tich, G. *Miami Winter Symp* 14: 263; 1977. (2 refs)

78-2836 **A Single Cleavage of Simian Virus 40 (SV4 DNA by a Site Specific Endonuclease fro** *Thermus aquaticus, Taq* I. (Eng) Sato, S. (Mitsubis Kasei Inst. Life Sciences, Minamiooya, Machida, Tok 194, Japan). *J Biochem (Tokyo)* 83(2): 633-63 1978.

A site specific endonuclease from *Thermus aquaticus, Taq* cleaved simian virus 40 (SV40) DNA at a single site. Th cleavage site was localized on the physical map by double digestions using the previously characterized fragments produced by digestion with *Hae* II, *Hae* III, *Alu* I, *Hha* I, *Hinf* I, or *Bst* I followed by acrylamide gel electrophoresis. The exact *Taq* I site was estimated from the *Hae* III/*Taq* I digestion to be located on the (*Hae* III)-A fragment 135 nucleotide pairs from the left end of (*Hae* III)-B. This site corresponds to the position that is 56.5% of the unit length from the *Eco* RI site, and it is very close to the position where early and late messenger RNA start to be transcribed in opposite directions. (3 refs)

78-2837 **Transfection of Permissive Monkey Cells with Restriction Endonuclease-derived Fragments of SV40 DNA. I. Transformation and Superinfection Properties of Recipient Cells (Meeting Abstract).** (Eng) Moyer, R. C. (Trinity Univ., San Antonio, TX); Moyer, M. P.; Hurtado, H. *Miami Winter Symp* 14: 261; 1977. (no refs)

78-2838 **Evidence for "Splicing" of SV40 16S mRNA.** (Eng) Haegeman, G. (Lab. Molecular Biology, Ledeganckstraat, 35, B-9000 Ghent, Belgium); Fiers, W. *Nature* 273(5657): 70-73; 1978.

The major simian virus 40 (SV40)-specific messenger RNA (mRNA), which sediments at 16S, was characterized by comparing its fingerprint with the nucleotide sequence of the DNA. The results indicate that the 16S mRNA is coded for by at least two separate regions on the genome. The 5′-leader sequence is about 180 nucleotides in length and is transcribed from the region 0.72 to 0.76 on the standard map. The body of the mRNA starts, at most, 40 nucleotides before the initiation codon for the VP1 protein and contains the whole structural VP1 gene, followed by the untranslated 3′-terminal end. It thus extends clockwise from coordinates 0.94 to 0.17. There is no evidence that the two parts of the RNA molecule are joined by anything other than a normal phosphodiester linkage. It is not known whether the splicing occurs during transcription or is the result of a subsequent excision-ligation event, but the latter mechanism seems more likely. The concept of splicing in the formation of eukaryotic mRNA may partly explain the complex processing events undergone by heterogeneous nuclear RNA. (38 refs)

78-2839 **Comparative Behavior of Simian Virus 40 T-Antigen and of Tumor-specific Surface and Transplantation Antigens During Partial Purification.** (Eng) Luborsky, S. W. (NCI, NIH, Bethesda, MD, 20014); Chang, C.; Pancake, S. J.; Mora, P. T. *Cancer Res* 38(8): 2367-2371; 1978.

Simian virus 40-specific T antigen was extracted from SV AL/N mouse embryo tissue culture cells by treatment with Triton X-100 detergent. The extracts contained tumor-specific transplantation antigen (TSTA) and tumor-specific surface antigen. After purification by ammonium sulfate precipitation and diethylaminoethylcellulose and phosphocellulose column chromatography, the extracts were assayed for the three antigens. T antigen, TSTA, and much of the tumor-specific surface antigen copurified through all the purification steps. This finding supports previous suggestions of the close homology among the protein species carrying these three antigenic determinants. The antibody-mediated cytolytic assay appeared to detect a new type of cell-surface antigen that is different from T antigen and TSTA. Two antigenic fractions were obtained from the phosphocellulose column that had tumor-specific surface antigen activity, but one of these did not have T-antigen or TSTA activities. (30 refs)

78-2840 **Kinetics of Appearance and Loss of S Antigen in Cells after Abortive or Lytic Infection with SV40.** (Rus) Matveeva, V. A. (Cancer Res. Center, Moscow, USSR); Klyuchareva, T. E.; Deichman, G. I. *Vopr Virusol* (3): 314-317; 1978.

A mixed hemadsorption assay was used to evaluate the kinetics of the appearance and loss of surface (S) antigen in hamster cell and green monkey kidney cell cultures after abortive and lytic infection with simian virus 40. S antigen could be detected in abortively infected hamster cells growing in monolayer; culturing of the hamster cells led to the rapid loss of antigen (after 2-3 passages). S antigen persisted in both stationary and proliferating cultures of green monkey cells. (7 refs)

78-2841 **A Growth Factor for SV40 Virus-Transformed Fibroblasts (SV3T3) from Isolated Rat Hepatocytes (Meeting Abstract).** (Eng) Schuler, M. (M. S. Hershey Medical Center, Pennsylvania State Univ., Hershey, PA, 17033); Feldhoff, R. C.; Jefferson, L. S.; Lipton, A. *Proc Am Assoc Cancer Res* 19: 220; 1978. (no refs)

78-2842 **Variant Lines of Mouse Kidney Transformed by SV40tsA Mutant Which Grow to High Saturation Density at the Nonpermissive Temperature (Meeting Abstract).** (Eng) Otsuka, H. (Baylor Coll. Medicine, Houston, TX, 77030); Dubbs, D. R.; Kit, S. *Proc Am Assoc Cancer Res* 19: 119; 1978. (no refs)

78-2843 **Uptake of Low Molecular Weight Substances by SV40-Transformed 3T3 Cells Is Invariant**

with Growth Rates in the Presence and Absence of Serum. (Eng) Bush, H. (Cancer Res. Campaign Dept. Medicine Oncology, Christie Hosp. and Holt Radium Inst., Manchester, England, M20 9BX); Shodell, M. *Exp Cell Res* 114(1): 27-30; 1978.

The uptake of 2-deoxy-D-glucose (DG) and α-aminoisobutyric acid (AIB) was measured in simian virus 40-transformed 3T3 cells growing in the presence and absence of serum to determine whether changes in proliferative behavior are due to changes in the rate of transport of low-mol-wt substances. Changes in the rates of growth and of DNA synthesis were not accompanied by parallel changes in the uptake of DG and AIB. Thus, nutrient supply is not a major regulatory factor in determining the transformed cell growth rate. (13 refs)

78-2844 **A Study of Cultured Fetal and SV40-transformed Chinese Hamster Kupffer Cells.** (Eng) Clark, J. M. (Dept. Medical Cell Genetics, Medical Nobel Inst., Karolinska Institutet, S-104 01 Stockholm, Sweden); Pateman, J. A. *Exp Cell Res* 114(1): 47-56; 1978.

The characteristics of normal and simian virus 40 (SV40)-transformed adult and fetal Chinese hamster Kupffer cells in culture were compared. The study included 65 SV40-transformed adult Kupffer cell lines (SV40-ACHK), 4 SV40-transformed fetal Kupffer cell lines (SV40-FCHK), and 24 untransformed fetal Kupffer cell lines. Both adult and fetal Kupffer cell lines expressed Kupffer cell functions for a considerable period in culture. Cloned SV40-transformed Kupffer cell lines exhibited properties associated with neoplastic transformation in culture: (1) SV40 tumor antigen; (2) irregular growth pattern; (3) growth in agar suspension culture; (4) growth in medium containing a low serum concentration; and (5) high saturation density. Prior to SV40 transformation, all adult and fetal Kupffer cell lines possessed peroxidase, microsomal heme oxygenase (MHO), and phagocytic activities. After 97 population doublings (PD), normal fetal cell lines had lost phagocytic activity but had retained MHO and peroxidase activities. By 8-9 PD after infection with SV40, none of these activities could be detected in the transformed cell lines. Some of the transformed cell lines remained diploid for at least 80 PD after infection. It is concluded that this cell culture system makes it possible to compare adult, fetal, and SV40-transformed cell lines of defined genetic and epigenetic origin. (31 refs)

78-2845 **Tumor Induction in Newborn and Adult Syrian Hamsters Infected with SV40 Temperature-sensitive Mutants.** (Eng) Deichman, G. (Cancer Res. Center, Acad. Medical Sciences, Moscow, USSR); Kashkina, L.; Rapp, F. *Intervirology* 10(2): 120-124; 1978.

Newborn and adult Syrian hamsters were inoculated with
wild-type simian virus 40 (SV40) and its temperature-sens
tive (*ts*) mutants A30, A209, A239, B201, and BC21
Wild-type SV40 and *ts* B201 were highly oncogenic for nev
born but not adult animals; *ts* A239, *ts* A30, and *ts* BC21
also induced tumors in some adult hamsters. Unexpectedl
the number of tumors induced by *ts* A209 and *ts* A239 i
newborn animals was very low. Tumor frequency was als
low in adults infected with *ts* A30, *ts* A239, and *ts* BC21
The duration of the latent period of tumors induced in adu
animals was the same as that in the newborns. The data o
tained are discussed in connection with the defectiveness
the mutants in inducing tumor-specific transplantation ant
gen activity in hamster cells. (15 refs)

78-2846 **Macrophage-T Cell Cooperation In Vivo in th**
Sensitization Against SV40 Tumors (Meetin
Abstract). (Eng) Blasecki, J. W. (Winter Res. Lab., Mou
Sinai Medical Center, Milwaukee, WI, 53233); Houston, K
J. *Fed Proc* 37(6): 1452; 1978. (no refs)

78-2847 **Cell Adhesion and Cell Surface Topography**
Aggregates of 3T3 and SV40-Viru
Transformed 3T3 Cells. Visualization of Interior Cells
Scanning Electron Microscopy. (Eng) Gershman, H. (Dep
Biochemistry and Biomedical Engineering, Case Western R
serve Univ., Cleveland, OH, 44106); Rosen, J. J. *J Cell Bi*
76(3): 639-651; 1978.

A technique for exposing the interior of aggregates of cu
tured cells was developed and used to examine, by scanni
electron microscopy, cell morphology and cell contact ultra
tructure in the interior of aggregates of BALB/c 3T3 a
simian virus 40 (SV40)-transformed 3T3 cells. The 3T3 ce
made initial intercellular contact by microvillar process
Over a period of 3-8 hr, some of these microvillar conta
were replaced by broader projections. In contrast, the SV4
transformed cells made initial intercellular contact
blebs or blunt projections that were also broadened a
extended over 3-8 hr. In both 3T3 and SV40-3T3 ce
the surfaces of cells forming the outer layer of t
aggregate resembled the surfaces of single cells fixed
suspension, regardless of how long the aggregates h
been cultured. These cells were covered with ma
cellular processes and were roughly hemispherical
profile. The surface of the internal cells of the aggrega
lost many of their cellular processes and develop
smooth patches, and many became irregular in shape. Th
smooth morphology was also observed on the surfaces of t
interior peripheral cell layer. It is concluded that the stabi
zation of adhesive contacts is a slow process that takes at lea
3-8 hr and that the surfaces of the outer layer of the periphe
al cells differ significantly from the those of the interior cell
Clear differences in surface topography exist between no
malignant 3T3 cells and their malignant SV40 transforman
(22 refs)

78-2848 **Cellular Transformation In Vitro by the Transfer of Isolated Metaphase Chromosomes.** (Fre) Cassingena, R. (Institut de Recherches scientifiques sur le cancer [C.N.R.S.], B.P. no. 8, 94800 Villejuif, France); Suarez, H. G.; Lavialle, C. *C R Acad Sci [D] (Paris)* 285(16): 1603-1606; 1977.

Metaphase chromosomes isolated from HeLa cells or human sc tissue cells transformed in vitro by simian virus 40 (SV40) were used to infect (transfect) cultures of line 3T3-4E murine cells and line 809 normal diploid human cells. The recipient cells were trypsinized and seeded, 24 hr after initial culture, in soft-agar medium containing 20% bovine serum or in liquid medium containing 1% serum. The infected mouse cells grew in soft-agar medium, indicating transformation had occurred, but continuous passage of these cells was not achieved. The infected human cells grew in both soft-agar and liquid medium, and up to 10 in vitro cell passages were achieved. The presence of SV40 tumor antigen on the human SV40-transformed cells was considered to be evidence of transformation by the chromosomes from these cells. This antigen was detected by immunofluorescence in all passages of the human cells infected with chromosomes from the SV40-infected cells. (16 refs)

78-2849 **SV40 Virus Interaction with Permissive Host Cells in Culture (Meeting Abstract).** (Eng) Gershey, E. L. (Rockefeller Univ., New York, NY, 10021). *In Vitro* 14(4): 368; 1978. (no refs)

78-2850 **Transcriptional Pattern of Chromatin from SV40-Transformed Cells (Meeting Abstract).** (Eng) Wong, T. K. (Dept. Biochemistry, Univ. North Carolina Cancer Research Center, Chapel Hill, NC, 27514). *Fed Proc* 37(6): 1643; 1978. (no refs)

78-2851 **A Virus-producing Cell Line Developed by Transformation of Human Parapharyngeal Cells with SV40.** (Eng) Rafferty, K. A. (Dept. Anatomy, Sch. Basic Medical Sciences, Univ. Illinois Medical Center, 1853 W. Polk St., Chicago, IL, 60612); Ruben, R. L.; Young, S. K. *In Vitro* 14(2): 227-235; 1978.

Normal cultures of epithelial appearance were initiated by trypsinization of a surgically resected, histologically normal parapharyngeal cyst. Cellular morphology was consistent with derivation from the stratified squamous epithelium of the cyst or from vascular endothelium. Infection with simian virus 40 (SV40) at a dose of 44 plaque-forming units per cell resulted in the appearance of grossly visible transformed foci within 25 days. The transformants were also epithelioid in appearance; they grew vigorously for 22-50 population doublings (23-32 subcultures) and then became quiescent. Virus was detectable at all stages by both direct isolation (cell extracts) and cocultivation with permissive cells. In two sublines in which selection for rapidly growing cell types occurred, virus was detected only by cocultivation. In no case was SV40 detected by electron microscopy, indicating that few cells were producers or that cells produced very few particles. It would appear from these studies that focus isolation tends to select against producer lines, whereas serial subculture at high density is more likely to result in development of producer lines. (27 refs)

78-2852 **Screening of Human Brain Tumors for SV40-related T Antigen.** (Eng) Tabuchi, K. (Univ. Colorado Medical Center, Div. Neurosurgery, Denver, CO 80262); Kirsch, W. M.; Low, M.; Gaskin, D.; Van Buskirk, J.; Maa, S. *Int J Cancer* 21(1): 12-17; 1978.

Thirty-nine human brain tumors were screened for the presence or absence of simian virus 40 (SV40) tumor (T) antigen by direct and indirect immunoperoxidase techniques. The tumors consisted of 12 glioblastomas, 5 astrocytomas, 1 ependymoma, 1 oligodendroglioma, 3 medulloblastomas, 1 neurinoma, 1 plexus papilloma, 10 meningiomas, 1 hemangioendothelioma, 1 angiofibroma, and 3 metastatic cancers. Only the ependymoma and the plexus papilloma had a positive immunoperoxidase reaction for the antigen both in vivo and in vitro. No staining was detected in the nuclei of tumor cells treated with either anti-SV40 V (anti-SV40 capsid antigen) or anti-human IgG conjugates; no positive staining was present after the tumors were heated to 55 C for 30 min. Nuclear extracts from the ependymoma also demonstrated nuclear antigen. Subcultures of these tumors at the sixth transfer were checked by an independent laboratory and found to be negative for T antigen by immunofluorescent screening. It is suggested that long-term subculture resulted in selective overgrowth of non-T-expressing cells, or the immunofluorescent test was not as sensitive as the immunoperoxidase test. (30 refs.)

78-2853 **The Sensitivity of SV40 Transformants to Amino Acids: Characteristics of Methionine Resistant Mutants of SV40 3T3 (SVT2) (Meeting Abstract).** (Eng) Heiser, W. (Dept. Biological Sciences, Univ. California, Santa Barbara, CA, 93106); Englesberg, E. *In Vitro* 14(4): 388; 1978. (no refs)

78-2854 **An Accumulation of Lanosterol and Dihydrolanosterol in SV40 Transformed Swiss 3T3 Cells (Meeting Abstract).** (Eng) Krueger, K. E. (Vanderbilt

Univ., Nashville, TN, 37232); Hellerqvist, C. G. *Fed Proc* 37(6): 1662; 1978. (no refs)

78-2855 **Characterization of Herpesvirus Saimiri-induced DNA-directed DNA Polymerase (Meeting Abstract).** (Eng) Ebert, P. S. (NIH, Bethesda, MD, 20014); Vande Woude, G. *Proc Am Assoc Cancer Res* 19: 231; 1978. (no refs)

78-2856 **Tumour Induction with DNA of Oncogenic Primate Herpesviruses.** (Eng) Fleckenstein, B. (New England Regional Primate Res. Center, Harvard Medical Sch., 1 Pine Hill Drive, Southborough, MA, 01572); Daniel, M. D.; Hunt, R. D.; Werner, J.; Falk, L. A.; Mulder, C. *Nature* 274(5666): 57-59; 1978.

The tumorigenicity of the DNA of *Herpesvirus saimiri* and *H. ateles* was determined in cotton-top marmoset monkeys. *H. saimiri* DNA (0.05-10 μg) was slowly injected iv, ip, or im into 14 marmosets. Three animals died 60-85 days later. Microscopic examination confirmed lymphoblastic-type malignant lymphoma with extensive invasion and replacement of the lymph nodes, spleen, bone marrow, thymus, liver, and kidney. All reisolation trials, except one, were successful. Four marmosets were infected similarly with 20 μg *H. ateles* DNA. One monkey died after 97 days with clinical and gross features of malignant lymphoma. The lesion resembled that of *H. ateles* in this species except that there was extensive necrosis of the lymphocytic follicles in the spleen and lymph nodes. Attempts to isolate virus or to demonstrate an immune response to structural viral antigens were unsuccessful. However, DNA-DNA hybridizations showed that at least 0.013% of liver DNA and 0.015% of spleen DNA, extracted from autopsy material, hybridized with radioactive L DNA of *H. ateles* (equal to at least 7 viral DNA copies/diploid liver cell, 8 copies/spleen cell). [L DNA is a region of unique DNA (36%-38% guanine + cytosine, mol wt 71,800,000) inserted between 2 termini of repetitive DNA with variable lengths.] The striking difference in incubation period between these lymphomas and those caused by infectious virus particles (60-97 vs 30 days) shows that transformed cells or biologically active DNA can persist in the organism over prolonged periods. The ability of herpesvirus DNA to induce tumors indicates that DNA molecules of large size can be incorporated into the cells of living organisms in higher mammalian species. (16 refs)

78-2857 **Experimental Infection of *Callithrix jacchus* Marmosets with *Herpesvirus ateles*, *Herpesvirus saimiri*, and Epstein Barr Virus.** (Eng) Ablashi, D. V. (NCI, Room A-906, Landow Building, 7910 Woodmont Ave., Bethesda, MD, 20014); Pearson, G.; Rabin, H.; Armstrong, G.; Easton, J.; Valerio, M.; Cicmanec, J. *Biomedicine [Expres* 29(1): 7-10; 1978.

The effect of an ip injection of *Herpesvirus ateles* (HVA strain 73, *Herpesvirus saimiri* (HVS), or Epstein-Barr viru (EBV) on common marmosets (*Callithrix jacchus*) was dete mined. HVA induced tumors containing several types cells, including lymphocytes, plasmacytoid lymphocytes, hi tiocytes, polymorphonuclear WBC, and giant cells characte istic of the Reed-Sternberg cells in Hodgkin's disease. HV and EBV did not induce tumors, but HVS was present in th lymphocytes and it elicited a strong antibody response. EB elicited only a variable antibody response. The common ma moset probably requires cofactors for the development of tu mors following EBV and HVS infection and is thus a mod complementary to the cotton-topped marmoset, which do not require these cofactors. (20 refs)

78-2858 **Measurements of the Molecular Size of th Simian Virus 40 Large T Antigen.** (Eng) Griffi J. D. (Div. Medical Oncology, Sidney Farber Cancer Ins Boston, MA, 02115); Light, S.; Livingston, D. M. *J Vir* 27(1): 218-226; 1978.

The mol wt of the large simian virus 40 (SV40) tumor (antigen was estimated by sodium dodecyl sulfa (SDS)-polyacrylamide gel electrophoresis, random-co chromatography in guanidine hydrochloride, and sediment tion-velocity analysis in a density gradient. In view of th strong theoretical basis for the guanidine method and th agreement with the sedimentation data, these findings su gest that the mol wt of the SV40 protein is 75,000-80,00 daltons (75K-80K) rather than 94K-100K. Therefore, co siderably less than the entire early region of SV40 is require to encode it. This size estimate is in accord with earlier r sults, which revealed a normal-size T antigen in cells infecte with viable deletion mutants lacking as much as 10% of th early region. Large T antigen isolated from an SV40-tran formed human fibroblast cell line and from a lytically infec ed African green monkey kidney cell line migrated in parall as a single 94K polypeptide in SDS-polyacrylamide gel However, both proteins had an approx mol wt of 75K by th other two methods. (57 refs)

78-2859 **An In Vitro Transformation Assay for SiSV and HL23V Using Feline Embryonic Fib blasts (40044).** (Eng) Markham, P. D. (Dept. C Biology, Litton Bionetics, Inc. Bethesda, MD, 2001 Dodge, W.; Salahuddin, S. Z.; Gallagher, R. E. *Pr Soc Exp Biol Med* 157(2): 312-318; 1978.

The focus-forming ability of simian sarcoma virus (SiSV and HL23V, a virus isolated from the cultured periphe blood and bone marrow WBC of a patient with acute m logenous leukemia, was determined in feline embryo fibroblasts (FEF), normal rat kidney cells, rat embryo fibroblasts, and several cultures of primate origin. The findings for these viruses were compared to those for baboon endogenous virus (BaEV) and gibbon ape leukemia virus (GaLV). SiSV-1 and HL23V were able to induce foci in all cell lines tested. GaLV and BaEV showed no indication of transformation. SiSV-1, HL23V, and GaLV were able to replicate in all cell lines; BaEV did not replicate in any of the lines. With FEF cells, foci were completely developed and ready for counting by the third or fourth day after infection, but the other lines required 7-10 days for max focus development. Transformation was 2-100 times more efficient with FEF cells than with cells normally used for SiSV-1 transformation. (25 refs)

78-2860 **Antigenic Relationship of Human Foamy Virus to the Simian Foamy Viruses.** (Eng) Nemo, G. J. (Lab. Central Nervous System Studies, Intramural Res. Program, Natl. Inst. Neurological and Communicative Disorders and Stroke, Bethesda, MD, 20014); Brown, P. W.; Gibbs, C. J.; Gajdusek, D. C. *Infect Immun* 20(1): 69-72; 1978.

A foamy virus previously isolated from a human nasopharyngeal carcinoma was studied by reciprocal cross-neutralization and cross-immunofluorescent techniques to determine its antigenic relationship to the eight known simian foamy viruses (SFV). Cross-immunofluorescent screening indicated a two-way cross-reactivity between human foamy virus and SFV-6, SFV-7, and, to a lesser degree, SFV-1. In the cross-neutralization studies, a reciprocal cross-relationship was observed between human foamy virus and SFV-6, but not between the human virus and SFV-7 or SFV-1. In a seroepidemiological survey, neutralizing antibody to the human foamy virus was not detected in the sera of 14 animal caretakers or 24 laboratory personnel who routinely handled a wide variety of nonhuman primates. It is concluded that the human isolate most probably represents a variant strain of SFV-6. (6 refs)

78-2861 **The Phosphodiesterase I-Alkaline Phosphatase System in Herpes Simplex Virus (Type 1) Transformed Hamster Cells. Biochemical and Biological Properties (Meeting Abstract).** (Ger) Schneider, D. (Abteilung fur Exptl. Virologie, Inst. Medizinische Mikrobiologie, Univ. Mainz, Hochhaus am Augustusplatz, D-6500 Mainz, W. Germany). *Hoppe Seylers Z Physiol Chem* 359(3): 317-318; 1978. (6 refs)

78-2862 **Infectious Mononucleosis, Herpes Simplex Infection, and Diffuse Lymphoma in a Renal Transplant Recipient.** (Eng) Briggs, J. D. (Renal Unit, Univ. Dept. Surgery, Western Infirmary, Glasgow, G11 6NT, Scotland); Hamilton, D. N.; MacSween, R. N.; Pennington, T. H. *Transplantation* 25(4): 227-228; 1978.

A 22-yr-old girl developed diffuse lymphoblastic lymphoma 4 yr after receiving a kidney transplant. At the time of presentation, herpes simplex lesions were present around the patient's mouth and vulva; she also had infectious mononucleosis and an Epstein-Barr virus infection. It is not known if the lymphoma preceded or followed the viral infections. (5 refs)

78-2863 **High Incidence of Virus Isolation from Donor and Recipient Tissues Associated with Renal Transplantation.** (Eng) Orsi, E. V. (Biology Dept., Seton Hall Univ., South Orange, NJ, 07079); Howard, J. L.; Baturay, N.; Ende, N.; Ribot, S.; Eslami, H. *Nature* 272(5651): 372-373; 1978.

Tissues from donor kidneys (10), livers (2), or spleens (2) and from 15 kidney recipients were examined by immunofluorescent staining for herpes simplex virus types 1 and 2 (HSV1 and HSV2) and cytomegalo-virus (CMV). HSV-1 was isolated from 1/14 donors and 3/15 recipients, HSV-2 from 5 donors and 7 recipients, human CMV from 11 donors and 12 recipients. Isolation of all three viruses from the same individual occurred in one donor and one recipient. The most frequent combination was HSV-2 and CMV. The frequency of CMV isolation agreed with the incidence of infection found in serological studies, but the rates of HSV-1 and HSV-2 isolation were unexpectedly high. The isolation of herpesvirus from tissue donors without apparent herpetic lesions may complicate attempts to relate these viruses to neoplasia and other diseases. (15 refs)

78-2864 **Relationship Between HSV-2-Antigen and Colpocytological Findings. (Meeting Abstract).** (Ger) Gorcs, J. (Sorhaz U. 4, H-7621 Pecs, Hungary). *Fortschr Med* 96(7): 350; 1978. (no refs)

78-2865 **Properties of Epstein-Barr Virus Transformed Woolly Monkey Lymphoblastoid Cell Lines.** (Eng) Andiman, W. A. (Dept. Pediatrics, Yale Univ. Sch. Medicine, 333 Cedar St., New Haven, CT, 06510); Miller, G. *Proc Soc Exp Biol Med* 157(3): 489-493; 1978.

The establishment of a lymphoblastoid cell line following in vitro transformation of woolly monkey WBC by the B95-8 strain of Epstein-Barr virus (EBV) is reported. Woolly monkey peripheral blood lymphocytes were relatively resistant to in vitro transformation by EBV: only 14/145 virus-exposed WBC cultures resulted in cell lines. No lines were derived without in vitro exposure to virus. Transformation frequency could not be increased by exposing the WBC to mitogens, depleting the number of T cells, or exposing the cells to virus after several days in culture. Almost half the lymphocytes in fresh blood had a receptor for IgFc, and 60%-90% of continuously cultured cells expressed receptors for both rabbit and human IgFc. On the lymphoblastoid lines, however, the IgFc receptor was qualitatively different from that induced by herpes simplex virus, which could be detected with rabbit but not human Ig. Monkeys inoculated with cell-free virus and autologous transformed cells showed no evidence of infection. Thus, although woolly monkey cells can be transformed in vitro by EBV, they are not oncogenic when replaced in the autologous host. (20 refs)

78-2866 **Heterogeneity of Epstein-Barr Virus. III. Comparison of a Transforming and a Nontransforming Virus by Partial Denaturation Mapping of Their DNAs.** (Eng) Delius, H. (European Molecular Biology Lab., 6900 Heidelberg, W. Germany); Bornkamm, G. W. *J Virol* 27(1): 81-89; 1978.

The DNA's of transforming (B95-8) and nontransforming (P3HR-1) Epstein-Barr virus (EBV) strains were compared by partial denaturation mapping. The former virus was derived from EBV-transformed marmoset lymphocytes, the latter from a Burkitt's lymphoma. B95-8 viral DNA showed a homogeneous denaturation pattern. In contrast, P3HR-1 viral DNA was heterogeneous: it contained at least two classes of molecules, Groups A and B, which were present in a ratio of about 2:1 to 3:1. No evidence was obtained that molecules from Groups A and B contained identical sequences present in different orientations, as described for herpes simplex viral DNA. Most of the sequences present in B95-8 and in P3HR-1 viral DNA Group A could be correlated by assuming that different sequences, about 12,000 base pairs long, were inserted or deleted, respectively, at different positions of both viral genomes. (23 refs)

78-2867 **Inhibition of DNA Metabolism in Human B Lymphocytes by a Substrain of Epstein-Barr Virus (P3HR-1): A Method for Virus Quantitation.** (Eng) Steinitz, M. (Dept. Tumor Biology, Karolinska Institutet, S-104 01 Stockholm, 60, Sweden); Bakacs, T.; Yefenof, E.; Klein, G. *Intervirology* 10(2): 125-131; 1978.

Pretreatment with the P3HR-1 substrain of Epstein-Barr virus (EBV) inhibited by approx 85% the DNA stimulation induced by *Staphylococcus aureus* in human B lymphocytes. In parallel experiments, the DNA stimulation induced by the transforming B95-8 substrain of EBV was almost completely inhibited by prior exposure to P3HR-1 virus. Phytohemagglutinin stimulation was inhibited only slightly but the DNA synthesis of continuously growing Raji cells was inhibited to a considerable extent. The degree of DNA inhibition was correlated with the induction of the EBV membrane, early, an
nuclear antigens in sensitive indicator cells. (14 refs)

78-2868 **A New Epstein Barr Virus (EBV) Associate
Cell Surface Antigen (Meeting Abstract).** (Eng
Rosenthal, K. S. (Sidney Farber Cancer Inst., Harvard Medi
cal Sch., Boston, MA, 02115); Shuman, H. *Fed Proc* 37(6
1561; 1978. (no refs)

78-2869 **Relationship Between Humoral Antibodies
Epstein-Barr Virus and Tumors of the Lyn
phoid System in Man.** (Rus) Mazurenko, N. P. (Cancer Re
Center, Moscow, USSR); Stepina, V. N.; Gurtsevich, V.
Yarymova, N. M.; Kaverzneva, M. M.; Lorie, Yu. I.; Kr
evsky, N. A.; Durnov, L. A.; Ermakov, E. S.; Probatova,
A.; Pokrovskaya, N. N. *Vestn Akad Med Nauk SSSR* (
60-71; 1977.

The level of antibodies against Epstein-Barr virus (EBV) w
assessed in sera of 28 clinically healthy children
14 yr), 69 children with various benign and maligna
tumors (neuroblastoma, Wilms' tumor, reticulosarcom
teratoblastoma, sarcoma of the soft tissues), 94 ad
patients with Hodgkin's disease and 55 adult patien
with various tumors of the lymphoid system. The
was no difference in the titer of humoral antibodi
against EBV between the healthy children (1:6
and children with benign (1:62) or malignant tumors (1:65
The patients with Hodgkin's disease had significantly i
creased antibody titers (1:150 in the patients wi
nodular-sclerous variant, 1:242 in mixed cellular varia
and 1:356 in the patients with lymphoid exhaustio
Simultaneously, the patients with all variants
Hodgkin's disease showed significant (almost two-
threefold) decrease of the indices of the T-cell i
munity. In spite of the marked tropism of EBV
the lymphoid tissues, the patients with tumors of t
lymphoid system showed no increase in the titer
antibodies against EBV. (50 refs.)

78-2870 **Lymphocytes Transformed by Epstein-Ba
Virus: Induction of Nuclear Antigen Reacti
with Antibody in Rheumatoid Arthritis.** (Eng) Alspaugh,
A. (Dept. Medicine, Univ. Louisiana Sch. Medicine, Ne
Orleans, LA); Jensen, F. C.; Rabin, H.; Tan, E. M. *J Exp M*
147(4): 1018-1027; 1978.

The indirect immunofluorescence test was used to detect t
presence, in several lymphocyte lines, of a nuclear antig
that would react with antibodies in the sera of patients wi
rheumatoid arthritis (RA). The lines tested included norm
and Epstein-Barr virus (EBV)-transformed human periphe

blood lymphocytes and established human (umbilical cord) and simian (cotton-topped marmoset, orangutan, baboon, and owl monkey) lymphocyte lines infected with EBV and related herpesviruses. The nuclear antigen reactive with antibody in the sera of RA patients could be induced in EBV-infected human lymphocytes. The EBV-infected cells did not react with normal human sera, and control uninfected cells did not react with RA sera. Simian cell lines that had been transformed by EBV contained the nuclear antigen reactive with RA sera, whereas human cord cells infected with herpesvirus papio and simian cell lines carrying herpeslike viruses were negative for this antigen. Although most cells containing RA-associated nuclear antigen also contained Epstein-Barr nuclear antigen (EBNA), there was a lack of complete concordance. The RA-associated nuclear antigen was differentiated from EBNA by immunofluorescence and immunodiffusion tests and by nuclear staining. The data do not indicate whether an etiological relationship exists between EBV infection and RA. (21 refs)

78-2871 **The Capacity of Lymphoblastoid Cells to Activate the Alternative Complement Pathway Is Correlated with Their Transformation by Epstein-Barr Virus (Meeting Abstract).** (Eng) McConnell, I. (MRC Group on Mechanisms on Tumour Immunity, Cambridge, England); Lachmann, P. J.; Lint, T.; Klein, G. *J Immunol* 120(5): 1787; 1978. (no refs)

78-2872 **Epstein-Barr Virus Related Antigens in Cultured Human Cancer Cells.** (Jpn) Inoue, J. (Dept. Surgery, Tokyo Medical Coll., Shinjuku-ku, Tokyo 160, Japan). *Tokyo Ika Daigaku Zasshi* 35(5): 771-782; 1977.

A total of 31 cultured cell lines from various types of human cancers (17 epithelial, 6 nonepithelial, 4 of neurogenic tumor origin, and 4 lymphoblastoid) were studied for the presence of the viral capsid antigen (VCA) and early antigen (EA) of Epstein-Barr virus (EBV). VCA-positive cells were detected only in the P$_3$HR-1, MK-Ly, and PC-5-Ly lymphoblastoid cell lines. EA-positive cells, however, were detected in the Raji and P$_3$HR-1 lymphoblastoid lines and in the OAT, PC-1, and PC-5 lines of pulmonary carcinoma origin. These findings suggest a possible role for EBV in the development of pulmonary carcinoma. (50 refs)

78-2873 **Partial Purification and Properties of the Epstein-Barr Virus-associated Nuclear Antigen.** (Eng) Baron, D. (Medizinische Universitaetsklinik, Otfried-Mueller-Strasse, D-74 Tubingen, W. Germany); Strominger, J. L. *J Biol Chem* 253(8): 2875-2881; 1978.

Epstein-Barr virus (EBV)-associated nuclear antigen (EBNA) was purified 85-fold from a nuclear pellet derived from an EBV-transformed B-lymphoblastoid cell line, and some of its properties were determined. The purification procedure consisted of five steps: preparation of extract, heating at 80 C in phosphate buffer, ammonium sulfate precipitation, preparative ultracentrifugation, and affinity chromatography on double-stranded DNA-cellulose. The most highly purified material contained a major polypeptide of 49,000 daltons together with several other polypeptides present in much smaller amounts; it is uncertain which of these is EBNA. The purified complement fixing antigen specifically blocked the anticomplement immunofluorescence assay for EBNA. Several properties indicate a close association of EBNA with chromatin: (1) precipitation of antigenic activity by phosphate buffer and subsequent thermal fractionation; (2) partial sensitivity of antigenic activity to DNase but not to RNase and restoration of activity by addition of calf thymus DNA; and (3) specific binding of EBNA to double-stranded DNA-cellulose. EBNA was unusually heat stable; no loss of antigenic activity was observed even after heating for 30 min at 80 C. This heat stability suggests that EBNA is either a single polypeptide chain or a homooligomer, because it is unlikely that all the different proteins of a heterooligomer would show this exceptional heat stability. (33 refs)

78-2874 **Persisting Oncogenic Herpesvirus Induced by the Tumour Promoter TPA.** (Eng) zur Hausen, H. (Inst. Virologie, Zum Hygiene, Univ. Freiburg, Freiburg, W. Germany); O'Neill, F. J.; Freese, U. K.; Hecker, E. *Nature* 272(5651): 373-375; 1978.

The effect of the cocarcinogen and tumor promotor 12-O-tetradecanoylphorbol-13-acetate (TPA: 20 nanograms/ml) on Epstein-Barr virus (EBV) production was studied in two producer cell lines [P3HR-I, of Burkitt's lymphoma (BL) origin, and B95-8, an EBV-transformed marmoset line of infectious mononucleosis origin] and two nonproducer cell lines (Raji BL cells and NC37 cells from a normal donor). TPA increased the number of antigen-positive cells in all four lines. Viral capsid antigen was increased in P3HR-I and B95-8, but only early antigen (EA) was induced in NC37 and Raji cells. Effective virus particle production in the producer line was demonstrated by electron microscopy. EA or VCA synthesis was also increased in 10 other EBV-transformed lymphoblastoid lines tested. Two baboon lines transformed by herpesvirus papio showed only a moderate antigen induction, but a T-cell line transformed by herpesvirus saimiri was efficiently induced after TPA treatment. The potential usefulness of P3HR-I and B95-8 cells for biological and immunologic studies of EBV is discussed. (14 refs)

78-2875 **DNA of Epstein-Barr Virus. III. Identification of Restriction Enzyme Fragments that Contain**

DNA Sequences which Differ among Strains of Epstein-Barr Virus. (Eng) Raab-Traub, N. (Section Infectious Diseases, Dept. Medicine, Univ. Chicago, Chicago, IL, 60637); Pritchett, R.; Kieff, E. *J Virol* 27(2): 388-398; 1978.

The HR-1, B95-8, and W91 strains of Epstein-Barr virus (EBV) were studied to identify the restriction enzyme fragments of the DNA of each of these strains that contain sequences not present in the other strains. The DNA's of the three strains differed in complexity. Sequences missing in the B95-8 strain were found in the *Eco*RI-C and -D and *Hsu* I-E and -N fragments of the HR-1 strain and in the *Eco*RI-C and *Hsu* I-D and -E fragments of the W91 strain. The HR-1 strain was missing DNA contained in *Eco*RI fragments A and J to K and *Hsu* I fragment B of the B95-8 strain and in the *Eco* RI-A and *Hsu* I-B fragments of the W91 strain. Available data suggest that the W91 strain contains all of the sequences present in both the B95-8 and HR-1 strains. These data studied in relationship to a linkage map of the restriction enzyme fragments of the DNA of B95-8 and W91 show that the additional DNA in these strains is in the region of overlap between *Eco*RI fragment A and *Hsu* fragment B of the E3V B95-8 and W91 DNA's. Indirect lines of evidence support the hypothesis that the DNA deleted in the HR-1 strain accounts for the inability of this strain to transform lymphocytes to lymphoblasts with indefinite growth in vitro. (29 refs.)

78-2876 **Epstein-Barr Virus Infection in Staff Treating Patients with Nasopharyngeal Carcinoma (Letter to Editor).** (Eng) Ho, H. C. (Medical and Health Dept., Inst. Radiology and Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong); Kwan, H. C.; Poon, Y. F.; Tse, K. C.; Ng, M. H. *Lancet* 1(8066): 710-711; 1978.

The IgA and IgG reactivities to the viral capsid antigen (VCA) of Epstein-Barr virus (EBV) were determined in hospital staff members treating patients with nasopharyngeal carcinoma (NPC). No definite IgA anti-VCA reaction was observed, but IgG anti-VCA titers were substantially increased in some staff members, suggesting greater EBV exposure. The increases were highest for nurses and clinicians and lowest for medical laboratory and clerical personnel. These findings suggest that NPC patients may constitute a source of EBV infection. (15 refs)

78-2877 **Do Viruses Have a Role in Causing Human Cancer?** (Eng) Temin, H. M. (Viral Oncology and Cell Biology at McArdle Lab., Medical Sch. Center for Health Sciences Univ. of Wisconsin, Madison, WI). *Trends Biochem Sci* 3(4): 80-83; 1978.

Although viruses do not have a direct role in the etiology of most human cancers, they may play necessary roles in the etiology of a few cancers. Various roles for viruses include (1) acting as transforming agents, (2) effecting genetic changes that result in cancer, (3) acting as partial or cocarcinogens, or (4) activating preexisting cancer genes or causing genetic variation leading to the formation of cancer genes. It is also possible that genetic variation in cellular processes related to those used primarily in viral replication could cause tumors. The most likely human cancers to have virus involvement are nasopharyngeal carcinoma and Burkitt's lymphoma. In both cases, the Epstein-Barr virus has been found in nearly all the cancers; however, the virus may be present only because the tumor cells provide a good host for virus persistence. Hepatitis or infection by hepatitis B virus appears to be a precursor of liver cancer and products related to or analogous to gene products of retroviruses have been reported in some human leukemias and mammary tumors. It is believed that genetic variation in cellular processes related to those used in virus replication could make cells susceptible to the action of non-viral carcinogens such as cigarette smoke and sunlight. (26 refs)

78-2878 **Hepatitis B Virus and Hepatocellular Carcinoma (Meeting Abstract).** (Eng) Omata, M. (USC-Liver Unit, John Wesley Hosp., Los Angeles, CA); Ashcavai, M.; Liew, C. T.; Peters, R. L. *Gastroenterology* 74(5, part 2): 1169; 1978. (no refs)

78-2879 **Comparative Optical Properties of Free and Assembled Hexon Capsomeres of Human Adenovirus Type 2.** (Eng) Boulanger, P. A. (Unite de Virologie No. 102, INSERM, Place de Verdun, Lille, France); Devaux, C.; Loucheux-Lefebvre, M. H. *Febs Lett* 85(1): 52-56; 1978.

The optical properties of free hexon capsomeres and of interlocking hexons (ninemers) of human adenovirus type 2 were studied to determine the effects of changes in the conformational structure of the protein chain upon capsomere assembly. The circular dichroism spectra at 180-250 nanometers (nm) were identical for both kinds of hexons. Between 250-320 nm, however, differences were noted. A shoulder observed at 302 nm for the ninemers was not found in the hexon monomer spectra. At 290 nm and 283 nm, the dichroic bands were more intense for the hexon ninemers than for the monomers. Furthermore, a dichroic band was visible at 275 nm only in the ninemer spectra. The amino acid composition of the ninemers and free hexon capsomeres was identical, and the bands could be correlated to specific residues involved in hexon assembly. The shoulder at approx 300 nm suggested that tryptophan residues were in a disturbed electronic environment within the ninemers. The band at 290 nm was induced by tryptophan-indole rings. The band at 283 nm was also probably due to tryptophan residues. (27 refs)

78-2880 **Characterization and Sequence Analysis of a Recombination Site in the Hybrid Virus $Ad2+ND_1$.** (Eng) Zain, B. S. (Cold Spring Harbor Lab., Cold Spring Harbor, NY, 11524); Roberts, R. J. *J Mol Biol* 120(1): 13-31; 1978.

Restriction endonuclease mapping of an adenovirus-simian virus 40 (SV40) hybrid virus and adenovirus 2 (Ad2) DNA allowed the characterization of fragments of the nondefective hybrid $Ad2+ND_1$, which contained the two junctions between SV40 sequences and Ad2 sequences. Nucleotide sequence analysis in $Ad2+ND_1$ and the two parent strains allowed identification of the nucleotides involved in recombination. No sequence homology was detected between the parent DNA's. (45 refs)

78-2881 **Groups of Adenovirus Type 2 mRNA's Derived from a Large Primary Transcript: Probable Nuclear Origin and Possible Common 3′ Ends.** (Eng) Nevins, J. R. (Rockefeller Univ., New York, NY, 10021); Darnell, J. E. *J Virol* 25(3): 811-823; 1978.

The number and relative amounts of various messenger RNA (mRNA) molecules derived from a large primary transcript during adenovirus type 2 infection were mapped and studied to determine whether the formation of all the relevant mRNA's is a nuclear event. Hybridization of labeled late mRNA to a series of DNA restriction fragments representing the right-hand 70% of the genome indicated at least 12 discrete mRNA's that appeared to fall into five groups, each possibly containing a common 3′ terminus. The processing necessary to generate these mRNA's apparently occurred in the nucleus. All the mRNA's appeared to contain a sequence of approx 100 nucleotides complementary to a fragment with viral DNA coordinates 25.5-27.9. Furthermore, this fragment contained one of the regions previously found to be spliced onto the 5′ end of late adenovirus type 2 mRNA's. Because the sequences to be spliced existed only once per large transcript, any of the mRNA-specific regions might only have been preserved from a small fraction of the transcripts. Measurement of the transport efficiency of regions of the large nuclear transcript showed that only 15%-25% of any particular region was transported to the cytoplasm. Thus, the large late nuclear transcript can be processed in the nucleus to yield any one of approx 12 mRNA's; the unused portions of the primary transcript then accumulate in the nucleus or are destroyed. (46 refs)

78-2882 **Transcription of Adenovirus RNA in Permissive and Nonpermissive Infections.** (Eng) Farber, M. S. (Dept. Medicine and Cell Biology, Albert Einstein Coll. Medicine, Bronx, NY, 10461); Baum, S. G. *J Virol* 27(1): 136-148; 1978.

The mechanism of blocked replication of human adenoviruses in monkey cells and the way in which simian virus 40 (SV40) coinfection overcomes this block were examined. Previous experiments had placed the block at the level of transcription or translation of adenovirus messenger RNA (mRNA). Thus, the transcription process was investigated in CV-1 monkey cells infected with adenovirus type 2 (Ad2) at 3 plaque-forming units (PFU)/cell (unenhanced infection), in CV-1 cells infected with Ad2 at 10 PFU/cell SV40 (enhanced infection), and in permissive human HeLa cells. Ad2 mRNA from the enhanced monkey cells appeared to be identical to Ad2 mRNA from the HeLa cells. This indicated that SV40 coinfection did not overcome the blocked replication by substituting for a missing Ad2 transcript. Comparison of viral mRNA from enhanced and unenhanced monkey cell infection revealed two types of transcriptional discrepancies. There was a decrease in both the complexity and the relative abundance of several regions of the enhanced adenovirus mRNA. However, neither of these transcriptional defects was sufficient to explain completely the difference in yields of infectious virus and viral protein seen in the two types of infection. (38 refs)

78-2883 **Nuclear Translational Units of Adenovirus-infected HeLa Cells (Meeting Abstract).** (Eng) Chatterjee, N. K. (Div. Lab. and Res., New York State Dept. Health, Albany, NY); Dickerman, H. W.; Beach, T. A. *Miami Winter Symp* 14: 267; 1977. (no refs)

78-2884 **Spliced Viral Messenger RNAs (Meeting Abstract).** (Eng) Sharp, P. A. (Center for Cancer Res., Massachusetts Inst. Technology, Cambridge, MA, 02139); Berk, A. J.; Berget, S. M. *Fed Proc* 37(6): 1271; 1978. (no refs)

78-2885 **Isolation and Characterization of a Slow-migrating Class of Adenovirus Type 2 Hexons.** (Eng) Boulanger, P. (Unite de Recherches, no. 102 de l'INSERM, 2, Place de Verdun, 59045 Lille Cedex, France); Devaux, C.; Lemay, P. *Virology* 84(2): 456-468; 1978.

The biochemical and immunological properties of two classes of hexons, normal hexons and slow hexons, isolated from KB cells infected with type 2 human adenovirus, were compared. The slow hexons were similar in shape and morphology to normal hexons but differed from them in electric charge. They had a lower electrophoretic mobility, a higher isoelectric point, and a greater type-specific antigenicity than the normal hexons. Polypeptide analysis of the slow hexons revealed that they consisted of a trimeric arrangement of polypeptide subunits of mol wt 115,000; ie, they contained 50 less amino acid residues than the normal hexons (mol wt 120,000). The N-terminal end of their polypeptide subunit was not blocked by acetylation, as in normal hexons, suggesting that proteolytic cleavage occurred at the N terminus. Since such proteolytically processed hexons could not be detected in the adenovirion, it seems likely that these slow hexons represent aberrant hexons occurring variably in some massively infected cell cultures and that they are not involved in adenovirus morphogenesis. (22 refs)

78-2886 **Adenovirus Core Protein Synthesis in the Absence of Viral DNA Synthesis Late in Infection.** (Eng) Kit, G. (Dept. Molecular Biology, Univ. California, Berkeley, CA, 94720); Daniell, E. *J Virol* 27(1): 74-80; 1978.

An attempt was made to determine if adenovirus (Ad) DNA synthesis and core protein synthesis exhibit a tight coupling similar to that observed between cell and/or papovavirus DNA synthesis and histone synthesis. Acid extraction of the Ad type 5 core proteins V, VII, and pVII (precursor to VII) from infected HeLa cells and subsequent electrophoresis of the proteins on a 15% acrylamide-2.5 M urea-0.9 N acetic acid (pH 2.7) gel revealed that the electrophoretic mobilities of peptide VII and histone H1, both of which have a mol wt of about 18,000, are similar. Peptide V had a much slower mobility. The core proteins were coded for by late adenovirus messenger RNA. When DNA synthesis was inhibited before its initiation by the addition of cytosine arabinoside (2, 20, or 200 μg/ml) to the medium or by growing Ad H5 ts125 (a temperature-sensitive mutant for DNA replication) at the nonpermissive temperature (40 C), core proteins were not synthesized. However, when DNA synthesis was inhibited after it had been initiated, core proteins continued to be synthesized. Because core protein synthesis continued > 8 hr after the inhibition of viral DNA synthesis, it appears that the uncoupling of viral DNA synthesis and core protein synthesis was permanent. This result is in contrast to the tight coupling of cell and/or papovavirus DNA synthesis and histone synthesis. In these cases, histone gene expression depends on continual DNA synthesis. (30 refs)

78-2887 **Analysis of the Genome of Type 7 Simian Adenovirus Using Restrictases.** (Eng) Naroditsky, B. S. (D.I. Ivanovsky Inst. Virology, USSR Acad. Medical Sciences, Gamaleya Str., 16, Moscow 123098, USSR); Zavizion, B. A.; Karamov, E. V.; Tikhonenko, T. I. *Nucleic Acids Res* 5(3): 999-1011; 1978.

The physical map of simian adenovirus-7 (SA-7) DNA was compiled using the following restricting endonucleases: R.EcoRI, R.BamI, and R.SalI. Since Sa-7 DNA has only one recognition site for R.EcoRI, cleavage with this enzyme gave rise to two fragments with mol wts of 12.0 and 10.0 x 10^6. Complete cleavage of SA-7 DNA with R. BamI gave rise to seven fragments with mol wts of 6.6, 5.9, 3.8, 2.7, 1.3, 0.7, and 0.6 x 10^6. Simultaneous cleavage of the genome by R.BamI and R.EcoRI showed that the recognition site for R.EcoRI is in R.BamI fragment C. The order of the R.BamI fragments in SA-7 DNA is E-A-D-F-C-G-B. Complete cleavage of SA-7 DNA by R.SalI yielded six fragments with mol wts of 8.1, 5.5, 4.3, 2.45, 1.2, and 0.6 x 10^6. The order of the R.SalI fragments is A-B-D-F-E-C, and the recognition site for R.EcoRI is localized in R.SalI fragment B. It is likely that the middle part of SA-7 DNA, where the recognition sites for R.BamI and R.SalI are localized, is rich in guanosine-cytidine pairs. According to previous studies, it is this part of the genome that is responsible for the oncogenicity of the virus. (15 refs)

78-2888 **Detection of Adenovirus 12-induced DNA-binding Protein in Infected and Transformed Cells with Monkey Antisera.** (Eng) Komatsu, T. (Dept. Enteroviruses, Natl. Inst. Health, Tokyo, Japan); Shiroki, K.; Shimojo, H. *Microbiol Immunol* 21(6): 339-342; 1977.

Antisera to adenovirus 12 (Ad12) DNA-binding protein (DBP) were prepared in the autochthonous monkey system and used to detect Ad12 DBP in Ad12-infected and -transformed cells. In human embryo kidney cells infected with Ad12 and stained with anti-DBP conjugate, fluorescent dots and granules were observed in the nucleus. When cynomolgus monkey kidney (CMK) cells were similarly infected and examined, fluorescent dots and granules were also observed. It is suggested that the fluorescent dots are specific structures related to the replication of Ad12 DNA. Studies with rat cells transformed with wild type or temperature-sensitive (ts) Ad12 indicated that most of the rat cell clones transformed by Ad12 wild-type or tsB contained DBP. None of the rat cells transformed by Ad12tsA contained DBP. Since AD12tsA is defective in the synthesis of DBP at the nonpermissive temperature, it is suggested that the tsA gene is either absent or defective in the synthesis of DBP in tsA-transformed cells. (9 refs)

78-2889 **RNA Associated with Murine Intracisternal Type A Particles Codes for the Main Particle Protein.** (Eng) Paterson, B. M. (Lab. Biochemistry, NCI, Bethesda, MD, 20014); Segal, S.; Lueders, K. K.; Kuff, E. L. *J Virol* 27(1): 118-126; 1978.

Intracisternal A-type particles were isolated from MOPC-104E myelomas grown sc in BALB/c mice and from cultured A/Jax mouse neuroblastoma (N4) cells. Polyadenylated RNA was prepared from the particles and tested in a cell-free translation system derived from rabbit reticulocytes. RNA from the two sources directed the synthesis of multiple polypeptides with similar distributions of electrophoretic mobilities in sodium dodecyl sulfate-polyacrylamide gels, including one component of the same size as the major A-particle structural protein (73,000 daltons). Analysis of the RNA's by electrophoresis in methyl mercury-agarose gels revealed a 35S component common to A particles from both cell types. This was the major component of the N4 preparations, but a 28S species predominated in the case of MOPC-104E. These two RNA's (35S from N4 cells and 28S from MOPC-104E), when isolated on isokinetic sucrose gradients, directed the synthesis of a 73,000-mol wt polypeptide that comigrated on gels with authentic A-particle structural protein. Identity of the cell-free product was confirmed by two-dimensional analysis of the ^{35}S-methionine-labeled tryptic peptides. The N4 RNA preparations also contained a major 32S component that did not code effectively for the A-particle structural protein. (39 refs)

78-2890 **Role of Gross A Virus-Induced Soluble Products as Potential Early Promoters of Thymic Carcinogenesis and Deregulators of Splenic Immune Responses in AKR Mice (Meeting Abstract).** (Eng) Ranney, D. F. (Northwestern Univ., Chicago, IL, 90024); Beardsley, T. R. *Fed Proc* 37(6): 1560; 1978. (no refs)

78-2891 **B-Tropic Oncornavirus Production by BALB/c Methylcholanthrene-induced Sarcoma Cells.** (Eng) Gillespie, G. Y. (Dept. Immunopathology, Scripps Clinic and Res. Foundation, 10666 N. Torrey Pines Rd., La Jolla, CA, 92037); Jensen, F. C.; Russell, S. W. *Int J Cancer* 21(2): 234-238; 1978.

Six oncogenic cell lines (M-133 to M-138) were established from primary sarcomas that developed in male BALB/cSt mice inoculated sc with 1 mg methylcholanthrene 5.5 mo after thymectomy. Injection of 10^5-10^6 cells from these lines induced tumors in all mice tested, and only M-135 failed to induce tumors with an inoculum size of 10^4 cells. Virion-associated reverse transcriptase (RT) activity was detected in supernatants from 4/6 lines, with M-138 supernatant fluid having the highest activity. One of the four RT-positive lines (M-137) produced xenotropic murine leukemia virus (MuLV) exclusively; the other three lines released only ecotropic MuLV, which infected secondary BALB/c mouse embryo fibroblasts but not those from NIH Swiss embryos. B-tropic MuLV from M-138 was used to infect subconfluent monolayers of BALB/c or NIH Swiss mouse embryo fibroblasts. Seven days later, B-tropic MuLV was present in large quantities in the BALB/c culture, but there was no evidence of infection in the NIH culture. Litters of BALB/c and NIH Swiss mice were inoculated ip with 3.6-6.0 x 10^4 plaque-forming units of M-138. All neonatal BALB/c mice demonstrated significant levels of p30 in their circulation, but the plasma from NIH mice remained negative for the antigen. Although the BALB/c mice remained positive for p30 for 9 mo, none of the animals developed clinical signs of leukemia. These findings show that endogenous MuLV may be produced by neoplastic cells induced in vivo with a chemical carcinogen. They underline the danger in assuming that a cell line is virus-free because it was isolated from a chemically-induced tumor. (35 refs)

78-2892 **T1 Oligonucleotides That Segregate with Tropism and with Properties of gp70 in Recombinants Between N- and B-tropic Murine Leukemia Viruses.** (Eng) Faller, D. V. (Dept. Biology, Center Cancer Res., Massachusetts Inst. Technology, Cambridge, MA, 02139); Hopkins, N. *J Virol* 26(1): 153-158; 1978.

The RNase T1-resistant oligonucleotides derived from the genomes of 16 recombinants between N- and B-tropic murine leukemia viruses of BALB/c mice were examined. SP-N, an N-tropic C-type virus that makes small XC plaques, and LP-B, a B-tropic virus that makes large XC plaques, served as parental viruses. These parental viruses also differed with respect to the electrophoretic mobility of virion proteins p15, p30, and gp70; their ability to induce GIX antigen on infected cells; and the presence of 6-8 of 36-38 large T1 oligonucleotides. One SP-N-specific T1 oligonucleotide was inherited by all 16 N-tropic recombinants and, thus, appears to be linked to N-tropism. This oligonucleotide lies in the 5′ third of the oligonucleotide map of SP-N. One LP-B-specific T1 oligonucleotide was inherited by all 11 recombinants whose gp70 had an electrophoretic mobility like that of LP-B gp70 and that, like LP-B, failed to induce GIX. This oligonucleotide lies in the 3′ third of the oligonucleotide map of LP-B. (23 refs)

78-2893 **Incidence in South-west Scotland of Hepatitis B Surface Antigen in the Liver of Patients with Hepatocellular Carcinoma.** (Eng) Turbitt, M. L. (Dept. Pathology, Glasgow Royal Infirmary, Stobhill Hosp., Glasgow, UK); Patrick, R. S.; Goudie, R.B.; Buchanan, W. M. *J Clin Pathol* 30(12): 1124-1128; 1977.

An immunoperoxidase technique for staining hepatitis B surface antigen (HBsAG) was applied to histologic sections of liver tissue stored as paraffin blocks, and a retrospective study was made of the incidence of the antigen in 81 cases of primary hepatocellular carcinoma (PHC) in southwest Scotland. PHC is responsible for only 0.46% of the deaths in this area, and, according to radioimmunoassay, HBs antigenemia is found in only 1/500 of the population. HBsAg was found in 8/81 cases of PHC and 4/82 cases of cirrhosis. There was no positive staining in 112 controls without overt liver disease matched for age and sex. Two mechanisms may occur; infection with hepatitis virus may progress to macronodular cirrhosis, which may later be complicated by PHC, or continuing infection may produce PHC in an otherwise normal liver. An alternative explantion for the findings could be that PHC activates latent hepatitis B virus, with the production of HBsAg in infected cells. This coincides with the observation that 7.5% of the normal people in southwest Scotland have antibody to HBsAg. (7 refs)

78-2894 **Rapid Induction of Colony Stimulating Activity (CSA)-Independent Granulocytic Colonies in Purified Bone Marrow Myeloid Cells by In Vitro Infection with RNA Type-C Viruses (Meeting Abstract).** (Eng) Greenberger, J. S. (Joint Center Radiation Therapy and Sidney Farber Cancer Inst., Boston, MA, 02115); Davisson, P. B.; Gans, P. J. *Proc Am Assoc Cancer Res* 19: 117; 1978. (no refs)

78-2895 **A Primary Target Demonstrating a Biologic Effect of RNA Type-C Leukemia Virus Infection is the Committed Granulocyte-Macrophage Progenitor Cell (Meeting Abstract).** (Eng) Greenberger, J. S. (Joint Center Radiation Therapy, Boston, MA); Davisson, P. B.; Gans, P. J. *Clin Res* 26(3): 347A; 1978. (no refs)

78-2896 **Structural Analysis of the Major Envelope Glycoprotein (gp70) of the Amphotropic and Ecotropic Type C Viruses of Wild Mice.** (Eng) Bryant, M. L. (Dept. Pathology, Univ. of Southern California Sch. Medicine, Los Angeles, CA); Pal, B. K.; Gardner, M. B.; Elder, J. H.; Jensen, F. C.; Lerner, R. A. *Virology* 84(2): 348-358; 1978.

Virus-associated and free serum gp70's major envelope glycoprotein were isolated from wild mice (*Mus musculus*) by immunoaffinity column chromatography and analyzed by two-dimensional tryptic peptide mapping procedures. The structure of the wild mouse gp70's was compared with that of homologous viral and serum gp70's of laboratory mouse strains. The results indicate that the gp70's of wild mouse ecotropic and amphotropic murine leukemia viruses (MuLV) are structurally divergent. The gp70's of wild mouse ecotropic viruses are more similar in structure to the Gross-AKR MuLV subgroup than to the Friend-Moloney-Rauscher subgroup. The gp70's of the amphotropic wild mouse viruses are highly conserved in structure and are closely related to those of one group of xenotropic MuLV found in laboratory mice. Non-virion-associated free serum gp70 of wild mice is homologous to that of inbred laboratory mouse strains and resembles the gp70 of a xenotropic virus spontaneously produced in NZB mice. These findings, combined with other results, suggest that multiple proviral sequences, each encoding a unique gp70 molecule, can be expressed in a give
mouse and that the presence of particular proviral gene se
quences can be identified by primary structural analysis o
specific gp70 molecules. (26 refs)

78-2897 **Biochemical Properties of Endogenous Rat C
type Viruses.** (Eng) Svec, J. (Cancer Res. Inst
Slovak Acad. Sciences, 880 32 Bratislava, Czechoslovakia
Michalides, R. *Neoplasma* 24(6): 601-614; 1977.

Biochemical properties of two endogenous rat le
kemia viruses (Sprague-Dawley, SD-RaLV, an
Wistar, W-RaLV) were examined in in vitro studie
Both SD-RaLV and W-RaLV particles, which we
obtained from spontaneously transformed and/or activ
ly replicating rat embryo fibroblasts, were mo
phologically and biochemically related to know
oncornaviruses. They are of the C-type, have a buo
ant density of 1.15 g/cm^3 in sucrose gradients, and contai
high-mol-wt RNA. The RaLV-associated DNA polymeras
exhibited both exogenous (polynucleotide-templated) and e
dogenous (viral RNA-templated) activity. The enzym
strongly preferred Mn^{2+} over Mg^{2+} ions, with max reactiv
ty occurring at 0.1 mM MnCl. The endogenous nature
SD-RaLV and W-RaLV was confirmed by molecular hybri
ization experiments. The number of proviral sequences w
calculated as 84 per haploid cell genome by competition h
bridization assays. Although it is not clear whether RaL
induced transformation is attributable to direct viral onc
genesis or to derepression or transduction of rat cellular onc
genes, these results demonstrate that the RaLV particles, a
though endogenous, contain the necessary enzymes f
reverse transcription and integration of the viral genome in
the host cell DNA. (38 refs)

78-2898 **Type-C Virus Structural Antigens on the Su
face of the Infected Cell as Determined by
Humoral Cytotoxicity Assay.** (Eng) Kende, M. (Lab. RN
Tumor Viruses, NCI, NIH, Bethesda, MD, 20014); Sass,
Hino, S.; Donahoe, R. M.; Kelloff, G. J. *Int J Cancer* 21(
194-203; 1978.

Antisera produced against the Rauscher murine leukem
virus (R-MuLV) proteins gp70, p12, p30, p10, and p
(E) were characterized by immunoprecipitation
highly purified labeled proteins of R-MuLV and sele
ed proteins of naturally occurring Class I, II, and
endogenous murine C-type viruses. The cytotoxici
of gp70 and p12 antisera showed a predominant ty
specificity to R-MuLV, but these antisera had va
ing degrees of cross-reactivity to the three kno
classes of endogenous C-type viruses. Immunoprec
tation of purified proteins with these antisera produced rea
tivity patterns approximating those obtained with humor

cytotoxicity. Unlike gp70, the cytotoxic reactivity of p12 antisera did not correlate well with the intact virus content or the virus-producing ability of different R-MuLV shedding mouse cells. Cytotoxic activity was not observed with p10 or p30 antisera, and virus-specific cytotoxic activity was not observed with p15 (E). Immunization of three strains of mice differing in their endogenous C-type virus expression with inactivated, disrupted R-MuLV yielded antisera active in radioimmunoassays with gp70, p12, p30, p15, and p10. The relationship of the gp70 and p12 immune response to the antiviral and antitumor effector immune responses operative in vivo is not known. (31 refs)

78-2899 **Use of Cowan I *Staphylococcus aureus* for Radioimmunoassay of Retrovirus Proteins.** (Fre) Deshayes, L. (Laboratoire d'Immunologie et de Virologie des Tumeurs, Hopital Cochin, Bat. G.-Roussy, 27, rue du Faubourg-Saint-Jacques, 75674 Paris Cedex 14, France); Levy, D.; Fournier, G.; Gilly, L. *C R Acad Sci [D] (Paris)* 286(5): 435-438; 1978.

Cowan I strain *Staphylococcus aureus* was used in the radioimmunoassay of C-type virus polypeptides in (1) bovine leukemia virus, (2) Rauscher murine leukemia virus, (3) serum from leukemic cows, (4) serum from a horse immunized against bovine leukemia virus, (5) serum from a rat immunized against a murine sarcoma virus-induced sarcoma, and (6) cow, rat and horse sera with rabbit antibodies. The use of *S. aureus* had several advantages (ease of preparation, speed and completeness of reaction between antigen and antibody) over the antibody immune sera generally used in radioimmunoassays. (11 refs)

78-2900 **Enhancement of the Growth of Implanted Tumors by a Murine Retrovirus (Meeting Abstract).** (Eng) Dennis, A. J. (Battelle's Columbus Lab., Columbus, OH, 43201); Barker, A. D.; Rice, J. M. *Fed Proc* 37(6): 1280; 1978. (no refs)

78-2901 **MAC-1, a New Genetically Transmitted Type C Virus of Primates: "Low Frequency" Activation from Stumptail Monkey Cell Cultures.** (Eng) Todaro, G. J. (Lab. Viral Carcinogenesis, NCI, Bethesda, MD, 20014); Benveniste, R. E.; Sherwin, S. A.; Sherr, C. J. *Cell* 13(4): 775-782; 1978.

A new class of endogenous primate C-type virus was isolated from a continuous tissue culture line of *Macaca arctoides* (stumptail monkey) spleen cells by cocultivation with a human cell line. The virus, designated MAC-1, can be transmitted to human and feline cells in tissue culture. By immunological and nucleic acid hybridization criteria, MAC-1 shows no detectable homology to the baboon C-type viruses, even though viral genes related to the latter group are readily detected in *M. arctoides* cellular DNA. Viral gene sequences related to the MAC-1 genome are present in multiple copies (50-150 per haploid genome) in Old World primates and are expressed in the cellular RNA's of uninfected and ostensibly virus-free primate cells and tissues. There are, therefore, at least two distinct sets of genetically transmitted Old World primate C-type viral genes, each of which is found in multiple copies in normal primate cellular DNA. (26 refs)

78-2902 **Infectivity of Endogenous Baboon Type-C Virus-Related Genes.** (Eng) McAllister, R. M. (Childrens Hosp. Los Angeles, Los Angeles, CA); Nicolson, M. O.; Heberling, R.; Charman, H.; Rice, N.; Gilden, R. V. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 135-138; 1978.

DNA's from two baboon placentas, four embryonic tissues, and three embryonic cell lines were tested for infectivity in human rhabdomyosarcoma TE-32 cells and dog D-17 sarcoma cells. The DNA's of two placentas and one cell line derived from two baboons were infectious. These materials released infectious baboon virus and contained about 100 viral genome equivalents per haploid gene. However, an embryonic cell line from a third baboon was not infectious, although it also contained 100 viral genomes and released virus. DNA's of the four embryonic tissues and one embryonic cell line from a fourth baboon contained only 20 genomes, did not release virus, and were not infectious. TE-32 and D-17 cells infected with baboon virus contained only 5-6 genome equivalents, but they released high virus titers and their DNA's contained infectious baboon provirus. These results demonstrate the complexity of the factors influencing the infectivity of baboon C-type virus-related genes. (11 refs)

78-2903 **Immunological Demonstration of Oncornavirus Genome in Leucocytes in Acute and Chronic Myelogenous Leukaemia and Preleukaemia.** (Eng) Toth, F. D. (Inst. Microbiology and 2nd Dept. Medicine, Univ. Medical Sch., H-4012 Debrecen, Hungary); Jako, J.; Vaczi, L.; Rak, K.; Redai, I. *Acta Microbiol Acad Sci Hung* 24(4): 331-339; 1977.

Anti-LPV p30 serum and antisera to the corresponding antigens of different mammalian C-type oncornaviruses were used to demonstrate C-type oncornavirus cytoplasmic antigens in the peripheral WBC of 10 patients with acute myelogenous leukemia (AML), 9 potentially preleukemic patients, 16 patients with chronic myelogenous leukemia (CML), and 42 control patients with cytopenia or nonhematologic disease. Indirect immunofluorescence testing showed the WBC of all AML patients to be positive with anti-LPV serum and negative with anti-feline leukemia virus serum. These WBC also cross-reacted with antisera to simian and/or murine cytoplasmic oncornavirus antigens, depending on the stage of disease of the patient. Of the 9 potentially preleukemic patients, 6 had LPV-p30-positive WBC, and most of these cross-reacted with antisera to simian p30 antigens. Of 11 CML patients responding to chemotherapy, 5 were negative for oncornavirus cytoplasmic antigens and 6 showed endogenous oncornavirus antigens in the WBC of the remaining five CML patients, including three poor responders in the early and fully developed blastic stages. Cytoplasmic fluorescence with anti-baboon serum was observed in two controls; all the others were negative to all antisera. (25 refs)

78-2904 **Cross-Reactivity of Mammalian C-Type Viruses in Virion Precipitation Reaction.** (Rus) Zakharova, L. G. (D. I. Ivanovsky Inst. Virology, Moscow, USSR); Al'tshtein, A. D.; Zhdanov, V. M. *Vopr Virusol* (1): 25-31; 1978.

Use of the virion precipitation reaction (VPR) to differentiate among the various C-type viruses is described. Incubation of Rauscher leukemia virus, Gross leukemia virus (G-MuLV), murine sarcoma virus (NIH-MSV), feline leukemia virus, endogenous feline virus (RD-114), endogenous baboon virus (BEV), exogenous simian sarcoma virus, exogenous gibbon lymphoma virus, Prague strain of avian Rous sarcoma virus (Pr-RSV), and murine mammary tumor virus (MMTV) with goat immune sera against the test viruses and against bovine leukemia virus (BLV) revealed cross-reacting antigenic determinants on the surface of RD-114, NIH-MuLV, and G-MuLV. These determinants could not be detected on the surface of BLV, Pr-RSV, and MMTV. (18 refs)

78-2905 **C-Type Oncornaviral Antigens in Cocultures of Human Leukaemic Bone Marrow Cells.** (Eng) Zurcher, C. (Inst. Experimental Gerontology TNO, Rijswijk, Netherlands); Coolen, J.; Nooter, K.; Haaijman, J. J.; Koch, G.; Bentvelzen, P. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 144-147; 1978.

In an attempt to increase the expression of C-type virus antigens in human bone tumor cultures, bone marrow (BM) from eight leukemia patients was cocultivated with a dog thymus cell line (A7573) permissive for the propagation of human C-type virus. Three leukemia BM cocultures showed a clear-cut cytoplasmic fluorescence after incubation with antisera against the p30 antigen of the simian sarcoma virus complex (SSV). The three patients with positive reactions were children with acute lymphoblastic leukemia. No fluorescence was found in cocultured BM from eight patients with nonleukemic hematological disorders or from seven normal controls. Reactions with antisera against Rauscher murine leukemia virus were negative. Tests of antisera specificity indicated that the SSV-related information was present in the original BM samples and that cocultivation with A7573 cells resulted in virus antigen expression. (4 refs)

78-2906 **Possible Significance of Type-C Viral Information in Human Leukemic Cells.** (Eng) Campbell, W. F. (Mayo Clinic, Rochester, MN, 55901); Hoagland, H. C.; Smith, D. M. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 150-153; 1978.

Short-term cultures of acute myelogenous leukemia (AML) cells released virus particles containing reverse transcriptase activity and a high-mol-wt RNA (60S) that shared limited homology with woolly monkey sarcoma virus, but not with baboon endogenous virus (BEV) or murine sarcoma virus (MSV). To determine the origin of the C-type virus, a complementary DNA (cDNA) transcript of the viral RNA was prepared using avian myeloblastosis virus reverse transcriptase. Preliminary hybridization studies indicated that DNA from leukemic and nonleukemic cells contained comparable levels of homology with the AML cDNA probe, but that selective expression of the genome occurred only in leukemic cells. The AML viral cDNA hybridized to 50%-100% of leukemic cell RNA, compared with 6%-9% of nonleukemic cell RNA. (15 refs)

78-2907 **Detection of Common Antigenic Determinants in the Capsids of D-Type (Mason-Pfizer) and C-Type Mammalian Viruses by Virion Precipitation Reaction.** (Rus) Al'tshtein, A. D. (D. I. Ivanovskii Inst. Virology, Moscow, USSR); Zakharova, L. G.; Zhdanov, V M. *Vopr Virusol* (3): 309-314; 1978.

The virion precipitation assay was used to analyze and compare the capsid antigens of D-type Mason-Pfizer monkey virus (M-PMV) and a series of B- and C-type oncornaviruses. Goat immune sera against M-PMV precipitated the mammalian C-type viruses Rauscher murine leukemia virus

(MuLV), Gross MuLV, NIH MuLV, feline leukemia virus, RD-114, baboon endogenous virus, simian sarcoma virus 1, and gibbon ape leukemia virus, but not mouse mammary tumor virus, Prague strain Rous sarcoma virus, and bovine leukemia virus. (13 refs)

78-2908 **Skunks Have Gene Sequences in Their Cellular DNA Related to Squirrel Monkey Retrovirus: Transmission Between Species of a New World Primate Endogenous Type D Retrovirus.** (Eng) Benveniste, R. E. (Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD, 20014); Wilson, G. L.; Todaro, G. J. *Biochem Biophys Res Commun* 81(4): 1363-1369; 1978.

The possibility that genetically transmitted viruses of New World primates could have been transmitted to the DNA of other families of mammals was tested using an endogenous D-type virus from squirrel monkeys. In a series of molecular hybridization experiments with the DNA's of a variety of primate and nonprimate species, sequences related to the squirrel monkey virus were not detected in any other primate species, including six other genera of New World monkeys. However, when the DNA's of the New World carnivors were examined, the sequences were detected in the cellular DNA of normal tissues of two members of the family Mustelidae, the skunks *Mephitis mephitis* and *Spilogale putorius*. Skunk cellular DNA did not hybridize significantly to a transcript prepared from the other endogenous New World primate virus (owl monkey), to any other primate virus (langur, baboon, stumptail, gibbon), or to cat or mouse retroviruses. It is concluded that the D-type virus was transmitted between the germ lines of the two evolutionarily distant species. This example of interspecies transmission is the first that has been described among New World species and also the first that has been demonstrated for retroviruses other than C-type viruses. (22 refs)

See also:

*(Rev.): 78-2429, 78-2442, 78-2443, 78-2444, 78-2445, 78-2446, 78-2452.
*(Chem.): 78-2531, 78-2547, 78-2609.
*(Phys.): 78-2740, 78-2747, 78-2748.
*(Immun.): 78-2918, 78-2920, 78-2921, 78-2922, 78-2923, 78-2924, 78-2925, 78-2926, 78-2927, 78-2928, 78-2929, 78-2931, 78-2932, 78-2934, 78-2935, 78-2936.
*(Path.): 78-2940, 78-2963.
*(Epid.-Biom.): 78-2981.

78-2909 **In Vitro Induction of Tumour-specific Immunity. V. Detection of Common Antigenic Determinants of Murine Fibrosarcomas.** (Eng) Burton, R. C. (Genetics Unit, Walter and Eliza Hall Inst. Medical Res., Melbourne, Australia); Warner, N. L. *Br J Cancer* 37(2): 159-170; 1978.

In vivo and in vitro tumor-immune responses of male BALB/c mice to two 3-methylcholanthrene-induced fibrosarcomas (WEHI-164 and -167) and one spontaneous fibrosarcoma (WEHI-11) were examined. When BALB/c mice were immunized to these fibrosarcomas by surgical tumor removal, cross-reacting tumor-associated transplantation antigens (TATA) were detected on all three tumors. Cytotoxic effector cells (CL) were then induced in vitro by coculture of BALB/c spleen cells with the spontaneous or one of the carcinogen-induced fibrosarcomas. These CL were shown to be cytotoxic T cells (Tc) and to be directed against cross-reacting tumor-associated antigens (TAA) on all three tumors by two ^{51}Cr-release assays. The TAA involved in the in vitro induction of Tc were not present on normal adult or fetal tissues. A secondary cytotoxic response was also detected in vitro when spleen cells from mice immunized to a carcinogen-induced fibrosarcoma were tested. The patterns of cross-reactivity detected by the in vivo and primary in vitro tumor-immune responses suggested that the in vivo TATA were not identical to the TAA detected in vitro. (43 refs)

78-2910 **Soluble Suppressor Factor from the Spleens of Tumor-bearing Mice.** (Eng) Subramanian, C. (Dept. Surgery, Univ. Minnesota, P.O. Box 85, Mayo Memorial Building, Minneapolis, MN, 55455); Yu, S.; McKhann, C. F. *Cancer Res* 38(7): 1996-2002; 1978.

A soluble suppressor factor released from the spleens of C3H/HeJ female mice bearing 3-methylcholanthrene-induced tumors (MC43 fibrosarcoma) is described. This factor was released in vitro into the media 3-5 days after culture of spleen cells from tumor-bearing animals, and it suppressed the stimulation of lymph node and spleen cells by tumor antigen or mitogens. Spleens from mice carrying the MC43 tumor for 14 days were capable of producing suppressor factor in vitro, but those from mice bearing the tumor for $\leq$10 days failed to do so. Lymph node cells from the same animals did not produce suppressor factor in vitro. The suppressor factor was produced by a nonadherent cell population, was heat-stable, was lost on dialysis, and did not appear to be tumor antigen or thymidine. (21 refs)

78-2911 **Perturbation of General and Anti-tumor Immune Competence in Mice Bearing a Chemically Induced Tumor (Meeting Abstract).** (Eng) Dean, J. H. (Litton Bionetics, Inc., Kensington, MD, 20795); McCoy, J. L.; Lewis, D. D.; Law, L. W. *Proc Am Assoc Cancer Res* 19: 208; 1978. (no refs)

78-2912 **Stimulation of Malignant Skin Cells by Antibody to Normal Skin Cells of Mice.** (Eng) Heidrick, M. L. (Dept. Biochemistry, Univ. Nebraska Coll. Medicine, 42nd St. and Dewey Ave., Omaha, NB, 68105); Ryan, W. L.; Curtis, G. L. *J Natl Cancer Inst* 60(6): 1419-1425; 1978.

The ability of antibodies developed against normal skin cells to stimulate skin cells transformed by 7,12-dimethylbenz(a)anthracene (DMBA) was determined. Primary cultures of normal skin, containing both fibroblasts and epithelial cells, were obtained from adult A/J mice. Malignant skin cells were obtained by treating a subculture of normal cells with medium containing 0.5 μg/ml DMBA. Transformation was demonstrated by increased growth rate, growth in soft agar, and production of tumors in A/J mice. Antisera developed against the normal cells were cytotoxic to both normal and malignant cells in the presence of complement at a 1:320 dilution. However, dilutions of 1:500 to 1:1,000 in the absence of complement produced growth enhancement of the malignant but not the normal cells. The growth-enhancing properties were present in the γ-globulin fraction of the antisera that contained IgG, IgM, and IgA antibodies. Immunofluorescence studies indicated that antibodies from the sera bound to membranes of both normal and malignant cells. These findings suggest that autoantibodies against normal tissues, which develop as a result of injury or other mechanisms, can specifically stimulate the growth of malignant or premalignant cells. (27 refs)

78-2913 ***Corynebacterium parvum*-Induced Resistance to a Methylcholanthrene Fibrosarcoma.** (Eng) Gupta, J. D. (Children's Medical Res. Foundation, Camperdown, New South Wales 2050, Australia); Morahan, P. S.; Kaplan, A. M. *J Reticuloendothel Soc* 23(1): 1-9; 1978.

The effectiveness of intratumor injections of *Corynebacterium parvum* against the 3-methylcholanthrene-induced tumor

MCA 2182 was determined in male C57BL/6 mice. Injection of 0.5-70 mg/kg *C. parvum* 3 days after tumor transplantation into the footpads produced 50%-88% tumor-free survivors; a dose of 0.056 mg/kg was ineffective. Treated animals with progressing tumors generally showed a survival time similar to that of control animals. Animals surviving without evidence of tumors after treatment with 70 mg/kg demonstrated specific tumor immunity when challenged with the same tumor, but not with a heterologous tumor. Prophylactic footpad inoculation with *C. parvum* 7 or 3 days before tumor inoculation produced more tumor-free survivors than did therapeutic treatment, but the surviving animals did not exhibit specific transplantation resistance. When tumors were treated on day 7 after tumor cell injection, only a dose of 70 mg/kg produced tumor-free survivors. Intratumor injection of *C. parvum*-activated macrophages on day 3, at a ratio of 25 macrophages to each tumor cell previously injected, produced a marked inhibition of tumor growth. Furthermore, a significant number of tumor-free survivors (50%) was observed with intratumor injection of activated macrophages compared to normal macrophages (10%). This effect was not observed with injection on day 7; in fact, a slight tumor growth enhancement was sometimes observed. (22 refs)

78-2914 **Vulnerability of Methylcholanthrene-induced Tumours to Immunity Aroused by Syngeneic Foetal Cells.** (Eng) Medawar, P. B. (Clinical Res. Centre, Watford Road, Harrow, Middlesex, England); Hunt, R. *Nature* 271(5641): 164-165; 1978.

Evidence is presented that protection against tumors is secured by immunity to embryo-specific antigens aroused by normal pregnancy or by deliberate inoculation of embryonic cells. Irradiated CBA fetal cells were injected into CBA mice 7 or 14 days before or after or at the same time as an injection of 50 μg methylcholanthrene (MC). Comparisons among the five groups indicated that injection of embryonic cells 7 or 14 days post-MC promoted tumor growth, but that injection of fetal cells 7 or 14 days pre-MC retarded the onset and lessened the number of induced tumors. These results might explain the influence of parity and age at first childbearing on susceptibility to mammary cancer in humans. The protective effect of an early pregnancy is simulated by the results of experiments in which immunity is aroused before the application of the oncogenic stimulus. The lessened effectiveness or even increased hazard of breast cancer associated with late pregnancy is simulated by the enhancing effect on tumor growth found when the inoculation is deferred until after the application of the oncogenic stimulus. The underlying assumption in both situations is that the transformation from normal to malignant growth occurs very shortly after MC injection and very early in reproductive life. (8 refs.)

78-2915 **Antigenic Differences Between AKR Lymphoma and Thymus Cells Leading to the Detection of a Tumor Antigen Associated with Immunological Enhancement.** (Eng) Laguens, R. P. (Catedra de Patologia II, Facultad de Medicina, Universidad Nacional de La Plata, Buenos Aires, Argentina); Colmerauer, M. E.; Segal, A.; Pasqualini, C. D. *Int J Cancer* 21(6): 779-783; 1978.

An indirect immunofluorescence (IF) test using lymphoma and thymus cells as targets was used to compare the sera of tumor (AKR lymphoma L15)-bearing BALB/c mice progressors with those of mice that had rejected the tumor (regressor). An intensively positive IF reaction was obtained when L15 lymphocyte and thymus cells, either unfixed or acetone-fixed, were incubated with progressor serum. After incubation with regressor serum, however, a positive IF reaction was observed only on unfixed L15 and fixed and unfixed thymus cells. The IF results indicated that the progressor serum contained three different antibodies, designated a, b, and c, that combined with the corresponding antigens, A, B, and C, present on L15 cells. The regressor serum, however, possessed only two of these antibodies, b and c, which reacted with the two antigens (B and C) that L15 shares with thymus cells. In vivo experiments showed that pretreatment of BALB/c mice with the acetone-resistant tumor antigen A of L15 cells led to a significant increase in allogeneic tumor incidence (76%, compared with 37% in controls). It is concluded that in this allogeneic model, an acetone-resistant tumor-specific antigen and its corresponding antibody are involved in tumor enhancement. (18 refs)

78-2916 **H-2 Antigens on a Murine Lymphoma Are Associated with Additional Proteins.** (Eng) Callahan, G. N. (Dept. Immunology, Scripps Clinic and Res. Foundation, 10666 N. Torrey Pines Road, La Jolla, CA 92037); Allison, J. P. *Nature* 271(5641): 165-167; 1978.

Immunochemical evidence is presented for the alteration of histocompatibility (H-2) antigens on tumor cells by association with additional proteins. Normal C3H splenocytes and 6C3HED tumor cells (an estrogen-induced lymphoma) were radiolabeled, extracted with detergent, and reacted with either anti-H2.23 (H-2.23 is the private *K*-locus antigen of the H-2K haplotype) or normal mouse serum. The resulting antigen-antibody complexes were analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Proteins with mol wts of 45,000 and 12,000, characteristic of detergent-solubilized H-2 and β_2-microglobulins (β_2-μ), were present in the extracts from both normal and tumor cells. Additional major peaks corresponding to mol wts of 70,000 and 55,000 were evident in the 6C3HED cell extract. Similar peaks were obtained when anti-H-2.23 and anti-β_2-μ were reacted with detergent eluates of tumor cells that had been radioiodinated with lactoperoxidase, indicating that the antigens detected were on the cell surface. One possible explanation for these results is that a complex composed of at least one β_2-μ, one H-2 heavy chain, and one or two higher mol wt proteins is formed on the tumor cells. In detergent extracts, this complex precipitates with anti-H-2.23 and anti-β_2-μ as a single entity and then is reduced to its individual protein components when the precipitates are prepared for electrophoresis. (16 refs.)

78-2917 **Resistance to Transplantation of Spontaneous AKR Lymphoma Cells by Substrains of AKR Mice. In Vivo Resistance and Generation of an H-2 Restricted CML Response.** (Eng) Zatz, M. M. (Immunology Branch, NCI, NIH, Bethesda, MD, 20014). *Cell Immunol* 36(2): 251-262; 1978.

The observation that the AKR/J (H-2k, Thy 1.1) and AKR/Cu (H-2k, Thy 1.2) substrains of AKR mice each have a high incidence of T-cell lymphoma between 6 and 12 mo of age, but they reject the spontaneously arising lymphoma of the reciprocal strain, was investigated. During the rejection, splenic precursor cells were generated that, upon secondary stimulation in vitro, resulted in specific cytotoxic T cells. The antigenic component of AKR cells resulting in this secondary cell-mediated lympholysis response resided on both lymphoma and normal T and B cells, and it was distinct from the Thy antigen. Primed AKR/J lymphocytes responded to and lysed only immunogens and targets homologous at the K and/or D end of the major histocompatibility complex (MHC), but primed AKR/Cu cells required homology at the D end of the MHC. (18 refs)

78-2918 **SIM and SIM-R Mice Are Histoincompatible.** (Eng) Furmanski, P. (Dept. Biology, Michigan Cancer Foundation, Detroit, MI, 48201); Dietz, M.; Hines, D. L.; Marcelletti, J. *Virology* 86(1): 287-290; 1978.

Attempts were made to breed SIM and SIM-R mice for congenicity at the Fv-1 locus. SIM mice were permissive for N- and NB-tropic viruses and restricted B-tropic viruses, but the SIM-R mice were permissive for B- and NB-tropic viruses and restricted N-tropic viruses. Histocompatibility was determined by skin graft reactions of the SIM and SIM-R mice. These mice rejected all skin grafts made between them within 14 days of grafting. Further analysis of histocompatibility differences was made in cytotoxicity tests using H-2 typing alloantisera. The results indicated that all antigenic specificities recognized by D-13 serum on SIM-R cells were also present on BALB/c cells, but that the converse was not true. Furthermore, there were differences between SIM and SIM-R cells other than those detected by the D-13 serum. Studies of these differences indicated that although there was a strong reaction in cultures of SIM or SIM-R cells with allogeneic BALB/c or C57BL/6 lymphocytes, there was no significant reaction in mixtures of SIM and SIM-R cells. Thus, although the SIM and SIM-R mice differ in antigens specified by the D end of the major histocompatibility complex (at least as determined by the D-13 serum), they are the same at the closely linked I locus. (23 refs)

78-2919 **Splenomegaly, Anemia and Reticular Neoplasms in the Congenic Resistant Mouse Strain B10.Y.** (Eng) Dux, A. (Dept. Genetics, Netherlands Cancer Inst., Amsterdam, Netherlands); Faraldo, M. J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 74-77; 1978.

The incidence of spontaneous malignancies was compared in two congenic C57BL mouse strains (B10 and B10.Y) that differ with respect to the major histocompatibility complex (H-2) haplotype. Splenomegaly, which persisted for life, was present in 3-mo-old B10.Y mice (H-2pa). Beginning at age 4 mo, the animals developed severe anemia. Lymphoproliferative lesions appeared by age 4 wk and reticulum cell sarcomas (mostly type B) by age 17 wk. A few type A reticular tumors, lymphocytic leukemias, and plasma cell tumors were also observed. The B10 mice (H-2b) did not develop splenomegaly or anemia, and reticular neoplasms in this strain appeared very late in life. The changes in the B10.Y strain are attributed to the effect of the H-2 complex. (5 refs)

78-2920 **Mammary Leukaemia (ML) Antigen Isolated from L 1210 Leukaemia Cells.** (Eng) Zak-Nejmark, T. (Dept. Tumour Immunology, Ludwik Hirszfeld Inst. Immunology and Experimental Therapy, Wroclaw, Poland); Steuden, J.; Radzikowski, C. *Int J Cancer* 21(4): 490-495; 1978.

Crude 3 M KCl extracts of murine leukemia L-1210 cells purified by polyacrylamide gel electrophoresis (PAGE) and the mammary leukemia (ML) isolated from these cells purified by affinity chromatography were characterized, and their identity was verified by anti-ML sera obtained from allogenic and xenogeneic hosts. The crude, soluble extract from L-1210 cells neutralized the cytotoxic activity from anti-ML and anti-H-2d antisera. The cytotoxic activity of anti-H-2d aloserum (diluted 1:8) was decreased from 100% to 17%. After absorption, the hetero- and alloanti-ML sera also appeared to be noncytotoxic for ML-positive leukemia cells. Selected purified ML-positive fractions and purified ML antigen were analyzed by PAGE. In each instance a single, sharp band was obtained with a mol wt estimated to be 73,500 daltons. Weak bands with mol wt of 80,000 to 90,000 were also observed. The 73,500-dalton band contained carbohydrate. It was estimated that ML antigen constitutes 0.044% of the total cellular proteins and that a single L-1210 cell contains 7.62 x 10^5 molecules of ML antigen. (30 refs)

78-2921 **Viral Structural Antigens on the Surface of Mouse Mammary Tumor Cells Detected by**

Serum Cytotoxicity, Membrane Immunofluorescence and Lactoperoxidase-Catalyzed Iodination (Meeting Abstract). (Eng) Massey, R. J. (Frederick Cancer Res. Center, Frederick, MD); Arthur, L. O.; Fine, D. L.; Schochetman, G. *Fed Proc* 37(6): 1772; 1978. (no refs)

78-2922 **Immunization of Mice Against Murine Mammary Tumor Virus Infection and Mammary Tumor Development.** (Eng) Sarkar, N. H. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Moore, D. H. *Cancer Res* 38(5): 1468-1472; 1978.

Formalin-inactivated whole murine mammary tumor virus (MuMTV), MuMTV membranes, the acid-soluble component of MuMTV, and purified MuMTV glycoprotein with a mol wt of 55,000 (gp55; also called gp52) were used as vaccines in an attempt to identify the MuMTV antigen(s) that can protect C57BL mice from exogenous MuMTV infection and subsequent tumor development. Formalin-inactivated whole MuMTV, MuMTV membranes, and purified MuMTV gp55 were effective immunogens, but the acid-soluble component of MuMTV (containing mainly MuMTV gp55) failed to protect mice from challenge with live virus. These results suggest that MuMTV gp55 is the major immunizing antigen and that its native conformation must be maintained for it to be an effective vaccine. Thus, MuMTV vaccines can control exogenous infection in animals that do not normally overtly express endogenous virus; they can also prevent tumor development in strains of mice that have a low virus load. (26 refs)

78-2923 **Stimulation of Immune Response in Hybrid Mice Following Rauscher Virus Infection.** (Eng) Okunewick, J. P. (Viral Oncology Section, Cancer Res. Unit, Clinical Radiation Therapy Res. Center, Allegheny General Hosp., Pittsburgh, PA, 15212); Meredith, R. F.; Brozovich, B.; Weaver, E. V. *Proc Soc Exp Biol Med* 157(3): 449-452; 1978.

The plaque-forming cell (PFC) response following infection by the NB-tropic strain of Rauscher leukemia virus (RLV) was investigated in the sensitive SJL/J mouse, the resistant C57BL/10 mouse, and their resistant (SJL/J x C57BL/10)F_1 hybrid. With SJL/J mice, a brief elevation in PFC response was observed when the virus was injected 1 day before or immediately before sheep RBC administration. This was followed by a depression in response, reaching minimum levels at day 21. C57BL/10 mice showed a slight depression in PFC response at day 2 followed by an increase that became significant at day 12. With the hybrid, a significant immunodepression was evident at day 1. This was followed by a rapid recovery in PFC response, peaking at a level twice normal by day 8; the level subsequently dropped off but remained high throughout the observation period. C57BL/10 mice showed no increase in spleen size following infection, and although the hybrid showed a threefold increase in spleen size, this did not correspond with the peak of PFC activity. However, some of the increases in PFC response were due to an overall elevated number of spleen cells in the infected hybrid. In contrast, SJL/J mice developed the greatest splenomegaly, but this strain showed virtually complete suppression of the total splenic PFC response. (25 refs)

78-2924 **Contrasting Immune Response Following Rauscher Leukemia Virus Infection in Virus-Susceptible vs. Virus-resistant Mice.** (Eng) Meredith, R. F. (Viral Oncology Section, Cancer Res. Unit, Clinical Radiation Therapy Res. Center, Div. Radiation Oncology, Allegheny General Hosp., Pittsburgh, PA, 15212); Okunewick, J. P.; Brozovich, B. J.; Weaver, E. V. *Dev Comp Immunol* 2(1): 133-146; 1978.

Humoral (plaque-forming) and cellular (mitogenic) immune responses were studied in virus-susceptible SJL/J mice and virus-resistant C57BL/10 mice following their infection with a threshold lethal dose of Rauscher leukemia virus. The plaque-forming response was suppressed in the SJL/J mice, but in the C57BL/10 mice, it was slightly elevated during the second and third weeks after infection. This suppression and elevation correlated with the development of severe viremia and a continued low level titer of virus, respectively. SJL/J mice exhibited splenomegaly with the virus infection, but C57BL/10 mice did not. Increasing the virus dose by a factor of 40 did not significantly alter the humoral response pattern of either strain, and it did not cause the C57BL/10 response to mimic that of the SJL/J mice. For both strains, the cellular and humoral immune response patterns differed. Depression of T-cell-mitogen responses occurred in the SJL/J mice, but at a later time than humoral response suppression. Neither T- nor B cell mitogen responses were affected in the C57BL/10 mice. The effect on mitogen responsiveness appeared to be independent of that on plaque formation in both strains. (40 refs)

78-2925 **T- and B-Cell Populations and Virus Antigen Expression in Lymphoid Tissues of Mice During Moloney- and Gross-Virus Leukemogenesis.** (Eng) Krueger, G. R. (Immunopathology Labs., Pathology Inst., Univ. Cologne, Clinical Center Lindenburg, 5000 Cologne 41, W. Germany); Bedoya, V. A.; Fischer, K. M.; Flesch, H. G. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 316-317; 1978.

The behavior of T and B cells in the lymphoid tissues of mice infected with leukemogenic viruses was studied and correlated with the appearance of virus-related antigens in lymphoid cells. Significant decreases in T- and B-cell receptors were noted before malignant proliferation could be diagnosed histologically. The expression of virus-related antigens was not quantitatively related to the expression of T- and B-cell receptors. Malignant lymphomas did not originate at the site where most cells carrying virus-related antigens were found initially. (6 refs)

78-2926 **Activation of Thymus-dependent Cell-mediated Cytotoxicity Against Virus Induced Tumor Cells by Antibody.** (Eng) Lamon, E. W. (Dept. Surgery, Sch. Medicine, Univ. Alabama in Birmingham, Birmingham, AL, 35294); Whitten, H. D.; Walia, A. S.; Fuson, E. W.; Cox, B. A.; Ayers, R. D. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 462-465; 1978.

The role of antibodies in the induction of antibody-dependent cell-mediated cytotoxicity (ADCC) was investigated. Serum from Moloney murine sarcoma virus (M-MuSV) regressor mice induced cytotoxicity specific for MuSV-transformed cells but not for polyoma virus-transformed cells. Serum from polyoma-immune mice induced thymocyte cytotoxicity only against polyoma-transformed cells and not against MuSV- or simian virus 40 (SV40)-transformed cells. However, antiserum raised against syngeneic embryo fibroblasts induced thymocyte cytotoxicity against all three cell lines. Further studies indicated that the effector cells included thymocytes, with cortisone-resistant medullary thymocytes being the most active. Both IgM and IgG induced ADCC, but the former was 400-500 times more efficient than the latter. The structural bases for IgG and IgM ADCC by T cells appear to be the Fc receptors on the T cells. A soluble IgG Fc receptor released from activated T cells was isolated and found to be a glycoprotein with a mol wt of 40,000 daltons. This protein increased the ability of T cells or macrophages to bind and lyse IgG-sensitized target cells. (12 refs)

78-2927 **Antibody Directed Against Endogenous C-Type Virus Is Immunosuppressive.** (Eng) Moroni, C. (Friedrich Miescher-Institut, P.O. Box 273, CH-4002 Basel, Switzerland); Schumann, G. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 174-175; 1978.

Antiserum against lipopolysaccharide-induced BALB/c endogenous xenotropic C-type virus suppressed the normal immune response of mice to sheep RBC. The immunosuppressive activity of the antiserum was reduced by absorption with xenotropic BALB/c virus and Friend leukemia virus, but not by absorption with Rous sarcoma virus or tobacco mosaic virus. The association between an endogenous viral antigen and immunosuppression is consistent with the hypothesis that endogenous C-type viruses play a role in the immune system. (2 refs)

78-2928 **Binding of Human Antibodies to Purified Primate C-Type Viral Antigens.** (Eng) Kurth, R. (Friedrich Miescher Laboratorium, Max Planck-Gesellschaft, Spemannstrasse 37-39, 74 Tubingen, W. Germany); Teich, N. M.; Weiss, R. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 41-45; 1978.

Human sera from (A) healthy blood donors and (B) laboratory personnel working with C-type animal viruses were examined for the presence of antigens to the simian sarcoma virus/simian sarcoma-associated virus [SSV(SSAV)] complex. Group B tended to have higher antibody titers to SSV(SSAV) than Group A, but wide variations were also apparent within Group B. Although the possibility that these antibodies were induced by nonviral antigens cannot be excluded, it is also possible that oncoviruses of monkeys share antigenic determinants with putative human oncoviruses. (12 refs)

78-2929 **Immune Reactivity of Visna Virus-inoculated Mice.** (Eng) Svennerholm, B. (Dept. Virology, Inst. Medical Microbiology, Univ. Goteborg, Goteborg, Sweden); Strannegard, O.; Lycke, E. *Infect Immun* 20(2): 412-417; 1978.

The effect of Visna virus on the development of humoral and cell-mediated immunity was investigated in Swiss albino mice. Mice were inoculated with crude or purified Visna virus and, 5 days later, immunized with sheep RBC; after another 5 days, direct and indirect plaque-forming cells (PFC) were assayed. A pronounced suppression of direct and indirect PFC was observed. The suppressive effect increased with decreasing concentrations of virus injected; max suppression occurred at a virus dilution of 10^{-4}. The effect of the virus on the secondary antibody response was measured by challenge with sheep RBC 28 days after the initial inoculation. Five days later, the direct PFC response, but not the indirect response was significantly suppressed. Injection of virus 4 or more days before sheep RBC injection inhibited the PFC response. This inhibition was most pronounced if 15 days had elapsed between the virus and sheep RBC injections, but it disappeared if 30 days had elapsed. If the virus was administered after the sheep RBC, enhancement was observed. Heating the virus at 56 C for 30 min abolished its immunosuppressive capacity. Further studies indicated that the number of "background" antibody-producing spleen cells increased slightly after virus injections. Virus was injected into DBA/1J mice before they were sensitized to picryl chloride (PC), and the animals were challenged with PC 5 days after sensitization. A suppressive effect was observed when virus was injected 2 wk before sensitization but not 3 or 4 wk before sensitization. In other experiments with DBA/1J mice, virus diluted 10^{-1} suppressed mitogen-induced DNA synthesis in thymus cell cultures significantly; virus diluted 10^{-3} enhanced the synthesis. No interferon activity could be demonstrated in the Visna virus inocula or in the sera of mice 0-14 days after virus inoculation. (19 refs)

78-2930 **The Detection of a Tumor-Specific Transplantation Antigen on Nonvirus-Producer Cells by Cell-Mediated Cytotoxicity.** (Eng) Smith, K. A. (Hematology Res., Dartmouth Medical Sch., Hanover, NH, 03755); Ferm, M. M.; Gillis, S.; Gillis, A. E. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 355-356; 1977.

Secondary and tertiary in vitro lymphocyte sensitization through mixed tumor-lymphocyte culture led to the successive enhancement of the generation of tumor-specific cytotoxic T lymphocytes, which has allowed the detection of a tumor-specific transplantation antigen on both virus-producing and nonproducer tumor cells. (2 refs)

78-2931 **Cell-mediated Immunity to Antigens Associated with Primate C-Type Oncornaviruses in Pregnant Women.** (Eng) Hirsch, M. S. (Massachusetts Gen. Hosp. and Harvard Med. Sch., Boston, MA); Kelly, A. P.; Kurth, R.; Chapin, D. S.; Fuller, T.; Black, P. H. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 103-105; 1978.

Cell-mediated and humoral immune responses against antigens associated with primate C-type oncornaviruses were evaluated in humans by microcytotoxicity and radioimmunoprecipitation assays. Five of six women tested sequentially during pregnancy developed selective cell-mediated and humoral reactivity against baboon endogenous virus antigens. The responses peaked during the second and third trimesters. No reactivity was observed in any of 10 healthy, nonpregnant subjects (4 women and 6 men). Immune responses against cells infected with the simian sarcoma virus-simian sarcoma-associated virus complex were observed repeatedly in 3/14 nonpregnant and 1/6 pregnant individuals. (15 refs)

78-2932 **Cell-mediated and Humoral Responses to Simian Oncornaviruses Antigens in Humans with Graft.** (Eng) Thiry, L. (Institut Pasteur du Brabant, Brussels, Belgium); Sprecher-Goldberger, S.; Vereerstraeten, P.; Bossens, M.; Neuray, F. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 106-110; 1978.

Cell-mediated responses to simian oncornavirus antigens were studied in 69 pregnant and 49 nonpregnant women. Specific lymphocyte stimulation by Mason-Pfizer (M-P) virus or baboon C-type virus antigens was found in 25%-65% of the pregnant women, the frequency being highest in those with a history of 5-10 pregnancies. Only 5%-13% of the nonpregnant women had lymphocytes responding to one of these two viruses, and only 2%-4% in either group had lymphocytes responding to simian sarcoma virus. From 27% to 30% of the women had antibodies to M-P virus, and the frequency did not alter with pregnancy. Among patients with chronic glomerulonephritis, 8/14 had M-P virus antibodies before kidney transplantation and 16/19 had antibodies posttransplantation. Repeated blood transfusions or some other event related to grafting may be the source of M-P virus antigenic stimulation. (4 refs)

78-2933 **Epstein-Barr Virus-Immune Complexes in Postmortem Kidneys from African Patients with Burkitt's Lymphoma and American Patients With and Without Lymphoma.** (Eng) Sutherland, J. C. (Dept. Pathology, Univ. Maryland, 22 S. Greene St., Baltimore, MD, 21201); Olweny, C. L.; Levine, P. H.; Mardiney, M. R. *J Natl Cancer Inst* 60(5): 941-946; 1978.

Postmortem kidneys from 14 African patients with lymphoma were examined by immunofluorescence (IF) for immunoglobulin (Ig), C3, and, along with 59 kidneys from American patients, for Epstein-Barr virus (EBV)-specific antigen (Ag). Of the American patients, 21 had leukemia, 11 lymphoma, 14 solid tumors, and 13 non-tumor-related illnesses. Both Ig and C3 were detected in a nonlinear pattern in the glomeruli of two kidneys from African patients, one from a patient with Burkitt's lymphoma (BL) and one from a patient with Hodgkin's disease (HD). Ig alone was detected in the glomeruli of three kidneys from BL patients, but two kidneys from BL patients and one from a patient with reticulum cell sarcoma were negative for both Ig and C3. Six kidneys from BL patients could not be evaluated by IF for Ig and C3. EBV-specific Ag was detected in one kidney from a BL patient and in one kidney from an HD patient, both Africans; this Ag was not detected in any of the other African kidneys or in any of the American kidneys. The kidneys from 8 African patients (7 with BL and 1 with HD) and 11 American patients (7 with tumors and 4 without) were homogenized. Viral capsid antigen (VCA) and early antigen antibodies (Ab) were detected in the acid eluates of 4 African kidneys, and VCA Ab alone was detected in 6 American kidneys (3 with tumors and 3 without); none of these kidneys had demonstrable EBV Ag on their glomerular basement membrane (GBM). These findings, which provide evidence for the deposition of EBV Ag-Ab complexes on the GBM of two African kidneys (1 BL and 1 HD), indicated that EBV-related immune complexes may be important in diseases other than BL. (19 refs)

78-2934 **An Immunological Basis for Inhibition of Transformation of Human Lymphocytes by EB Virus.** (Eng) Moss, D. J. (Queensland Inst. Medical Res., Brisbane, Australia); Scott, W.; Pope, J. H. *Nature* 268(5622): 735-736; 1977.

The inhibition of Epstein-Barr virus (EBV)-induced transformation of human lymphocytes was investigated. Inhibition was noted when EBV-infected lymphocytes of three donors with antibody to EBV were cocultivated with fibroblasts from the skin of two adult donors, but not when they were cocultivated with fibroblasts from four fetuses. Inhibition of transformation was not demonstrated with lymphocytes from a donor with antibody to EBV. Nonadherent mononuclear cell populations from 14 donors (10 men, 4 women, aged 5-46 yr) were infected with 0.1 TD50 of QIMR-WIL strain EBV. Nonadherent lymphocytes, with or without antibody to EBV, were uniformly transformed when cultivated alone or with fetal fibroblasts. With adult fibroblasts, however, transformation occurred with lymphocytes from the eight donors without EBV antibody but with none of the five lymphocytes from the six donors with antibody to EBV. Transformation was not dependent on virus dose or cell concentration. Further studies showed that inhibition was dependent on previous EBV infection of the lymphocyte donor, but not of the donor of the adult fibroblast donor. It is concluded that an immunological system capable of inhibiting lymphocyte transformation by EBV is inherent in the mononuclear cells from persons with previous EBV infection. (14 refs)

78-2935 **Factors Affecting Serum IgA Antibody to Epstein-Barr Viral Capsid Antigens in Nasopharyngeal Carcinoma.** (Eng) Ho, H. C. (M. and H. Dept. Inst. Radiology and Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong); Ng, M. H.; Kwan, H. C. *Br J Cancer* 37(3): 356-362; 1978.

Horizontal and longitudinal studies were performed with nasopharyngeal carcinoma (NPC) patients before and after radiotherapy (RT) and with their family members, who have a higher risk of this disease than family members of patients with other cancers. Sera or plasma were obtained from Chinese NPC patients, their apparently healthy family members, and Tunisian and Caucasian NPC patients. Regardless of the ethnic origin of the patient, NPC appeared to stimulate the production of IgA antibodies against viral capsid antigen (VCA). There was, however, a wider scatter in IgA anti-VCA titers than in titers of IgG reactivity. The IgA anti-VCA antibodies were detected at high frequency and titers in sera from NPC patients but only rarely in sera from control subjects. Most relapse-free survivors tested 1-12 yr after RT sustained a detectable level of IgA anti-VCA. Serum titers of IgA anti-VCA remained relatively unchanged in individual NPC patients after RT, regardless of the course of the disease. Furthermore, these antibodies were detected in serum from one individual 9 mo before NPC, and the titer rose concomitantly with its clinical onset. Titers of IgA anti-VCA in multiple serum specimens from individual NPC patients and in sera from different NPC patients did not correlate with titers of IgG anti-VCA or serum IgA protein concentrations. Apparently healthy persons with detectable IgA anti-VCA tended to aggregate in families of NPC patients. The distribution of siblings of these families showed the binomial distribution expected for an autosomal recessive trait, implying the involvement of an autosomal recessive gene in the IgA anti-VCA response. (14 refs)

78-2936 **Evidence for a Specific Immunological Response Against Reverse Transcriptase (RT) as Evidenced by Natural Ig Found on the Membrane of Leukocytes from Patients with Leukemia.** (Eng) Saxinger, W. C. (Lab. Tumor Cell Biology, NCI, Bethesda, MD, 20014); Jacquemin, P. C.; Gallo, R. C. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Disease, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York Elsevier/North Holland Biomedical Press): 503 pp.; 466-470; 1978.

Purified IgG was extracted from the blood cells of leukemic patients and tested for immunological responsiveness against the reverse transcriptases (RTs) of various C-type RNA tumor viruses. The amount of antibody required for 50% inhibition of purified RT was investigated using antibody preparations from two patients with CML and one with AML. With the two CML patients, the most neutralized enzyme was feline leukemia virus RT; however, the antibodies from the second CML patient showed greater cross-reactivity with the RT's from other mammalian oncornaviruses than did the antibodies from the first patient. The AML patient's antibodies reacted only with simian sarcoma virus and human HL23 virus RT. The amount of IgG required for neutralization of the viral RTs was 10 to 100 times less than the amount necessary with laboratory generated hyperimmune sera. In a preliminary survey of membrane-associated anti-RT IgG, 5 μg of IgG were obtained from patients with CML, AML, AMML, and lymphoid leukemias and from normal buffy-coated cells and bone marrow cells and compared for RT neutralization. A total of 10/15 samples from CML patients resulted in a mean neutralization of 65% of the RT; 9/19 AML and AMML samples produced 40% neutralization; 3/13 lymphoid leukemia samples produced 33% neutralization; and with the buffy-coated and normal marrow cells, 7/23 samples produced 55% inhibition. The presence of natural antibodies that neutralize purified viral RT provides new evidence for the presence of viral information in association with some human leukemias. (18 refs)

78-2937 **Cross-linked Surface Ig Attaches to Actin.** (Eng) Flanagan, J. (Medical Res. Council, Lab. Molecular Biology, Hills Rd., Cambridge, England); Koch, G. L. *Nature* 273(5660): 278-281; 1978.

Studies were conducted to determine whether surface immunoglobulin (Ig) was attached to actin isolated from lymphocytes and P3 myeloma cells. The surface Ig was marked by reaction with ^{125}I-labeled rabbit antimouse Ig antibody (RaM Ig), either as the F(ab)$_2$ or Fab fragments. Actin was separated from other cell components by the myosin affinity technique. The amount of binding between actin and surface Ig was very low under non-cross-linking conditions but the binding increased after the application of capping conditions. Conditions that prevented capping but not patching did not preclude the attachment of actin, suggesting that the crucial step in the attachment was the cross-linking of the surface Ig. The possibility that the cross-linked surface Ig became attached to the actin after cell lysis was examined by carrying out the lysis and myosin extraction over a 20-fold dilution of cells. The proportion of surface Ig bound to the myosin remained constant, indicating that the attachment of the cross-linked surface Ig to the actin filaments took place in the intact cell. It is postulated that the cross-linking leads to attachment because of its effect on a preexisting equilibrium between surface components and microfilaments. (10 refs)

See also:
*(Rev.): 78-2444.
*(Chem.): 78-2468, 78-2469, 78-2470, 78-2472, 78-2524, 78-2596, 78-2719.
*(Viral): 78-2784, 78-2789, 78-2793, 78-2796, 78-2797, 78-2798, 78-2804, 78-2808, 78-2810, 78-2811, 78-2816, 78-2820, 78-2825, 78-2839, 78-2846, 78-2864, 78-2868, 78-2869, 78-2870, 78-2871, 78-2890, 78-2893, 78-2898, 78-2899, 78-2900, 78-2903, 78-2904, 78-2905.
*(Path.): 78-2938, 78-2946.

78-2938 **Lacunar Cells of Nodular Sclerosing Hodgkin's Disease: An Ultrastructural and Immunohistologic Study.** (Eng.) Anagnostou, D. (Evangelismos Hosp., Athens, Greece) Parker, J. W.; Taylor, C. R.; Tindle, B. H.; Lukes, R. J. *Cancer* 39(3): 1032-1043; 1977.

Tissues from 2 patients with nodular sclerosing Hodgkin's disease were studied by light and electron microscopy in conjunction with immunohistologic and cytochemical staining. The lipid in the cytoplasm of lacunar cells was probably responsible for the distinctive lacunar appearance of the cells. Marked morphologic similarities between blast cells resulting from mitogen stimulation of lymphocytes in vitro, immunoblasts in reactive lymphoid tissues, and mononuclear Hodgkin's cells in Hodgkin's disease suggested that all three cell types may result from lymphocyte transformation. There was an apparent developmental sequence from lymphocyte to transformed lymphocyte to the abnormal mononuclear Hodgkin's cell, with further progression (through increasing size and nuclear lobulation) to the lacunar cell or, alternatively, to the diagnostic Reed-Sternberg cell. This proposed sequence was supported by immunoperoxidase studies that demonstrated cytoplasmic immunoglobulin in mononuclear Hodgkin's cells, lacunar cells, and Reed-Sternberg cells. The relationship between these cells was further supported by the findings of both κ and λ chains, a pattern not seen in reactive transformed lymphocytes. (39 refs.)

78-2939 **Familial Lymphoma.** (Eng) Fradis, M. (Dept. Otolaryngology, Rothschild Univ. Hosp., Aba Khoushy Sch. Medicine, Haifa, Israel); Podoshin, L.; Zismann, D. *Ear Nose Throat J* 57(7): 310-314; 1978.

Three families in which lymphoma of the head and neck region was found in the mother and one of her children are reported. The mothers/children (2 girls, 1 boy) were aged 40/12, 40/15, and 57/15 yr, respectively. The time lapse between discovery of the lymphomas in the mother and child was 8, 2, and 4 yr, precluding a viral etiology. The most likely explanation is a genetic one, even though no chromosome abnormalities were found. (20 refs)

78-2940 **Studies of Japanese Burkitt's Lymphoma. Part 1. Establishment of a Japanese Burkitt's Lymphoma Cell Line.** (Jpn) Kishimoto, H. (Dept. Internal Medicine, Okayama Univ. Medical Sch., Okayama, Japan). *Jpn J Clin Hematol* 19(2): 93-99; 1978.

A new cell line (JBL) was established from the pleural effusion of a 29-yr-old Japanese woman with Burkitt's lymphoma. She was Epstein-Barr virus (EBV)-seropositive, but the JBL cells and the lymphoma cells were EBV genome-free on repeated testing. Most cells from both sources had monoclonal surface IgM with λ light chains. The JBL line grew in suspension with a doubling time of 30 hr, and it consisted of primitive lymphoid cells with large nuclei, prominent nucleoli, and a vacuolated cytoplasm. The JBL cells had a pseudodiploid karyotype with a large subtelocentric marker involving chromosome 11. They lacked the chromosome 14 translocation characteristic of African Burkitt's lymphoma. (20 refs)

78-2941 **Ultrastructural Study of 28 Cases of Ewing's Sarcoma: Typical and Atypical Forms.** (Eng) Llombart-Bosch, A. (Departamento de Patologia, Facultad de Medicina, Paseo al Mar, 15, Valencia, 10, Spain); Blache, R.; Peydro-Olaya, A. *Cancer* 41(4): 1362-1373; 1978.

The results of an ultrastructural study of 28 cases of Ewing's sarcoma are presented. These tumors were diagnosed in 22 females and 6 males, aged 1-25 yr. Morphological diversity within the group allowed the differentiation of a conventional form of the disease (18 women, 3 men), and an atypical variant form (4 women, 3 men). The former was characterized by blastemic cells and secondary degenerative cells, and the structure of the latter had either an immature mesenchymal or histiocytic appearance. The atypical variant was characterized by variability in size and a grooved nucleus that contained prominent nucleoli. The amount of glycogen in the cytoplasm varied from cell to cell, and a comparison with the conventional form revealed more highly differentiated structures in the variant. The variant cells also contained cytoplasmic filaments, mitochondrial complexes, desmosomiclike junctions, and profiles of endoplasmic reticulum. There were no transitional forms between the conventional and the atypical variants. No clinical or anatomical differences could be established between either variant of Ewing's sarcoma. Because of the varying amounts of glycogen in these Ewing's sarcoma cells and the presence of glycogen in undifferentiated mesenchymal tumors and malignant hemoblastosis cells, it is suggested that this commonly used diagnostic feature of Ewing's sarcoma is of restricted value. (43 refs)

78-2942 **Leiomyosarcoma of the Scrotum: A Case Report with Electron Microscopy.** (Eng) Johnson, S. (Dept. Pathology, Univ. Texas Health Science Center, Southwestern Sch. Medicine, Dallas, TX, 75235); Rundell, M.; Platt, W. *Cancer* 41(5): 1830-1835; 1978.

The case of an 89-yr-old man with a leiomyosarcoma of the right scrotum is presented. The tumor, which measured 3 x 2.5 x 2 cm, had been present as a slowly enlarging mass for 3 mo. Light microscopy demonstrated the typical interlacing fascicles of neoplastic cells with eccentric cigar-shaped nuclei, and electron microscopy confirmed the smooth muscle origin of the tumor. This is only the fifth reported case of leiomyosarcoma confined to the scrotal wall. (19 refs)

78-2943 **Malignant Fibrous Histiocytoma and Pleomorphic Sarcoma in Association with Medullary Bone Infarcts.** (Eng) Galli, S. J. (Dept. Pathology, Massachusetts General Hosp., Boston, MA, 02114); Weintraub, H. P.; Proppe, K. H. *Cancer* 41(2): 607-619; 1978.

A malignant fibrous histiocytoma arose in the proximal tibia of a 40-yr-old man who had multiple medullary bone infarcts involving the distal femora and the tibiae. Despite amputation and chemotherapy, widespread metastases developed and the patient died 19 mo after surgery. A pleomorphic sarcoma, probably representing an anaplastic malignant fibrous histiocytoma, arose in association with a single medullary infarct in the proximal humerus of a 33-yr-old woman. She remains well without evidence of disease 5 yr after treatment by radical radiotherapy followed by shoulder disarticulation. Sarcoma arising in association with bone infarction is a rare entity. In the 16 known cases, including the present 2, the sarcomas arose in the tibia in 9, the femur in 6, and the humerus in 1. The av age of the patients was 49 yr, and 13 were men. All but two had multiple bone infarctions. Four of the patients had caisson disease, 3 apparently had hereditary bone dysplasia, 1 had sickle cell disease, and 8 had infarcts of unknown etiology. Most patients have had a rapidly fatal outcome. Thirteen of the sarcomas were fibrosarcomas or malignant fibrous histiocytomas, both rare primary bone tumors. Analysis of the published data on bone infarction-related sarcomas suggests that the risk of developing a sarcoma is greatest in infarcts with large medullary components. (30 refs)

78-2944 **Ultrastructural Observations on the Histogenesis of Alveolar Rhabdomyosarcoma.** (Eng) Churg, A. (Dept. Pathology, Univ. Chicago, 950 E. 59th St., Chicago, IL, 60615); Ringus, J. *Cancer* 41(4): 1355-1361; 1978.

An alveolar rhabdomyosarcoma (ARMS) from the left forerm of a 9-yr-old boy was studied ultrastructurally, with particular emphasis on the detection of myofilaments. The bulk f the tumor was composed of small polygonal cells containng polyribosomes, short strands of rough endoplasmic eticulum, and variable amounts of glycogen; the cells were oined by small desmosomelike structures. Similar cells, as vell as larger cells apparently derived from the smaller ones, vere observed along the septa; the larger forms developed a copious cytoplasm and occasionally produced myofilaments. Some of these cells were separated from the septa by basement membranes. Giant cells were present in the alveolar spaces; myofilament bundles were not present in these cells and they were not surrounded by a basement membrane. The septa were mostly composed of collagen and fibroblasts. These observations suggest that the ARMS was mostly epitheliallike; it may correspond to the organization of mesenchymal cells at the stage of somite differentiation. Since this tumor has several features in common with Ewing's sarcoma, it may also be a proliferation of neoplastic mesenchymal cells corresponding to those seen early in embryogenesis and showing no discernible differentiation. (19 refs)

78-2945 **Mycosis Fungoides Evolving to Myelomonocytic Leukemia.** (Eng) Lofgren, R. K. (Dept. Dermatology, Mayo Clinic and Mayo Foundation, Rochester, MN, 55901); Wiltsie, J. C.; Winkelmann, R. K. *Arch Dermatol* 114(6): 916-920; 1978.

Two cases of long-standing mycosis fungoides (MF) developing 5 and 15 yr later into acute myelomonocytic leukemia (AML) are reported. The two female patients (aged 50 and 57 yr) had been treated with topical mechlorethamine and focal radiation throughout the course of the MF. One patient had also received chlorambucil (2 mg/day for 5 mo). Five literature cases of myelogenous leukemia as the terminal event of MF are also reported. The duration of the MF was 2-3, 5, 7, 11, and 18 yr, and treatment comprised various combinations of x-rays, grenz rays, iv mechlorethamine, urethan, corticotropin, and cyclophosphamide. Chronic myelogenous, acute myeloblastic, myeloid, chronic granulocytic (or leukemoid reaction), and myeloid leukemia were the types that resulted. By depressing the immune surveillance system, the therapy for MF may participate in the development of leukemia; however, a definite relationship to therapy could not be established. The development of leukemia in the two present cases may be a complication of therapy, a manifestation of some predisposing factor common to AML and MF, or a result of the direct evolution of MF. (13 refs)

78-2946 Familial (Erythro)-Leukemia: 4 Cases of DiGuglielmo's Syndrome in Close Relatives (Meeting Abstract). (Eng) Nissenblat, M. (Johns Hopkins Oncology Center, Johns Hopkins Hosp., Baltimore, MD, 21205); Bias, W.; Borgaonkar, D.; Dixon, S. *Proc Am Assoc Cancer Res* 19: 412; 1978. (no refs)

78-2947 **Aplastic Anemia and Acute Leukemia in Two Siblings.** (Jpn) Sawa, F. (Dept. Pediatrics, Toho Univ. Sch. Medicine, Toho, Japan); Shinomiya, N.; Maruyama, S.; Nakagawa, T.; Tsukimoto, I.; Nakayama, K. *Jpn J Clin Hematol* 19(2): 180-186; 1978.

Case reports are presented for a sister and brother who developed idiopathic aplastic anemia and acute leukoblastic leukemia, respectively. The girl was 23 mo old at the time of diagnosis, the boy 7 mo old. No evidence of a preleukemic stage was found in the sister, who died of an intracranial hemorrhage due to a traffic accident within 3 yr of the diagnosis. In the course of her disease, she had three intussusception episodes that were treated sucessfully with high-pressure enemas. Although complete remission was induced in the boy with VMP therapy (vincristine, mercaptopurine, prednisone), he died of bone marrow relapse, meningeal infiltration, intracranial hemorrhage, and sepsis 1 yr after diagnosis. No blood disease or malignant neoplasms were found in the family of these siblings, and the family members had a normal karyotype. At age 4 yr, the mother had been exposed to the atomic bomb radiation in Hiroshima (2,000 meters from the hypocenter). (28 refs)

78-2948 **Breast Cancer and Acute Leukemia: Report of 24 Cases and Review of the Literature.** (Eng) Rosner, F. (Hematology Div., Queens Hosp. Center, Long Island Jewish-Hillside Medical Center, 82-68 164 St., Jamaica, NY, 11432); Carey, R. W.; Zarrabi, M. H. *Am J Hematol* 4(2): 151-172; 1978.

The association of acute leukemia and breast cancer is reviewed, and the possible etiological role of breast cancer treatment in the development of acute leukemia is examined. Twenty-four cases of breast cancer + acute leukemia and three cases of breast cancer + chronic myelocytic leukemia are briefly described, and an additional 54 cases of acute leukemia and 13 of chronic myelocytic leukemia associated with breast cancer are reviewed from the literature. The mean interval between the diagnosis of breast cancer and the occurrence of acute leukemia was 6.9 yr. In nine patients, the diseases occurred simultaneously or within 1 yr of each other. Thirteen patients received no postmastectomy radiotherapy or chemotherapy, and the occurrence of acute leukemia 1-23 yr later cannot be attributed to breast cancer therapy. Forty-seven patients received postoperative radiotherapy to the mastectomy site. Combination chemotherapy was given to eight patients, three of whom were also treated with radiotherapy. Acute leukemia is estimated to occur in patients treated for breast cancer with a sevenfold increased frequency over the expected number. This increased incidence may be due to an increased risk of a second neoplasm in patients with a primary tumor, or it may be related to the radiotherapy and/or chemotherapy administered to treat the breast cancer. Spontaneous leukemia in breast cancer patients who received neither radiation nor chemotherapy probably has a different mode of genesis than that developing in patients who received these treatments. (100 refs)

78-2949 **Lobular Carcinoma In Situ on a Breast Fibroadenoma.** (Fre) Parache, R. M. (Centre Alexis-Vautrin, 54500 Vandoeuvre, France); Poisson, P.; Barlier, C. *Arch Anat Cytol Pathol (Paris)* 26(1): 47-49; 1978.

Lobular carcinoma in situ developed within a breast fibroadenoma in a 43-yr-old woman. Less than 40 such cases have been reported in the literature. (12 refs)

78-2950 **Familial Breast Cancer: Report of a Family Pedigree.** (Eng) Armstrong, A. E. (Dept. Community Medicine, Univ. Coll. Hosp. Medical Sch., London WC1, England); Davies, J. M. *Br J Cancer* 37(2): 294-307; 1978.

Following a report of several relatives suffering from breast cancer, the occurrence of neoplasms in three generations of a large family was carefully checked. Members of 1/8 branches had a high incidence of breast cancer, with six women being affected, four of them under the age of 40. As well as early onset, these women presented other features typical of breast cancer families: bilateral breast cancer, other second primary tumors, ovarian cancer in the daughter of one affected patient, and benign breast disease in the sister of another. The chance of the young women in the fourth generation developing breast cancer must be judged very high, possibly higher than the av 30% risk of developing the disease before the age of 40 deduced previously. (16 refs)

78-2951 **Endocrinic Correlates in Familial Breast Cancer (Meeting Abstract).** (Eng) Harris, R. E. (Creighton Univ. Sch. Medicine, Omaha, NB, 68178); Fishman, J.; Fukushima, D. K.; Maloney, K.; Carmody, L.; Lynch, J. F.; Marrero, K. *Proc Am Assoc Cancer Res* 19: 411; 1978. (no refs)

78-2952 **Squamous Carcinoma of the Breast.** (Eng) Hasleton, P. S. (Dept. Pathology, Univ. Hosp. South Manchester, Manchester, England); Misch, K. A.; Vasudev, K. S.; George, D. *J Clin Pathol* 31(2): 116-124; 1978.

Two women, one aged 64 and one aged 63 yr, developed squamous cell carcinoma of the breast. In each case, no lymph node invasion was found, the nipple was histologically normal, and no other type of cancer was found in the specimen. Histological evaluation suggested that the first tumor arose in a epidermoid cyst and that the second arose from metaplastic ductal epithelium. The former tumor also had a pseudosarcomatous stroma. It was concluded that myoepithelial cells did not play a role in the histogenesis of these tumors. A literature review of similar tumors is presented. (21 refs)

78-2953 **Ultrastructural Comparison of Differentiation of Stem Cells of Murine Adenocarcinomas of Colon and Breast with Their Normal Counterparts.** (Eng) Pierce, G. B. (Dept. Pathology, Sch. Medicine, Univ. Colorado, 4200 E. Ninth St., Denver, CO 80262); Nakane, P. K.; Martinez-Hernandez, A.; Ward, J. M. *J Natl Cancer Inst* 58(5): 1329-1345; 1977.

Two F344 rats with chemically induced transplantable adenocarcinomas of the colon were given ^{3}H-thymidine pulses, and autoradiography with electron microscopes was used to compare the degrees of differentiation of the tumor and colon stem cells. The most differentiated portions of the tumor had acini composed of vacuolated, mucous, and argentaffin cells in various stages of differentiation. Vacuolated and mucous cells incorporated ^{3}H-thymidine and corresponded in degree of differentiation to that of their labeled normal counterparts in the normal colon. An exceedingly undifferentiated labeled cell was identified in the tumor and crypts; this may be an undifferentiated colon stem cell that differentiates into vacuolated and mucous stem cells and/or argentaffin cells. Stem cells of normal mammary epithelium and malignant stem cells of spontaneous mammary adenocarcinomas of C3H mice had comparable degrees of differentiation. Since normal stem cells were as undifferentiated as the least-differentiated malignant stem cells, there is no need to postulate dedifferentiation as a mechanism of carcinogenesis. (40 refs.)

78-2954 **Malignant Mixed Tumors of the Mammary Gland.** (Ger) Hager, J. (Pathologisches Institut der Universitat, Mullerstrasse 44, A-6020 Innsbruck, Austria); Lederer, B. *Zentralbl Allg Pathol* 121(6): 522-525; 1977.

A malignant mixed carcinosarcoma with mesenchymal, epithelial, chondroosteoid, and myxomatous structures was found in a 34-yr-old woman. Histological examination revealed the myogenic origin of the tumor. (18 refs.)

78-2955 **Focal Nodular Hyperplasia of the Liver and Oral Contraceptive Usage.** (Ger) Gogler, H. (Chirurgische Klinik und Poliklinik, Klinikum Charlottenburg der Freien Universitat, Spandauer Damm 130, D-1000 Berlin 19, W. Germany); Beger, H. G.; Zuschneid, W.; Bucherl, E. S.; Vogel, M. *Chirurg* 49(3): 172-179; 1978.

Benign liver tumors occurred in three young women who had taken oral contraceptives for 6, 7, and 16 yr, respectively. Histologically, focal nodular hyperplasia was found in these women. Pain in the upper abdomen and general weakness were the main symptoms. Selective angiography is the most valuable diagnostic procedure. Fifty-eight cases from the literature are also reported. (15 refs)

78-2956 **Hepatocarcinoma and Oral Contraceptives (Letter to Editor).** (Eng) Trias, R. (Pathology Dept., Hospital de la Santa Creu i Sant Pau, Autonomous Univ., Barcelona, Spain); Rius, X.; Autonell, J.; Algaba, F. *Lancet* 1(8086): 821; 1978.

A tumor showing the classical features of hepatocarcinoma was removed from a 28-yr-old woman who had been taking ethinylestradiol (50 μg) and norgestrel (0.5 mg) for 13 mo for menstrual disorders. Connective tissue and vascular lesions of this type are associated with focal nodular hyperplasia, the incidence of which is increasing in women on oral contraceptives. (6 refs)

78-2957 **Liver Cell Carcinoma Associated with Oral Contraceptives.** (Fre) Leclere, J. (Service d'Endocrinologie et Medecine E, C.H.U. de Nancy, Hopital de Brabois, F 54500 Vandoeuvre-les-Nancy, France); Meot, B.; Marchal, F.; Mizrahi, R.; Collard, F.; Rauber, G.; Hartemann, P. *Ann Endocrinol (Paris)* 38(6): 361-362; 1977.

A malignant hepatic tumor occurred in a 42-yr-old woman who had been taking oral contraceptives for 10 yr. Histologically, it was a well-differentiated diffuse carcinoma. The presence of benign and malignant tumor tissue in the livers of women on steroid contraceptives supports the theory that hormones have a role in the development of hepatic lesions. (4 refs)

78-2958 **Endometrial Hyperplasias and Their Relationship to Endometrial Carcinoma.** (Ger) Ritzmann, H. (Universitats-Frauenklinik, D-7800 Freiburg im Breisgau, W. Germany); Hillemanns, H. G. *Arch Gynaekol* 223(4): 345-355; 1977.

Hyperplastic alterations were studied in the peripheral endometrium of 196 women who underwent hysterectomy for uterine carcinoma and in 208 control women of the same age who underwent hysterectomy for diseases other than carcinoma. The av age of the carcinoma patients who had adenomatous hyperplasia (HP) of the peripheral endometrium was 52.2 yr, that of all carcinoma patients was 61.9 yr, and that of the carcinoma patients with glandular cystic HP was 60.8 yr. The incidence of adenomatous HP of the peripheral endometrium was 38/196, that of glandular cystic HP 35/198. Cyclic changes or slight endometrial proliferation was seen in 47 cases, atrophy in 76. The incidence of adenomatous HP was 31.5% in carcinoma patients and 4.5% in controls aged 45-49 yr, vs 26.4% and 1.6%, respectively, in those aged 50-54 yr, 25% and 0% in those aged 55-59 yr, 13.4% and 0% in those aged 60-65 yr, and 7.7% and 3.8% in those aged >65 yr. The incidence of glandular cystic HP was 10.5% in carcinoma patients and 6.1% in controls aged 45-49 yr, 11.7% and 4.9%, respectively, in those aged 50-54 yr, 15.9% and 0% in those aged 55-59 yr, 25% and 16.1% in those aged 60-65 yr, and 20.5% and 3.8% in those aged >65 yr. The simultaneous occurrence of adenomatous HP and endometrial carcinoma suggests that this form of HP is precancerous. Before menopause, glandular cystic HP does not seem to play any role in the development of endometrial carcinoma. The cause of the high frequency of glandular cystic HP in postmenopausal women is unknown. (36 refs)

78-2959 **A Benign Serous Ovarian Cystadenoma Studied by Chromosome and Quantitative DNA Analysis.** (Eng) Wager, J. (Dept. Gynecology, Huddinge Hosp., Stockholm, Sweden); Granberg, I.; Vass, L.; Auer, G. *Acta Cytol (Baltimore)* 21(6): 774-776; 1977.

Chromosome and quantitative DNA analyses were performed on a benign serous ovarian cystadenoma removed from a 78-yr-old woman with a 3-yr history of abdominal enlargement. Only a few metaphases were noted in the primary tumor and ascitic fluid, and all were diploid. Cells from the cystic fluid, however, contained 50% diploid, 47% triploid, and 3% hectaploid metaphases. DNA analyses of cells taken from the tumor wall or cystic fluid showed 94% with diploid DNA values and 6% with tetraploid values. In cells from the cystic fluid, 60% had DNA values in the diploid region, 36% in the triploid region, and 4% in the hectaploid region. Karyotyping of cells in the cystic fluid indicated that of four near triploid metaphases examined, at least one large submedian marker chromosome was found. Cytology slides from the cystic fluid showed large atypical groups of cells with marked nucleoli. It is suggested that the triploid cells in the cystic fluid represent an abnormal population, possibly a developing step in carcinogenesis. (2 refs)

78-2960 **Incidence of Subclinical Metastasis in Stage I and II Ovarian Carcinoma.** (Eng) Piver, M. S. (Dept. Gynecologic Oncology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263); Barlow, J. J; Lele, S. B. *Obstet Gynecol* 52(1): 100-104; 1978.

The incidence of unsuspected, subclinical metastasis to the diaphragm, retroperitoneal lymph nodes, and omentum and of positive peritoneal cytology was determined in 31 consecutive patients referred to Roswell Park Memorial Institute (Buffalo, New York) from 1975 to 1977, 5 additional patients referred recently, and cases from the literature, all with Stage I or II ovarian carcinoma. Of the women with presumed Stage I ovarian cancer, 11.3% were found to have diaphragmatic metastases, 13.3% had aortic lymph node metastases, 8.1% had pelvic lymph node metastases, 3.2% had omental metastases, and 32.9% had malignant peritoneal washings. In State II ovarian cancer, 23% had diaphragmatic metastases, 10.0% had aortic lymph node metastases 0% had omental metastases, and 12.5% had malignant peritoneal washings. These are minimal figures for subclinical metastasis, especially in those cases in which only selected aortic and pelvic node biopsies were carried out. (20 refs)

78-2961 **Morphology and DNA Content of Cultures from Normal and Dysplastic Epithelium, Carcinoma In Situ, and Carcinoma of the Cervix Uteri.** (Ger) Ebeling, K. (Zentralinstitut fur Krebsforschung, Akademie der Wissenschaften der DDR, Lindenberger Weg 80, DDR-1115 Berlin-Buch, E. Germany); Krug, H.; Bilek, K. *Arch Geschwulstforsch* 48(1): 17-34; 1978.

The amount of DNA in cell nuclei was studied cytophotometrically in explant cultures from human normal and dysplastic squamous epithelium, carcinoma in situ, and squamous epithelial carcinoma of the uterine cervix. The mean DNA values, expressed in working units (WU; product of the nucleus surface x extinction), were 39.32 WU in dysplastic epithelium, 33.42 WU in carcinoma in situ, and 38.46 WU in squamous epithelial carcinoma, values significantly higher ($p < 0.05$) than that for normal squamous epithelium (25.01 WU). The differences in DNA levels among the dysplasia, carcinoma in situ, and carcinoma were not significant. Aneuploidy, polyploidy, and a predominance of periploidy were seen in the atypical cervical epithelium. As opposed to the normal epithelial cells which grew in monolayers, cells in the atypical epithelium and carcinoma cultures lost their adhesiveness, which led to loss of the monolayer structure and to overlapping of the cells as the most distinct characteristics of atypia. (35 refs)

78-2962 **Cervical Adeno-carcinoma and Partial Hydatidiform Mole.** (Eng) Mazor, B. (Dept. Obstetrics and Gynecology, Carmel Hosp., Haifa, Israel); Bar-Am, A.; Deligdish, L. *Acta Obstet Gynecol Scand* 57(3): 273-276; 1978.

A 39-yr-old multiparous Jewish woman was found to have a polypoid cervical well-differentiated cervical adenocarcinoma. At hysterectomy 2 wk after excision of this tumor, an unsuspected partial hydatidiform mole was found. Granulation tissue with squamous metaplasia and mild dysplasia of the cervix uteri were also found. At follow-up 4.5 yr later, there was no evidence of metastases. The hormonal influence of the molar pregnancy, with its marked trophoblastic hyperplasia, could have triggered transformation of a glandular polyp into the adenocarcinoma. The dysplastic proliferation of the cervical epithelium could also have been influenced by the trophoblastic hyperactivity of the partial mole. (11 refs)

78-2963 **Morphologic Evidence for Retrovirus Production by Epithelial Cells Derived from a Human Testicular Tumor Metastasis: Brief Communication.** (Eng) Bronson, D. L. (Dept. Urologic Surgery, Univ. Minnesota Coll. Health Sciences, Box 394, Mavo Memorial Bldg., 420 Delaware St. SE, Minneapolis, MN, 55455); Ritzi, D. M.; Fraley, E. E.; Dalton, A. J. *J Natl Cancer Inst* 60(6): 1305-1308; 1978.

Ultrastructural examination of primary and subcultured epithelial cells established in vitro from an abdominal metastasis of a human testicular tumor revealed particles with the morphology of retroviruses. These structures, found only after extensive scanning of the cells, were observed budding from microvilli or the outer cell membrane and as extracellular particles. Virus particle production was stimulated by incubation of the cells in culture medium containing 5-iodo-2′-deoxyuridine and dexamethasone. (21 refs)

78-2964 **A Family with Basal Cell Nevus Syndrome and Testicular Tumors: Accidental Association? (Meeting Abstract).** (Ger) Kuske, K. (Pathologisches Institut, Kantonsspital Munsterlingen, Munsterlingen, Switzerland); Hardmeier, T. *Schweiz Med Wochenschr* 108(13): 507; 1978. (2 refs)

See also:
*(Rev.): 78-2402, 78-2405, 78-2428, 78-2448, 78-2449, 78-2450, 78-2454, 78-2457, 78-2463.
*(Chem.): 78-2471, 78-2479, 78-2481, 78-2491, 78-2495, 78-2503, 78-2508, 78-2510, 78-2513, 78-2517, 78-2519, 78-2522, 78-2527, 78-2529, 78-2533, 78-2545, 78-2548, 78-2557, 78-2558, 78-2562, 78-2567, 78-2571, 78-2581, 78-2595, 78-2600, 78-2602, 78-2611, 78-2619, 78-2623, 78-2625, 78-2632, 78-2637, 78-2644, 78-2647, 78-2649, 78-2653, 78-2660, 78-2661, 78-2662, 78-2667, 78-2672, 78-2675, 78-2677, 78-2681, 78-2685, 78-2698, 78-2699, 78-2702, 78-2711, 78-2712, 78-2724, 78-2725, 78-2726.
*(Phys.): 78-2732, 78-2735, 78-2739, 78-2748, 78-2752, 78-2753, 78-2758, 78-2759, 78-2760.
*(Viral): 78-2780, 78-2799, 78-2800, 78-2801, 78-2807, 78-2809, 78-2827, 78-2847, 78-2851, 78-2878.
*(Immun.): 78-2919.
*(Epid.-Biom.): 78-2968.

78-2965 **Mortality Among Women First Employed Before 1930 in the U. S. Radium Dial-Painting Industry. A Group Ascertained from Employment Lists.** (Eng) Polednak, A. P. (Center Human Radiobiology, Radiological and Environmental Res. Div., Argonne Natl. Lab., Argonne, IL, 60439); Stehney, A. F.; Rowland, R. E. *Am J Epidemiol* 107(3): 179-195; 1978.

To determine the possible effects of exposure to radium on the development of cancer, a cohort of 634 women who worked in the US radium dial-painting industry between 1915 and 1929 was studied. Compared with mortality rates for all US white women, mortality from all cancers (84 observed vs 43.36 expected), bone cancer (22 vs 0.3), cancer of other and unspecified sites (18 vs 2.6), cancer of the colon (10 vs 5.0) was increased significantly among the study population. The number of deaths attributed to all cancers, bone cancer, and leukemia and diseases of the blood and blood-forming organs was significantly lower among women first exposed to radium after 1924 than among those first exposed before 1924. Mortality ratios were markedly lower for the 1965-1975 time period relative to earlier time periods, and the number of deaths from leukemia and diseases of the blood was significantly greater than expected during the period before 1945. Mortality ratios for bone cancer were higher among women aged < 20 yr at exposure than among those $\geqslant 20$ yr old at exposure. Increased mortality from all causes was observed only among women with radium body-burdens $\geqslant 50\ \mu$Ci; only the number of deaths due to colon cancer was greater than the number expected (5 vs 1.72) in those with body-burdens of $< 50\ \mu$Ci. (56 refs)

78-2966 **Mortality Among Individuals Occupationally Exposed to Benzene.** (Eng) Ott, M. G. (Dow Chemical Co., Midland, MI); Townsend, J. C.; Fishbeck, W. A.; Langner, R. A. *Arch Environ Health* 33(1): 3-10; 1978.

The long-term mortality experience among 594 individuals occupationally exposed to benzene was studied using a retrospective cohort design. Excluding 53 individuals who were also exposed to arsenicals, asbestos, or vinyl chloride, no statistically significant increases over US white male mortality rates were found among the benzene workers in any cause-of-death category. When the 53 patients were included, there were higher than expected incidences of anemia (2 vs 0.2 expected) and leukemia (3 vs 0.8). The records of these five decedents were examined in detail. One of those who died of leukemia had also been exposed to nitrobenzene, bromine, and vinyl and vinylidene chloride, another had been employed previously in the manufacture of veneer, and the third had probable exposure to p-chlorophenol, ethyl chloride phenetidine, acetic anhydride, and phenacetin dus Therefore, although the observed incidence of leukemi in the study group significantly exceeded the expecte incidence, a retrospective assessment of the possibl relationship to benzene exposure is difficult. (11 refs

78-2967 **Non-occupational Exposure to Asbestos and Malignant Mesothelioma in Females.** (Eng Vianna, N. J. (Bureau Occupational Health and Chronic Disease Res., New York State Dept. Health, Empire State Plaza Tower Building, Albany, NY, 12237); Polan, A. K. *Lancet* 1(8073): 1061-1063; 1978.

The records of 52 female residents of New York State who died between 1967 and 1977 with histologically confirmed malignant mesothelioma were studied to assess the possibility that household exposure to asbestos might be a major risk factor for the disease. The subjects were all Caucasian, and their mean age at diagnosis was 60.2 yr. Six subjects had worked at asbestos-related occupations, and 10 subjects had husbands and/or fathers who worked at asbestos-related occupations. Seventeen subjects had fathers and/or husbands whose occupations were associated with pneumoconioses other than asbestosis. A significantly greater number of the mesothelioma cases had husbands or fathers who worked in asbestos-related industries than did controls who had died during the same period from causes other than cancer. The relative risk for this factor was 10. The frequency of parental cancer, especially gastrointestinal malignancy, was also significantly greater for the cases than for the controls. There were no differences between subjects and controls in race, religion, duration of married life, other illnesses requiring hospital admission, personal histories, or socioeconomic status. The data suggest that women run the risk of malignant mesothelioma not only if they work in asbestos-related industries but also if they are indirectly exposed to environmental asbestos. The possibility of a genetic predisposition to malignant mesothelioma is also suggested. (25 refs)

78-2968 **Risk of Bronchial Carcinoma in Persons with Tuberculotic Lesions.** (Ger) Trefny, J. (Forschungsinstitut fur Tuberkulose und Respirationskrankheiten, 8 Bulovka, CS-180 71 Prague, Czechoslovakia). *Z Erkr Atmungsorgane* 149(3): 313-318; 1977.

A study was made of the incidence of bronchial carcinoma n persons with tuberculotic and fibrotic (probably posttuberculotic) pulmonary lesions and in the general population 177,000) of three districts of Prague during 1965-1967 and 1970-1972. Bronchial carcinoma was found n 504 patients (447 men and 57 women), and tuberculotic or posttuberculotic lesions were found in 134 patients. Relative to the nonstandardized general population aged ≥ 15 yr, the incidence of bronchial carcinoma was 355.5/100,000 in men and 31.9/100,000 n women with tuberculotic lesions, vs 71/100,000 n men and 8.7/100,000 in women without such lesions. The corresponding incidences in the age- and sex-standardized population were 176.4/100,000 in men and 8.9/100,000 in women with tuberculotic lesions and 5.6/100,000 in men and 8.6/100,000 in women without such lesions. The difference in the incidences of bronchial carcinoma in persons with and without tuberculotic lesions remained significant even after the influence of smoking was eliminated. The autopsy findings in 94 carcinoma patients with tuberculotic lesions revealed a topological link between the two pathologies in 34% and homolateral neoplastic and tuberculotic processes in 42%. The tuberculotic lesions appeared fibrotic in 77%, and active tuberculosis was found in 3%. The findings indicate that the probability of bronchial carcinoma is at least twice as high in persons with tuberculotic or fibrotic lesions than in those without such lesions. No relationship was found between the tuberculotic lesion and the histological type of the carcinoma. (6 refs)

8-2969 **A Comparison of Lithocholic Acid Metabolism by Intestinal Microflora in Subjects of High- and Low-Risk Colon Cancer Populations (Meeting Abstract).** (Eng) Kelsey, M. I. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD); Iwang, K. K. *Digestion* 16(3): 263; 1977. (no refs)

8-2970 **Faecal Bile Acid Concentration in Patients at Increased Risk of Large Bowel Cancer (Meeting Abstract).** (Eng) Mudd, D. G. (Dept. Surgery, Queen's Univ., Belfast, Northern Ireland); McKelvey, S. T.; Elmore, D. T. *Br J Surg* 65(5): 357; 1978. (3 refs)

8-2971 **Is There an Increased Risk of Large Bowel Cancer Following Cholecystectomy? (Meeting Abstract).** (Eng) Turnbull, P. R. (Wellington Clinical Sch. Medicine, Univ. Otago, Wellington, New Zealand); Isbister, V. H. *Digestion* 16(3): 280; 1977. (2 refs)

8-2972 **Gastric Cancer Incidence: Suggestive Evidence for Persisting Effects of Experiences During 1940-1950 (World War II and Postwar) (Meeting Abstract).** (Eng) Goldsmith, J. R. (State Dept. Health, Berkeley, CA); Steinitz, R.; Wronkowski, Z. *Digestion* 16(3): 241; 1977. (no refs)

78-2973 **Prostatic Cancer: Mortality and Incidence Rates by Race and Social Class.** (Eng) Ernster, V. L. (G. W. Hooper Foundation, Univ. California, San Francisco, CA, 94143); Selvin, S.; Sacks, S. T.; Austin, D. F.; Brown, S. M.; Winkelstein, W. *Am J Epidemiol* 107(4): 311-320; 1978.

A study was conducted to determine whether the excess rates of prostatic cancer among blacks could be explained by controlling for race or social class. The mortality data consisted of 400 prostatic cancer deaths occurring in Alameda County, California, during 1968-1972; incidence data were drawn from the population-based series of cases (n = 750) for Alameda County included in the National Cancer Survey (1969-1971). Comparison of age-specific mortality and incidence rates by socioeconomic status revealed no gradient for prostatic cancer in either whites or blacks. Excepting age groups over age 80, the black excess risk held up within age-specific comparisons for every socioeconomic level for which sufficient numbers existed for calculating rates. Previous studies on the relationship between socioeconomic status and prostatic cancer are reviewed. (27 refs)

78-2974 **Does Pregnancy Protect Against Ovarian Cancer?** (Eng) Beral, V. (Epidemiological Monitoring Unit, Dept. Medical Statistics and Epidemiology, London Sch. Hygiene and Tropical Medicine, Keppel St., London WC1E 7HT, England); Fraser, P.; Chilvers, C. *Lancet* 1(8073): 1083-1087; 1978.

The relation between the reproductive histories of different populations of women and their subsequent risk of ovarian cancer was examined. When the ovarian cancer mortality ratio for each 5-yr birth cohort in England, Wales, and the US was plotted against the corresponding av completed family size, a striking negative association was seen. The trends in cohort mortality in 20 countries were inversely related to cohort fertility, and the variation in ovarian cancer mortality from one country to another was inversely related to differences in av completed family size between countries. The low rates of ovarian cancer in developing countries corresponded to the generally high fertility rates. In England and Wales, mortality rates from ovarian cancer among women aged 55-64 yr were consistently higher among single vs married women and among the upper vs the lower social classes. The reported variations in ovarian cancer rates in different religious, immigrant, and ethnic groups were also consistent with an inverse relation between the risk of ovarian cancer and family size. The relative risks of ovarian cancer in women with two and four children were calculated to be 2.0 to 1 and 3.1 to 1, respectively. (28 refs)

78-2975 **Oral Contraceptives and Breast Disease. An Epidemiological Study.** (Eng) Kelsey, J. L. (Dept. Epidemiology Public Health and Human Development, Yale Univ. Sch. Medicine, New Haven, CT, 06510); Holford, T. R.; White, C.; Mayer, E. S.; Kilty, S. E.; Acheson, R. M. *Am J Epidemiol* 107(3): 236-244; 1978.

The relationship between the use of oral contraceptives and the development of benign and malignant breast disease, cancer of the ovary, and cancer of the corpus uteri was investigated in a case-control study of 20- to 44-yr-old premenopausal women in several large Connecticut hospitals. The relative risk of breast cancer was not significantly associated with use of oral contraceptives, per se, length of use, age at first use, length of time since first use, or whether sequential or combination pills were used. The relative risk was increased among women who had used only pills containing mestranol, and it was reduced among those who had used both pills containing ethinyl estradiol and those containing mestranol. Consideration of the progestogen content of the pills did not yield any useful results. The relative risk of benign breast disease decreased as a function of length of use of sequential oral contraceptives, but there was no reduction in risk associated with the use of combination pills. The small number of patients studied with cancer of the ovary or corpus uteri did not permit any conclusions to be drawn regarding these tumors. (17 refs)

78-2976 **Familial Association of Breast/Ovarian Carcinoma.** (Eng) Lynch, H. T. (Dept. Preventive Medicine/Public Health, Creighton Univ. Sch. Medicine, Omaha, NB, 68178); Harris, R. E.; Guirgis, H. A.; Maloney, K.; Carmody, L. L.; Lynch, J. F. *Cancer* 41(4): 1543-1549; 1978.

A genetic and statistical analysis was performed on 12 pedigrees that showed familial clustering of breast and ovarian cancer. These pedigrees contained 91 women diagnosed with breast cancer (60), ovarian cancer (28), or both (3). The significant linear decline in estimates of cumulative breast ovarian cancer risk to women with a diminishing genetic relationship to probands and index cases, plus the lack of excessive site-specific cancer risk to male relatives, supported a sex-limited genetic etiology. Breast/ovarian cancer typically occurred at an early age (50.1 yr for breast cancer, 52.4 yr for ovarian cancer), another characteristic of hereditary cancer. Examples of apparent mother-to-daughter and father-to-daughter genetic transmission of proclivity for breast/ovarian carcinoma were prevalent in these pedigrees. Excluding probands and index cases, the estimated cumulative risk of breast/ovarian cancer to daughters of affected mothers was 46% between the ages of 20-80 yr, suggestin that affected mothers were transmitting a deleterious cancer-predisposing gene to approx half their daughters. Eigh sibships involving putative carrier men contained 15 femal progeny over the age of 20. Of these, six have had breast an five have had ovarian cancer. Genetic heterogeneity of transmission must be considered an etiological agent in these families. (22 refs)

78-2977 **Recent Time Trends in Breast Cancer Incidence.** (Eng) DeWaard, F. (Dept. Epidemiology, Inst. Social Medicine, Univ. Utrecht, Utrecht, Netherlands). *Prev Med* 7(2): 160-167; 1978.

Time-trends in the incidence of breast cancer are discusse based on the third volume of "Cancer Incidence in Five Continents." Analysis of the data indicated that the registere age-standardized incidence has increased in almost all countries and populations, in particular in Icelanders, Israel's Sabras (Jews born in Israel), and Hawaiian Japanese. In th Hawaiian Japanese, there is evidence that the rise in incidenc is largely confined to cohorts born after 1905 (nisei who gre up in an American environment). Under these circumstance an increase in body height occurred that set the stage for new somatotype of taller and heavier people. A combinatio of increased wt and height has been shown to increase th risk of breast cancer in various populations (notably, th Japanese) in the peri- and postmenopausal period. The hypothesis that increased height plays a part in the new epidemic among Hawaiian Japanese explains why the incidence ros cohort-wise and has shown a long latency period since migration. (13 refs)

78-2978 **Time-Trend Data of Breast Cancer in Japa Clinicopathological Findings.** (Eng) Sugano, H (Dept. Pathology, Cancer Inst., Tokyo 170, Japan); Sakamoto, G. *Prev Med* 7(2): 168-172; 1978.

The time-trend change in the clinicopathological behavior breast cancer in Japan was analyzed based on 2,965 patien mastectomized at the Cancer Institute Hospital in Tokyo between 1946 and 1976. Various clinicopathological aspec such as age, histological type, tumor size, lymph node metastasis, and survival rates did not change markedly. Howeve an increase in postmenopausal lobular carcinoma and a sligh increase in premenopausal common-type breast cancer we noted over the past 15 yr. Since dietary conditions in Japa have improved greatly during the past 15 yr, it is suggeste that these changes are probably due to the effect of diet a promotor. (10 refs)

78-2979 **Oesophageal Cancer in Indian Jews in Beersheba, Israel (Meeting Abstract).** (Eng) Ode

H. S. (Gastrointestinal Unit, Soroka Medical Center, Beersheva, Israel); Krawiec, J. *Digestion* 16(3): 266; 1977. (no refs)

78-2980 **Comparative Analysis of Incidence of Cancer of Lips, Skin, and Oral Cavity in Bulgaria.** (Bul) Dimova, I. (Cancer Res. Inst., Sofia, Bulgaria). *Onkologiia* 14(4): 174-178; 1977.

The incidence of lip and skin cancer during 1970-1974 and that of cancer of the oral cavity during 1973-1974 were studied in Bulgaria. The ratio of lip cancer to skin cancer averaged 5.7 (3.5 for men, 17.1 for women, 6.8 for urban dwellers, and 5.1 for rural dwellers). The standardized lip cancer morbidity (per 100,000 inhabitants) was 6.1 (10.4 for men, and 1.9 for women, 4.8 for urban dwellers, and 7 for rural dwellers). The standardized skin cancer morbidity was 34.6 (35.3 for men, 33 for women, 34 for urban dwellers, and 34.3 for rural dwellers). The standardized incidence of cancer of the oral cavity was 3.13 (4.01 in men, 2.26 in women, 2.88 in urban dwellers, and 3.48 in rural dwellers). The incidence of skin cancer increased with advancing age. There was no correlation between the lip cancer morbidity and mean annual solar radiation. (8 refs)

78-2981 **Leukemia Morbidity in Two Neighboring Regions of Szczecin Province, Poland.** (Pol) Rybkowski, W. (Oddzial Wewnetrzyny, Szpital u Szczecinie, ul. Sienkiewicza 4 m. 1, 71-311 Szczecin, Poland). *Wiad Lek* 31(5): 323-325; 1978.

Leukemia morbidity was studied in the adult population of two neighboring regions of Szczecin province, Poland, during 1970-1974. The morbidity (per 100,000 inhabitants) was 9.2 in region A (3.8 for acute leukemia, 1.1 for chronic granulocytic leukemia, and 4.3 for chronic lymphocytic leukemia), and 0.8 in region B. The difference is statistically significant. Leukemia morbidity in cattle was 1,758.6/10,000 in region A vs 886.6/10,000 in region B. The findings suggest a common etiology of leukemia in men and cattle. (26 refs)

78-2982 **Epidemiology of Malignant Laryngeal Neoplasms in Lodz, Poland, for the Years 1967-1974.** (Pol) Fajndt, S. (Klinika Laryngologiczna, ul. Kopcinskiego 22, 90-153 Lodz, Poland); Tronczynska, J.; Dyla, S. *Otolaryngol Pol* 31(5): 499-504; 1977.

The epidemiology of malignant laryngeal neoplasms in the city of Lodz, Poland, and in the surrounding region was examined for the years 1967-1974. Men residing in Lodz had the highest incidence, but the male incidence in the surrounding area was high and on the increase. During this period, the greatest number of new cases was found in manual workers. (10 refs)

78-2983 **Characteristic Features of Cancer Epidemiology in Kirgiz Republic, USSR.** (Rus) Saenko, A. I. (Kirgiz Res. Inst. Oncology, Frunze, USSR). *Vopr Onkol* 24(6): 104-109; 1978.

Cancer morbidity in Kirgiz Republic, USSR, is reviewed. Between 1966 and 1976 the incidence of stomach cancer, cervical carcinoma, and lip cancer decreased (from 24.5%, 14.1%, and 3.6% to 22.8%, 9.0%, and 2.6%, respectively), but the incidence of lung cancer, breast cancer, and rectal carcinoma increased from 7.3%, 4.7%, and 1.7% to 12.8%, 6.6%, and 2.4%, respectively. Lung and breast cancer mortality tended to increase, but mortality from stomach cancer and cervical carcinoma showed a tendency to decrease. (2 refs)

78-2984 **Kinetic Models of C3H Mouse Mammary Tumor Growth: Implications Regarding Tumor Cell Loss.** (Eng) Roti Roti, J. L. (Dept. Radiology, Univ. Utah, Salt Lake City, UT, 84132); Bohling, V.; Dethlefsen, L. A. *Cell Tissue Kinet* 11(1): 1-21; 1978.

Three models of tumor cell loss are described. Each model is based on a different cell-loss mechanism, and equations for the cell-cycle, cell-frequency distribution, the growth of proliferating and nonproliferating cell populations, the growth fraction, and the relative rate of volumetric growth are derived. The pulse labeling index, the mitotic index, and the labeling index as a function of time after single or multiple ^{3}H-thymidine (^{3}H-TdR) injections are simulated for each model. All three models have the same mathematical form for the volumetric growth rate. The percent labeled mitosis (PLM) curves predicted by each model for the two mammary tumor lines studied (S102F and Slow) were not appreciably different. The predicted initial labeling index and mitotic index may differ significantly among the models depending on the tumor line. The most striking difference among the models was in the predictions regarding the labeling index as a function of time after a single or a series of ^{3}H-TdR injections. (21 refs)

78-2985 **Model for the Incidence of Embryonal Cancers: Application to Retinoblastoma.** (Eng) Hethcote, H. W. (Dept. Mathematics, Univ. Iowa, Iowa City, IA, 52242); Knudson, A. G. *Proc Natl Acad Sci USA* 75(5): 2453-2457; 1978.

A mathematical model for embryonal cancer incidence was developed that relates age-specific incidence data explicitly to the number of embryonal cell divisions and to rates of somatic mutation per cell division. The Poisson distribution is used to relate incidence data to cellular events. This generalized two-mutation model, which can be used for hereditary and nonhereditary cases, fits well to age-specific incidence data for retinoblastoma. (12 refs)

78-2986 **Growth Fraction as the Major Determinant of Multicellular Tumor Spheroid Growth Rates.** (Eng) Yuhas, J. M. (Cancer Res. and Treatment Center, Univ. New Mexico, 900 Camino de Salud, N.E., Albuquerque, NM, 87131); Li, A. P. *Cancer Res* 38(6): 1528-1532; 1978.

Multicellular tumor spheroids (MTS) from seven murine solid tumor lines were studied to determine the range of their growth rates and the role of the fraction of cells in cycle in these determinations. Growth rates ranged from a low of 19 μm/day to a max of 106 μm/day. This was a far wider range of growth rates than the range observed when the cells were grown as monolayers. A ^{125}I-iodouridine deoxyribose incorporation assay indicated that the cells within the MTS that are in cycle double at a slower rate than they do in monolayers and that the depth of the dividing shell ranges from <20 to >100 μm. The depth of the dividing shell or growth fraction was highly correlated with the MTS growth rate. It is concluded that the major factor that determines the wide range of MTS growth rates is the fraction of cells that are in cycle. (10 refs)

See also:

*(Rev.): 78-2412, 78-2415, 78-2420, 78-2423, 78-2424
78-2425, 78-2426, 78-2427, 78-2429, 78-2431
78-2432, 78-2433, 78-2435, 78-2449, 78-2450
78-2451, 78-2452, 78-2453, 78-2455, 78-2456
78-2459, 78-2460, 78-2461, 78-2462, 78-2463
78-2464.

*(Chem.): 78-2465, 78-2518, 78-2520, 78-2546
78-2550, 78-2554, 78-2560, 78-2626
78-2641, 78-2695.

*(Phys.): 78-2735, 78-2736, 78-2982, 78-2759
78-2761, 78-2762, 78-2765.

*(Viral): 78-2824, 78-2825, 78-2864, 78-2878, 78-2893

*(Path.): 78-2943, 78-2948, 78-2950, 78-2951.

-2987 **A Transitional Cell Carcinoma Cell Line.** (Eng) Moore, G. E. (Dept. Surgery, Denver General osp., W. 8th Ave. and Cherokee St., Denver, CO, 80204); organ, R. T.; Quinn, L. A.; Woods, L. K. *In Vitro* 14(3): 1-306; 1978.

e establishment of a cell line, COLO 232, from a grade 3 ansitional cell carcinoma of the urinary bladder in a -yr-old Caucasian man is reported. In culture, COLO 232 d a doubling time of 48 hr and retained distinct uroepitheli- phenotypic traits. Electron microscopic examination re- aled no viruslike particles. The line also produced both car- oembryonic antigen and ACTH. COLO 232 had a modal romosome number of 58, and it retained the X and Y romosomes. Ten marker chromosomes were identified; five ditional abnormal chromosomes of unknown origin were casionally observed. Injection of culture cells into irradiat- C3H/HeJ mice resulted in tumors at the inoculation site at were morphologically similar to a differentiated bladder rcinoma. The tumor grew in soft agar with a 34% efficien- . (23 refs)

-2988 **Growth and Characteristics of Small Cell Carcinoma of the Lung In Vitro and in Nude Mice leeting Abstract).** (Eng) Gazdar, A. F. (Medical Oncology anch [DCT, NCI, NIH], Veterans Admin. Hosp., ashington, DC, 20422); Sims, H. L.; Stull, H. B. *oc Am Assoc Cancer Res* 19: 116; 1978. (no refs)

-2989 **Ganglioside and Morphological Changes in Mouse Embryo Cells with Time.** (Eng) Langen- ch, R. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical nter, Omaha, NB, 68105); Malick, L.; Kennedy, S. *Cancer tt* 4(1): 13-19; 1978.

e simultaneous glycolipid, growth, and morphological anges in C3H/10T1/2CL8 cells maintained over a 4-yr riod are reported. During this period, the saturation densi- of the cells increased threefold while the cells were passed nine times and intermittently frozen in liquid nitrogen r approx 3.5 yr. Incorporation of glucosamine-labeled gan- osides into GM_3 (N-acetylneuraminylgalactosylglu- sylceramide) increased about twofold in higher ssage cells and about threefold in MCA-TCL15, a oned chemical transformant from low-passage C3H/ T1/2CL8 cells. The complex ganglioside GD_1a, ich is postulated to function in the density-dependent gulation of cell growth, decreased with passage in the C3H cells; no detectable changes in the ganglioside composition of the MCA cells were observed during 3.5 yr. High-passage C3H cells were fusiform in shape, less flattened, and they exhibited a greater propensity for growing over one another than did low-passage cells. However, they did not exhibit the random growth and aberrant morphology of the MCA cells. The data suggest that there is a relationship among ganglioside composition, cellular morphology, and regulation of cell growth. (27 refs)

78-2990 **Altered Growth Properties of Chinese Hamster Cells Exposed to 1-Methylguanine and 7-Methylguanine.** (Eng) Trewyn, R. W. (Dept. Biochemistry, Univ. Colorado Medical Center, Denver, CO 80262); Kerr, S. J. *Cancer Res* 38(8): 2285-2289; 1978.

Primary Chinese hamster embryo cell cultures generally yield cell lines with a finite lifetime in culture. However, when early-passage cells were exposed chronically to either of two normal degradation products of transfer RNA (tRNA), 1-methylguanine or 7-methylguanine, they were converted to continuous lines with altered growth characteristics and morphology. The continuous cell lines have saturation densities two- to tenfold higher than did finite control cultures, and some have the ability to grow in soft agar. Certain cultures have the general appearance of fibroblasts, but others are more epitheliallike. Quantitative and qualitative alterations in tRNA methyltransferases are early markers for neoplastic transformation in vivo and in vitro. The activity of these enzymes in the continuous lines is elevated compared with that of finite Chinese hamster cells. Neoplastic transformation has been demonstrated for a 1-methylguanine-derived line, and both 1-methylguanine- and 7-methylguanine-treated cell lines exhibit characteristics similar to those of Chinese hamster cells transformed with benzo(a)pyrene and simian virus 40. (24 refs)

78-2991 **Regional Differences in the Incidence and Growth of Mouse Tumors Following Intradermal or Subcutaneous Inoculation.** (Eng) Auerbach, R. (Dept. Zoology, Univ. Wisconsin, Madison, WI, 53706); Morrissey, L. W.; Sidky, Y. A. *Cancer Res* 38(6): 1739-1744; 1978.

The effect of a sc or id inoculation site on the growth of various tumor types (mastocytomas, teratomas, sarcomas, lymphomas, and adenocarcinomas) was investigated in several mouse strains, including athymic or irradiated hosts. With sc and id inoculation, all tumor types, and all strains, tumor cells injected into more cranial regions of the lateral trunk showed greater tumor growth and development than similar cells injected more caudally. At low tumor doses, the incidence anteriorly was double that found posteriorly, and tumors became detectable more rapidly anteriorly; at higher cell doses, the anterior:posterior ratio of tumor wt was 4:1. These findings were not influenced by the sex of the host, the immunogenicity of the tumor, or the immunological competence of the tumor recipient. These findings suggest that "natural variability" in tumor growth could be the result of an experimental protocol and that the regional differences in tumor growth could have an important effect on chemotherapeutic and immunotherapeutic treatment. Studies of innervation, the relationship of specific nerves, and differences in neural connections in the region of a developing tumor may help to explain the present findings. (14 refs)

78-2992 **Successful Transplantation of Human Benign Breast Tumors into the Athymic Nude Mouse and Demonstration of Enhanced DNA Synthesis by Human Placental Lactogen.** (Eng) McManus, M. J. (Dept. Anatomy, Michigan State Univ., East Lansing, MI, 48824); Dombroske, S. E.; Pienkowski, M. M.; Anderson, T. J.; Mann, L. C.; Schuster, J. S.; Vollwiler, L. L.; Welsch, C. W. *Cancer Res* 38(8): 2343-2349; 1978.

Each of 10 biopsy specimens of benign human breast tumors was cut into slices (30 slices/specimen) and transplanted sc into 3 female athymic nude mice (30 mice, 10 grafts/mouse). Of the 3 mice in each of the 10 triads, 2 mice were intact and 1 mouse was ovariectomized 30 days before transplantation. Ten days after transplantation, the following sc injections were administered twice daily for 20 days: 0.9% NaCl soln to one intact and one ovariectomized mouse and 0.5 mg of human placental lactogen (HPL) in 0.9% NaCl solution to one intact mouse of each triad. On the 20th day of injections, each mouse received an ip injection of ^{3}H-thymidine and was sacrificed 1 hr later. Grafts were removed and processed for histological and autoradiographical analyses. Evaluation of DNA synthesis was obtained by counting the total number of ^{3}H-thymidine-labeled epithelial cells in six sections from each graft. All ten biopsy specimens, regardless of host hormonal treatment, were accepted and maintained by these mice. The grafts retained their original morphology. The ductal epithelium of these grafts synthesized DNA, an activity that was significantly increased by HPL. Ovariectomy did not significantly influence the mean DNA incorporation of ^{3}H-thymidine. These results indicate that HPL is a direct stimulant of DNA synthesis of the ductal epithelium of benign human breast tumors. In addition, the athymic nude mouse appears to be a promising model for the in vivo study of factors influencing the development and growth of the human breast. (24 refs)

78-2993 **Cell Population Kinetics of Fast- and Slow-Growing Transplantable Tumors Derived from Spontaneous Mammary Tumors of the DBA/2 Ha-DD Mouse.** (Eng) Pavelic, Z. P. (Dept. Experimental Therapeutics, Roswell Park Memorial Inst., New York State Dep
Health, Buffalo, NY, 14263); Porter, C. W.; Allen, L. M
Mihich, E. *Cancer Res* 38(6): 1533-1538; 1978.

The proliferation kinetics of a fast-growing spontaneo
mouse mammary tumor subline (SMT-F) and a slow-growi
spontaneous mouse mammary tumor subline (SMT-S) tum
were determined autoradiographically at two stages of tum
growth to see if differences in the growth fraction or in c
mitotic cycle times were responsible for differences in tum
growth rate. The length of the cell cycle and the growth fra
tion for SMT-F were 11.2 hr and 0.85, respectively, on d
14, and 12.1 hr and 0.78 on day 28. For SMT-S, the
parameters were 15.6 and 0.50 on day 14, and 16
hr and 0.45 on day 28. On days 14 and 28, the mito
index was 1.3% and 1.0%, respectively, for SMT
and 2.2% and 1.9% for SMT-F. The cell loss rate, c
loss factor, and cell loss were significantly higher f
SMT-F than for SMT-S. It is concluded that t
difference in the growth rates for these two tum
lines is the result of a slight prolongation of the length of t
cell cycle and a reduction in the growth fraction of SMT-
(13 refs)

78-2994 **Bile Acid and Neutral Sterol Excretion in Ca
cinoma of the Colon, Other Cancers, and Co
trol Subjects (Meeting Abstract).** (Eng) Moskovitz,
(Dept. Medicine, Norwalk Hosp., Norwalk, CT); White,
Floch, M. H. *Gastroenterology* 74(5, part 2): 1071; 1978. (
refs)

78-2995 **Acid and Neutral Sterol Excretion in C
cinoma of the Colon and Control Subje
(Meeting Abstract).** (Eng) Moskovitz, M. (Dept. Me
cine, Norwalk Hosp., Norwalk, CT); White, C.; Flo
M. H. *Clin Res* 26(3): 584A; 1978. (no refs)

78-2996 **Tubulin and Actin in Paired Nonneoplastic a
Spontaneously Transformed Neoplastic C
Lines In Vitro: Fluorescent Antibody Studies.** (Eng) Tuck
R. W. (Sidney Farber Cancer Inst., Boston, MA, 02115); Sa
ford, K. K.; Frankel, F. R. *Cell* 13(4): 629-642; 1978.

Pairs of nonneoplastic and spontaneously transformed ne
plastic cells were derived from rat, mouse, and hams
embryos. The neoplastic cells of each pair had po
ly spread cellular morphology, grew in agarose in vit
and produced invasive sarcomas in vivo; the nonn

astic cells exhibited none of these properties. The stribution of microtubules and microfilament bundles tress fibers or actin cables) was examined in five ch paired lines and in 3T3 and simian virus 40-trans- rmed 3T3 cells by indirect immunofluorescent micro- opy of fixed cells treated with rabbit antibody ainst bovine brain tubulin or guinea pig smooth muscle tin. Actin cables in all the neoplastic cells were thinner and arser than those in the paired nonneoplastic cells. These fferences were also observed in living cells with polarization icroscopy. In contrast, microtubules were similar in neo- astic and nonneoplastic cells, both in areas of thin peripher- lamellar cytoplasm that allowed a clear visualization of ie, curving microtubules and in regions of thick, central idoplasm that obscured individual microtubules. The main orphological difference between neoplastic and nonneo- astic cells was the relative amount of lamellar cytoplasm endoplasm, rather than the appearance of microtubules in ther region. Thus, the distinctive growth properties and re- acted cellular morphology of neoplastic cells did not corre- te with decreased or disorganized microtubules, but with in and sparse actin cables. (39 refs)

3-2997 **A Specific Progesterone-binding Component of Human Breast Cyst Fluid: Its Isolation and haracterization (Meeting Abstract).** (Eng) Pearlman, W. H. Dept. Pharmacology, Univ. North Carolina Sch. Medicine, hapel Hill, NC, 27514); Peng, L. H.; Mazoujian, G.; Haa- ensen, D. E.; Wells, S. A.; Kister, S. J. *J Endocrinol* 75(3): 9P-21P; 1977. (3 refs)

3-2998 **Inhibition by Retinol of ODC Induction and DNA Synthesis During Cell Cycle Progression leeting Abstract).** (Eng) Haddox, M. K. (Dept. Phar- acology, Univ. Arizona Health Sciences Center, Tucson, Z, 85724); Russell, D. H. *Fed Proc* 37(6): 1432; 1978. (no fs)

3-2999 Altered Levels of Cyclic Nucleotides, Cyclic **AMP Phosphodiesterase and Adenylyl Cyclase ctivities in Normal, Dysplastic and Neoplastic Human lammary Tissue.** (Eng) Kung, W. (Hormone Lab. and Ex- rimental Endocrinology, Dept. Gynecology, Univ. Clinic ledical Sch., 4004 Basel, Switzerland); Bechtel, E.; Geyer, ; Salokangas, A.; Preisz, J.; Huber, P.; Torhorst, J.; Jung- ann, R. A.; Talmadge, K.; Eppenberger, U. *FEBS Lett* !(1): 102-106; 1977.

yclic AMP (cAMP), cyclic guanosine monophosphate 3MP), cAMP phosphodiesterase, and adenyl cyclase levels were investigated in normal, dysplastic, and neoplastic human breast tissue obtained during mastectomy, biospy, or plastic surgery. cAMP levels were significantly higher in primary carcinoma (CA) tissue than in normal or slightly dysplastic tissue. cAMP concentrations in fibroadenomas were slightly lower than those in CAs. cAMP levels were significantly elevated in CA tissue compared to normal and simple dysplastic tissue. Phosphodiesterase concentrations were measured at 6 x 10^{-8} and 1 x 10^{-6} M cAMP. At both concentrations, phosphodiesterase activities were significantly increased in CA as opposed to normal tissue. At the lower substrate concentration, phosphodiesterase activity was significantly higher in proliferative dysplasia, CA, and fibroadenoma tissue. Adenyl cyclase activities were significantly elevated in simple proliferative dysplasia and CA tissue compared to normal tissue. When relative cellular levels were considered, there were markedly lower levels of adenyl cyclase activity in CA and lower cAMP and cGMP levels in CA and proliferative dysplasia tissue compared to normal tissue. Similarly, relative cellular phosphodiesterase activities were lower in CA and proliferative dysplasia tissue than in normal tissue. (8 refs)

78-3000 **Inhibition of Ovariectomy-induced Rat Mammary Tumor Regression by a Mammosomatotropic Tumor.** (Eng) Diamond, E. J. (Res. Inst., Hosp. Joint Diseases, Mount Sinai Sch. Medicine, New York, NY, 10035); Khan, S.; Koprak, S.; Hollander, V. P. *Cancer Res* 38(8): 2239-2245; 1978.

The growth of the transplantable rat mammary carcinoma MTW9 is stimulated by elevated serum prolactin concentrations. MTW9 grown in rats with high serum (PL) produced by coimplantation of a mammosomatotropic tumor, MtTW10 (MTW9-MtT), does not regress after ovariectomy (OVEX). MTW9 grown in rats with high serum PL produced by daily administration of perphenazine (MTW9-P) does regress after ovariectomy whether perphenazine treatment is continued (MTW9-P) or not (MTW9-PD). Experiments were designed to determine whether some hormonal abnormality in MtTW10-bearing rats might prevent OVEX-induced mammary tumor regression. The hormone environment of hosts bearing MtTW10 was simulated in rats bearing MTW9-PD. High doses of ACTH, growth hormone (GH) 17-hydroxyprogesterone caproate, and PL failed to inhibit OVEX-induced regression. A high-dose mixture of PL, ACTH, and GH administered to a limited number of rats produced extreme mammary stimulation, but it also failed to inhibit tumor regression. Implantation of MtTW10 into animals bearing MTW9-PD prevented OVEX-induced regression of mammary tumor. These data are consistent with the suggestion that OVEX-induced regression is inhibited by the secretion of a factor other than PL, GH, ACTH, although some combination of hormones present in MtTW10-bearing rats cannot be excluded. (34 refs)

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CARCINOGENESIS ABSTRACTS makes available abstracts, annotations or citations of significant carcinogenesis articles collected from the current major biomedical sources of world literature. This service is provided by the National Cancer Institute through a contract with the Franklin Research Center for preparation of the publication, under Contract No. NOI-CP-75885 with the National Cancer Institute, U.S. Department of Health, Education and Welfare. Published and distributed by the Franklin Institute Press^{SM}.

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LANGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

ABBREVIATIONS used in abstracts:

A	angstrom(s)
ACTH	adrenocorticotropic hormone
ADP	adenosine diphosphate
AMP	adenosine monophosphate
ATP	adenosine triphosphate
approx	approximately
av	average
BCG	bacillus Calmette-Guerin
bid	twice daily
C	degree(s) centigrade
cal	calorie(s)
kcal	kilocalorie(s)
cc	cubic centimeter(s)
Ci	curie(s)
mCi	millicurie(s)
μCi	microcurie(s)
cm	centimeter(s)
CNS	central nervous system
cpm	counts per minute
DNA	deoxyribonucleic acid
ED_{50}	median effective dose
EDTA	ethylenediamine tetraacetic acid
g	gram(s)
kg	kilogram(s)
mg	milligram(s)
μg	microgram(s)
Hb	hemoglobin
hr	hour(s)
ia	intra-arterial
id	intradermal
IgA	Immunoglobulin A
IgB	Immunoglobulin B
IgG	Immunoglobulin G
IgM	Immunoglobulin M
ILS	increased life span
im	intramuscular
ip	intraperitoneal
IU	International Unit(s)
iv	intravenous
Km	Michaelis constant
LD	lethal dose
LD_{50}	median lethal dose
M	molar
μM	micromolar

mOsm	milliosmolar
max	maximum
mEq	milliequivalent(s)
min	minute(s)
ml	milliliter(s)
μl	microliter(s)
mm	millimeter(s)
mo	month(s)
mol wt	molecular weight
N	normal concentration
NAD	nicotinamide adenine dinucleotide
NADH	reduced nicotinamide adenine dinucleotide
NADP	nicotinamide adenine dinucleotidephosphate
NADPH	reduced nicotinamide adenine dinucleotidephosphate
NCI	National Cancer Institute
NIH	National Institutes of Health
PAS	periodic acid-Schiff
po	orally
ppb	parts per billion
ppm	parts per million
qid	four times daily
qod	every other day
QO_2	oxygen quotient
R	roentgen
RBC	red blood cells (erythrocytes)
RNA	ribonucleic acid
rpm	revolutions per minute
sc	subcutaneous
sec	second(s)
SGOT	serum glutamic-oxaloacetic transaminase
SGPT	serum glutamic-pyruvic transaminase
soln	solution
TCD	tissue culture dose
TCD_{50}	median tissue culture dose
tid	three times daily
UV	ultraviolet
WBC	white blood cells (leukocytes)
wk	week(s)
wt	weight
X	times
yr	year(s)

REVIEW

78-3001 **Health Effects of Exposure to Low Levels of Regulated Air Pollutants. A Critical Review.** (Eng) Ferris, B. G. (Harvard Sch. Public Health, 665 Huntington Ave., Boston, MA, 02115). *J Air Pollut Control Assoc* 28(5): 482-497; 1978.

The health effects of various air pollutants are discussed and the means of control are reviewed. The present primary standards for particulate matter, sulfur oxides, carbon monoxide, nitrogen dioxide, photochemical oxidants, and hydrocarbons seem adequate to protect public health. There are insufficient and inadequate data to establish a standard for sulfur oxides at this time but the evidence to date does not seem to support the contention that the concentration of sulfur oxides in suburban atmospheres in the US plays a significant role in the production of lung cancer. (142 refs)

78-3002 **In Vitro Chemical Carcinogenesis and Co-Carcinogenesis in Human Cells Initiated by Hydrazine & Polynuclear Components of Jet Fuel (Meeting Abstract).** (Eng) Milo, G. E. (Ohio State Univ., Sch. Veterinary Medicine, Columbus, OH, 43210). *Sci Tech Aerospace Rep* 16(12): P-30; 1978. (no refs)

78-3003 **Analytical Problems in Meeting WHO Limits for PAHs in Water.** (Eng) Crathorne, B. (Water Res. Centre, Medmenham Lab., P.O. Box 16, Henley Road, Medmenham, Marlow, Buckinghamshire, SL7 2HD, England). *Proc Analyt Div Chem Soc* 15(5): 155-158; 1978.

Problems involved in analyzing drinking water for polycyclic aromatic hydrocarbons (PAH) in order to meet the World Health Organization's standard of a max 200 nanograms/liter for total PAH are discussed. Total PAH refers to the sum of the levels of six specified PAH. The choice of these PAH makes it difficult to apply alternative analytical techniques to improve the accuracy of the data. (6 refs)

78-3004 **Analytical Aspects of Polycyclic Aromatic Hydrocarbons in Food.** (Eng) Knowles, M. E. (Ministry Agriculture, Fisheries and Food, 65 Romney St., London SW1P 3RD, England). *Proc Analyt Div Chem Soc* 15(5): 153-155; 1978.

Two major analytical methods for identifying polycyclic aromatic hydrocarbons in food are reviewed. The first is the collective or total method, which does not involve the separation or identification of individual components. The second consists of various chromatographic systems for separating and identifying selected or total components of the hydrocarbon extract. (no refs)

78-3005 **The Determination of Polycyclic Aromatic Hydrocarbons.** (Eng) Roe, F. J. (4 Kings Road, Wimbledon, London, SW19 8QN, England). *Proc Analyt Div Chem Soc* 15(5): 148-153; 1978.

The carcinogenicity of polycyclic aromatic hydrocarbons is reviewed. The various topics include the historical perspective, results of animal and human epidemiological studies, chemical studies of the mechanism of carcinogenesis and identification of the ultimate carcinogen, and interpretation of cocarcinogenicity studies. (42 refs)

78-3006 **Comments on Extrapolation of Cancer Response from High Dose to Low Dose.** (Eng) Nelson, N. (Inst. Environmental Medicine, New York Univ. Medical Center, 550 First Ave., New York, NY, 10016). *Environ Health Perspect* 22: 93-95; 1978.

In making judgments as to the risk of cancer from low-level exposure to carcinogens, it is generally necessary to base these judgments on high-dose or high-incidence data from laboratory or epidemiological studies. The use of laboratory animal models for estimates of carcinogenic response that are meaningful for humans can be of value in some of the simpler situations, such as those involving high-dose, direct-acting agents (eg, ionizing radiation or alkylating chemicals). However, laboratory data become progressively less reliable as the situations increase in complexity, involving extrapolation from one species to another, agents requiring activation, activation in one organ with production of tumors in another, and coacting enzymes with stimulatory or inhibitory influences. Curves relating cancer response to dose appear to exhibit a quasi-threshold, although closer inspection usually shows them to be lines with a lower slope starting from the origin then moving up exponentially. Numerous dose-response models have also been devised, although these models have restricted practical application since the basic data are rarely available. It appears that at present, the most confident estimations of the quantitative impact of air pollution on human cancer will come from epidemiological data that, in some instances, may be reinforced by data on laboratory animals. (11 refs)

78-3007 **Dose-Effect Relationships in Carcinogenesis and the Matter of Threshold of Carcinogenesis.** (Eng) Jones, H. (Medical Physics and Physiology, Donner Lab., Univ. California, Berkeley, CA, 94720). *Environ Health Perspect* 22: 171-172; 1978.

The relationships between the degree of exposure to a carcinogen and both the risk that neoplasms will occur and the time of their appearance serve as bases for an explanation of the nature of carcinogenesis. A model constructed from quantitative expressions of these relationships implies that neoplasms resulting from a single exposure to a carcinogen should be closely distributed in time, producing a pulse of cases and then being essentially without effect. The stages in the development of cancer appear to be as follows: (1) changes affecting cellular functions are induced by carcinogens and involve alterations of DNA or RNA that are transmissible to daughter cells; (2) interactions occur between clones of altered cells, and these are responsible for the relationship between dose and time of tumor appearance; (3) organ changes produced by these cellular alterations may reach a level at which they cause failure of the tissue-organizing systems that normally prevent the development of neoplasms; (4) the cancer-suppressing mechanism of the entire body appears to fail, allowing extension of a neoplasm to threaten the function of the body as a whole; and (5) uncontrolled growth typical of acute malignancies. (3 refs)

78-3008 Biologic Evidence for the Existence of Thresholds in Chemical Carcinogenesis. (Eng) Falk, H. L. (Office Health Hazard Assessment, Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709). *Environ Health Perspect* 22: 167-170; 1978.

Aspects of the first phases of carcinogenesis are presented as evidence for the existence of thresholds in this process. Several processes that have no relationship to carcinogenesis, such as absorption by the organism and metabolic change, have great bearing on the concept of thresholds because of their potential capacity to reduce the number of active molecules available for carcinogenesis. Additional variables that influence thresholds are inherent in the reactions between DNA and the chemicals that are effective alkylating agents, and those that affect DNA repair are especially critical. The extent to which DNA damage can be repaired depends on the site of alkylation, and DNA repair of succeeding alkylations is less effective. Since, at least in mice, there are levels of carcinogen exposure that will produce only dormant, initiated cells unless adequate promotion is supplied, a promoter must lower the threshold for the production of observabl tumors. The existence of thresholds in carcinogenesis is als supported by the interactions between carcinogens and structurally related chemicals, which in experimental animals depend on the dose and type of treatment. Thresholds for carcinogenesis may be further influenced by inhibitory processe such as metabolic detoxification and factors such as nutrition intercurrent disease, and exposure to other chemicals. Th fact that a number of carcinogenic molecules in low concentrations are essential constituents of the mammalian systen throughout life without causing cancer is more evidence fo the reality of thresholds in carcinogenesis. (13 refs)

78-3009 **Extrapolation of Carcinogenesis Data.** (Eng Lepkowski, W. (1620 Wainright Drive, Reston VA, 22090). *Environ Health Perspect* 22: 173-181; 1978.

The state of the art of biomedical research, as used to estimat the potential public health consequences of long-term huma exposure to chemical agents in the environment, was discussed at a 1976 conference in Pinehurst, North Carolina Presumptive tests in single cell organisms and mammalia cells can be used to determine whether or not a substance i carcinogenic, and animal tests can be used to quantitativel estimate the risk of carcinogenicity in humans. Problems i making extrapolations from animals to man include those o metabolic variability among and within species and the diversity in susceptibility to cancer among different human subgroups. No one species can serve as a model for the metabolism of all compounds in humans, and science has no progressed to the point of allowing assured species-to-specie extrapolations. A major point of controversy is whether tim or the latency period will nullify the carcinogenicity of chemicals in small doses. Some of the mathematical bases for ris extrapolation include the threshold hypothesis, the single-event hypothesis, and the probit and other hypotheses implying a dose-response curve that is concave upward. I would seem prudent to employ the single-event hypothesi in making risk estimates. Other points made at the conferenc were that the cancer age distribution is dose-independent bu risk is dose-proportional, that different statistical model yield markedly different extrapolations, and that low-dos linearity cannot be ruled out statistically. Thresholds and th contributions of epidemiological data to the problem of extrapolation were also discussed. (no refs)

78-3010 **Structural Analysis as a Means of Predictin Carcinogenic Potential.** (Eng) Ashby, J. (Imperial Chemical Industries Ltd., Central Toxicology Lab. Alderley Park, Cheshire, England). *Br J Cancer* 37(6) 904-923; 1978.

If structural analysis is to be used as a means of predictin carcinogenic potential, attention must be given to whic structural features are associated with carcinogenicity and which structural changes can nullify that association. A full understanding of inter- and intraseries effects will only follow from the consideration of factors such as metabolic transformations, the ultimate carcinogenic form, changes in basicity, acidity partition coefficients, and relative steric effects. Some broad structure-activity comparisons are outlined for the aromatic amines; arylamine carcinogens; 4-nitroquinoline N-oxide and its analogs; the polycyclic aromatic hydrocarbons; nitrosamines, nitrosamides, hydrazines, and azoxyalkyl compounds; alkylating agents; and other miscellaneous groups. For each class, an attempt was made to generate generalized structures associated with carcinogenicity, and the range of molecular changes that could be made to a carcinogen without loss of carcinogenic activity was identified. If structural analysis is to be used as a method of predicting carcinogenicity, certain problems must be recognized; eg, many compounds may be classed as potential carcinogens by virtue of an activation pathway in their metabolism, but only those for which such a pathway is significant in their overall metabolism would prove active. Furthermore, compounds such as saccharin and phenobarbitone, which induce tumors in animals, are not classified as carcinogens on the basis of structural analysis. (179 refs)

78-3011 **Carcinogenicity Testing of Chemical Substances.** (Slo) Slamenova, D. (Ustav experimentalnej onkologie, Slovenska akademie vied, Mlynske Nivy 59, 880 32 Bratislava, Czechoslovakia); Maskova, L.; Budayova, E. *Biologia* 33(3): 233-242; 1978.

Certain microorganisms, such as *Salmonella typhimurium*, *Escherichia coli*, and *Saccharomyces cerevisiae*, are suitable for the carcinogenicity and mutagenicity testing of chemical substances and for study of the metabolic activation of precarcinogens. (22 refs)

78-3012 **Relevance of Short-term Carcinogenicity Tests to the Study of the Carcinogenic Potential of Urban Air.** (Eng) Harnden, D. G. (Dept. Cancer Studies, Univ. Birmingham, England). *Environ Health Perspect* 22: 67-70; 1978.

A variety of short-term tests have been developed to evaluate the potential carcinogenicity of chemical compounds. These tests depend on mutagenicity, stimulation of DNA repair, induction of chromosome damage, and other actions to indicate carcinogenic potential. These assays are rapid and cheap, and some tests can be carried out using human cells. The short-term assays may have advantages in the context of air pollution since they are sensitive down to very low dose levels and can deal with complex mixtures. Combinations of such assays may be of more value than any single test. Their particular value may be a preliminary screening procedure in a tiered testing program. The early warning that such studies could give might help in reducing pollution as soon as possible and serve to prevent human morbidity. (19 refs)

78-3013 **The Value and Significance of Carcinogenic, Mutagenic, and Teratogenic Tests.** (Eng) Brusick, D. J. (Dept. Genetics, Litton Bionetics, Inc., Kensington, MD). *In: Cutaneous Toxicity.* Drill, V.A; Lazar, P.,eds. (New York: Academic Press Inc.) : pp. 189-201; 1978.

The value and reliability of in vivo and in vitro tests for determining the carcinogenicity, mutagenicity, and teratogenicity of a compound are discussed. Studies have found a positive correlation of approx 0.90 between carcinogenicity and mutagenicity and a positive correlation of approx 0.98 between noncarcinogenicity and nonmutagenicity. Reasons for the lack of complete correlation are outlined. The absorption of cutaneously applied substances, their systemic disposition, and the placental/gonadal penetration of these substances are considered. Based on these considerations, a composite test system to screen compounds for carcinogenicity, mutagenicity, and teratogenicity is proposed. The critical feature of this composite series of tests is the use of a T-strain of mice homozygous for several coat color genes. End points that can be detected and quantitated are enumerated. For each compound, the overall cost of the test system would be approx 20,000 dollars, and the test would take only 3-4 mo. (30 refs)

78-3014 **Biological Interactions of N-Nitroso Compounds: A Review.** (Eng) Olajos, E. J. (Inst. Comparative and Human Toxicology, Albany Medical Coll., Union Univ., Albany, NY, 12208). *Ecotoxicol Environ Saf* 1(2): 175-196; 1977.

The chemistry, metabolism, and biological interactions of N-nitroso compounds are reviewed. The possibility that alkylation of proteins and nucleic acids by these compounds is related to their mutagenicity or carcinogenicity is discussed. Genetic and epigenetic mechanisms by which the active forms of chemical mutagens and carcinogens react with informational molecules and proteins are summarized. The usefulness of nitrosamines and nitrosamides in mutagenicity and carcinogenicity studies is stressed. Continued work in this area may lead to an understanding of carcinogenic organotropism, mechanisms of genetically influenced susceptibility to chemically induced cancers, and initial processes involved in carcinogenesis and mutagenesis. (167 refs)

78-3015 **Toxicologic Perspectives of Chemicals Commonly Applied to Skin.** (Eng) Maibach, H. I.

(Dept. Dermatology, Univ. California Sch. Medicine, San Francisco, CA); Marzulli, F. N. *In: Cutaneous Toxicity.* Drill, V. A.; Lazar, P., eds. (New York: Academic Press, Inc.): 277 pp.; 247-264; 1977.

Toxicological tests used to evaluate chemicals commonly applied to human skin are reviewed, and the results obtained with propylene glycol are presented as an example. Unfortunately, data on the cutaneous and systemic effects of applied chemicals are not complete. More effective ways of determining the hazards of these chemicals to humans must be developed. (38 refs)

78-3016 **Metabolism of Halogenated Ethylenes.** (Eng) Leibman, K. C. (Dept. Pharmacology and Therapeutics, Univ. Florida Medical Sch., Gainesville, FL, 32610); Ortiz, E. *Environ Health Perspect* 21: 91-97; 1977.

Literature on the metabolic transformations of various chlorinated ethylenes is reviewed, and the results of studies on the products of metabolism of some of these compounds are presented. The metabolism of chlorinated ethylenes may be explained by the formation of chloroethylene epoxides as the first intermediate products. These epoxides appear to rearrange with the migration of chlorine to form chloroacetaldehydes and chloroacetyl chlorides. In experiments in which 1,1-dichloroethylene, trichloroethylene, and tetrachloroethylene were incubated with rat liver microsomal systems, monochloroacetic acid, chloral hydrate, and trichloroacetic acid, respectively, were found. Rearrangements of chloroethylene glycols formed from the epoxides by hydration may also occur, but they appear, at least in the case of 1,1-dichloroethylene, to be quantitatively less important. The unstable epoxide ring formed during the metabolism of chlorinated ethylenes may react with a variety of groups in compounds of biological interest. When these compounds are essential to cellular function the reaction may lead to alteration of cellular metabolism and subsequent cell necrosis or to carcinogenic or mutagenic events. (51 refs)

78-3017 **Mutagenic and Carcinogenic Risks Associated with Halogenated Olefins.** (Eng) Infante, P. F. (Industry-Wide Studies Branch, Div. Surveillance, Hazard Evaluations and Field Studies, Natl. Inst. Occupational Safety and Health, Center Disease Control, Dept. Health, Education and Welfare, Cincinnati, OH, 45202). *Environ Health Perspect* 21: 251-254; 1978.

The mutagenicity and carcinogenicity of vinyl chloride (VC), vinylidene chloride, trichloroethylene, perchloroethylene, and chloroprene are reviewed. Of the first four compounds, all except perchloroethylene have been shown to be mutagenic in test systems, and all have induced tumors in experimental animals. Studies with chloroprene have indicated that it causes sterility in male mice and rats. Rat studies have also indicated that concentrations ranging from 0.04 to 1.0 ppm result in a dominant lethal effect, affect sperm, cause testicular atrophy, and cause chromosomal aberrations in bone marrow cells. Human studies have indicated both an increase in chromosomal aberrations and a decrease in mobility of the sperm of exposed workers. Several studies have indicated an excess of lung and skin cancer in exposed workers, and one report confirmed a case of angiosarcoma of the liver in a worker with extensive exposure to finished polychloroprene. Studies with VC have indicated that in addition to angiosarcoma, there appears to be a dose-response relationship between exposure and the induction of mammary cancer in rats. A study of women employed in the VC industry indicated a 38% excess of breast cancer in those exposed. These findings could indicate another site of VC carcinogenesis. (29 refs)

78-3018 **Perspectives in the Investigation of Health Hazards in the Chemical Industry.** (Eng) Selikoff, I. J. (Mount Sinai Sch. Medicine, City Univ. New York, Fifth Ave. and 100th St., New York, NY, 10029). *Ecotoxicol Environ Saf* 1(3): 387-397; 1977.

The prediction in the early half of this century that the synthesis of new chemicals by the chemical industry would lead to a large increase in the number of cancers has not, for the most part, been fulfilled. Further investigations in the laboratory and in industry, as well as epidemiological studies, are necessary to determine whether or not this prediction is valid. (3 refs)

78-3019 **Benzene and Leukaemia (Letter to Editor).** (Eng) Aksoy, M. (Sect. Haematology, Dept. Internal Medicine, Instanbul Medical Sch., Capa-Istanbul, Turkey). *Lancet* 1(8061): 441; 1978.

Evidence of relationship between benzene exposure and leukemia was found in several studies. Permissible exposure to benzene should be as low as possible in view of individual susceptibility, because the relation between duration of exposure and bone marrow abnormalities varies, and because abnormalities in porphyrin metabolism and lowered WBC alkaline phosphatase activity can occur at exposure levels as low as 10 ppm. (10 refs)

78-3020 **Asbestos and Cancer.** (Fre) Bignon, J. (Service de Pneumologie, Centre Hospitalier Intercommunal, 40, avenue de Verdun, F 94000 Creteil, France); Bader, J. P. *Gastroenterol Clin Biol* 2(5): 453-457; 1978.

Literature on the carcinogenic effects of asbestos, with emphasis on cancers produced in the gastrointestinal tract as a result of ingestion of fibers via drinking water and food, is reviewed. Exposure to asbestos is not limited to the working environment and cancers related to asbestos exposure are not limited to the respiratory tract and lungs. (42 refs)

78-3021 **Malignant Mesothelioma and Its Relation to Asbestos Pollution.** (Fre) Bignon, J. (Service de Pneumologie, Centre Hospitalier Intercommunal, 40, av. de Verdun, 94010 Creteil, France); Ruffie, P. *Pollut Atmos* 19(76): 367-371; 1977.

The literature on the association of asbestos and cancers of the lung and respiratory tract is reviewed. A program has been initiated by the Commission of the European Community to determine the dose-effect relationship of asbestos to cancer. Mesothelioma was selected as the tumor to be investigated, since it is rare (1-4/1,000,000 population) and 80% of the patients have a history of asbestos exposure. Epidemiologically, mesotheliomas are more likely to occur in port or large industrial cities. The av age of death from mesotheliomas is 55-60 yr, more than 10 yr younger than the av age of death from bronchopulmonary cancer. All types of asbestos except anthophyllite appear to be responsible for mesotheliomas, and cigarette smoking does not appear to be a cofactor in tumor occurrence. Registries have already been established to collect data on asbestos and mesotheliomas in Great Britain and the Netherlands. (53 refs)

78-3022 **Colorectal Cancer--An Occupational Disease?** (Eng) Jansson, B. (Natl. Large Bowel Cancer Project, M.D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030). *Gastroenterology* 75(2): 321-323; 1978.

Environmental factors that promote colorectal cancer are reviewed. These include diet, selenium deficiency, and employment in certain areas of the textile and steel industries and in industries involving contact with asbestos. (9 refs)

78-3023 **Cancer and the Environment.** (Eng) Koch, H. (Dusseldorf Univ. Hosp. Maxillo-Facial and Plastic Surgery, Moorenstrasse 1, 4000, Dusseldorf 1, W. Germany). *Int Dent J* 27(2): 154-164; 1977.

The significance of harmful exogenous substances that promote carcinogenesis is reviewed. Approx 30 of these substances are carcinogenic in humans. They include not only chemicals (industrial, domestic exposure), but also radiation (natural, diagnostic, therapeutic). Various natural carcinogens are present in plant products. Although some human tumors may be caused by viruses, most have nonviral etiologies. (14 refs)

78-3024 **Genetic Aspects of Neoplastic Diseases.** (Pol) Zbytniewski, Z. (Zaklad Biologii, Inst. Biol. Med. MM, ul. Debinki 1a, 80-211 Gdansk, Poland); Kanclerz, A. *Wiad Lek* 31(5): 327-333; 1978.

The increased pollution of the human environment by chemical carcinogens has resulted in an increased incidence of neoplasms and mutations. Most human neoplasms are due to environmental factors. (31 refs)

78-3025 **Environmental and Genetic Determinants of Cancer.** (Eng) Fraumeni, J. F. (Environmental Epidemiology Branch, NCI, Bethesda, MD, 20014). *J Environ Pathol Toxicol* 1(2): 19-30; 1977.

Patterns of human cancer that may reflect environmental and genetic contributions are summarized. Available epidemiologic data indicate that up to 90% of human cancer is influenced by environmental stimuli. The evidence from various tumors suggests that a multifactorial process, including environmental cofactors and genetic-environmental interactions, is involved in their development. Genetic predisposition is possible even for tumors with marked geographic and temporal variations, since mutant genes often require environmental exposures for expression, as in the case of xeroderma pigmentosum, galactosemia, lactase deficiency, and phenylketonuria. Clinical observations suggest that familial tendencies may predispose to leukemia following contact with benzene, to liver cancer following hepatitis B infection, and to various cancers following radiation exposure. Except in hereditary syndromes or familial cancer, a genetic predisposition is usually too subtle for detection by ordinary clinical or epidemiologic means. Appropriate biological markers need to be developed. Preliminary findings that cultured cells from lung cancer patients have increased levels of inducible aryl hydrocarbon hydroxylase activity imply that a genetically regulated enzyme defect affects the metabolism and conversion of hydrocarbons to carcinogens. (38 refs.)

78-3026 **The Molecular Biology of the Induction of the Hepatic Mixed Function Oxidases.** (Eng) Bresnick, E. (Dept. Biochemistry, Univ. Vermont Coll. Medicine, Burlington, VT, 05401). *Pharmacol Ther [A]* 2(2): 319-335; 1978.

The biological events surrounding the induction of hepatic mixed-function oxidases are reviewed. Particular attention is focused on inhibitor studies; liver growth, RNA synthesis, and induction; chromatin activation and nuclear protein profiles; protein synthesis; receptors and interaction with inducers; and nuclear activation. (94 refs)

78-3027 **Reconstituted Mammalian Mixed-Function Oxidases: Requirements, Specificities and Other Properties.** (Eng) Lu, A. Y. (Dept. Biochemistry and Drug Metabolism, Hoffmann-La Roche Inc., Nutley, NJ, 07110); West, S. B. *Pharmacol Ther [A]* 2(2): 337-358; 1978.

The requirements, specificities, and various other properties of the reconstituted mixed-function oxidase systems of several mammalian tissues are reviewed. The liver microsomal system is described in detail, and the reconstituted systems from other tissues are briefly discussed and then compared with the liver microsomal system. (136 refs)

78-3028 **Metabolism of Chemical Blastomogenic Agents and Carcinogenesis.** (Rus) Balanski, R. M. (Lab. Chemical Carcinogens, N. N. Petrov Scientific Res. Inst. Oncology, Leningrad, USSR). *Vopr Onkol* 24(2): 100-106; 1978.

In this review, literature pertaining to the effect of inducers or inhibitors of microsomal oxidation on chemical carcinogenesis is summarized. Combined application of several polycyclic aromatic hydrocarbons can reduce the total number of induced skin, pulmonary, and liver tumors in experimental animals and it can elongate the latent period of tumor development. Preliminary exposure of animals to 7,8-benzoflavone inhibited the carcinogenic effect of dimethylbenzanthracene by 39%-90%; disulfiram completely inhibited the growth of dimethylhydrazine-induced tumors of the large intestine in mice. (76 refs)

78-3029 **Diet and Intestinal Diseases.** (Eng) Vaezzadeh, K. (Dept. Surgery, Pahlavi Univ., Shiraz, Iran); Dutz, W. *Am J Proctol* 29(2): 22-25; 1978.

The high-calorie, low-bulk processed food of industrial nations may be involved in the pathogenesis of diverticulosis, ulcerative colitis, Crohn's disease, polyps, and colorectal cancer. The high-bulk, low-calorie diet of rural Asian and African populations may prevent these pathologies, but it is responsible for volvulus of the small intestine, cecum, and sigmoid colon. (21 refs)

78-3030 **Impact of Westernization on the Nutrition of Japanese: Changes in Physique, Cancer, Longevity and Centenarians.** (Eng) Kagawa, Y. (Dept. Biochemistry, Jichi Medical Sch., Minamikawachi-machi, Tochigi-ken, 329-04, Japan). *Prev Med* 7(2): 205-217; 1978.

The effects of a westernized diet on the physique, longevity, and cancer incidence of the Japanese was investigated. Between 1950 and 1975, the intake of milk (15-fold); meat, poultry, and eggs (7.5-fold); and fat (6-fold) has increased, and the consumption of barley (2.5%), potatoes (50%), and rice (70%) has decreased. This westernization is more pronounced in the younger generation and among rich people, nonfarmers, and city dwellers. However, the quantities of western foods consumed in Japan are still much less than those consumed in the US and Europe. The quality of the nutrients is also very different: amylopectin, long chain polyunsaturated fatty acids, and indigestible polysaccharides are much more abundant in the Japanese diet. During this period, the Japanese became taller and heavier. The incidence of breast, colon, and lung cancer has increased (2- to 3-fold), but that of the stomach and uterine cancer has decreased 60% and 30%, respectively. High correlations were found between total fat intake and breast cancer mortality; the highest correlation was found between per capita pork intake and breast cancer mortality. The life expectancy has been increased an av of 12 yr for men and 14 yr for women. (32 refs)

78-3031 **Lung Cancer Etiology. Critical Appraisal.** (Eng) Burrascano, J. J. (700 Park Ave, New York, NY, 10021). *NY State J Med* 78(6): 924-934; 1978.

Factors involved in the etiology of lung cancer are reviewed. Cigarette smoking is the dominant factor, with polycyclic aromatic hydrocarbons being the major contributors to tobacco carcinogenicity. Others include industrial products, chemicals, and metals. Experimental and epidemiologic evidence shows a positive relationship between radiation and respiratory cancer. The role of viruses in these tumors is uncertain. The role of the immune system in the etiology and behavior of lung tumors is discussed. (158 refs)

78-3032 **Cancer Epidemiology. Part 11. Breast Cancer.** (Eng) Friedman, R. (Wisconsin Clinical Cancer Center, 1900 University Ave., Madison, WI, 53705); Harberg, J.; Buchanan-Davidson, D. J. *Wis Med J* 77(5): 14-15; 1978.

Risk factors, precancerous conditions, and possible causal agents of breast cancer are reviewed. The risk factors include family history of breast cancer and ages at first childbirth, menarch, and menopause. Precancerous conditions include gross cystic disease, multiple intraductal papillomas, and lobular neoplasia. Possible causal agents include dietary fat and estrogen intake. (36 refs)

78-3033 **Retinoblastoma: A Prototypic Hereditary Neoplasm.** (Eng) Knudson, A. G. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA, 19111). *Semin Oncol* 5(1): 57-65; 1978.

Data on the hereditary and nonhereditary forms of retinoblastoma are reviewed. It is estimated that 60% of all cases are nonhereditary and the remainder are hereditary. Most retinoblastoma gene carriers develop bilateral tumors; however, 5% of the carriers do not develop tumors, and some carriers have not only bilateral tumors, but also multiple tumors in one eye. The incidence of retinoblastoma is approx 5/100,000 children. It is suggested that two factors are involved in tumor development: a mutation (in germinal cells in hereditary forms or in somatic cells in nonhereditary forms) followed by some sort of stimulus (possibly another mutation). Karyotypic studies have indicated that the primary mutation in both forms appears to be associated with chromosome 13. Retinoblastoma never develops after the age of 10. This is undoubtedly related to the fact that cell division and DNA synthesis normally cease in the retinoblast cell line as differentiation proceeds. It is suggested that the tissue specific function that is altered by the retinoblastoma mutation affects DNA synthesis indirectly. (17 refs)

78-3034 **Biological Features of Tumor Progression.** (Rus) Shapot, V. S. (Cancer Res. Center, Moscow, USSR). *Vestn Akad Med Nauk SSSR* (10): 19-24; 1977.

Characteristic features of various stages of tumorigenesis are discussed with special attention to the Fould's theory. It is suggested that Fould's principle of the independent origin and development of tumor traits should be supplemented by the notion of the inequality of these traits. All tumor traits can be divided into primary (uncontrollable growth) and secondary types. The latter, in turn, can be subdivided into optional (metastatic spread, anaplasia, chromosome aberrations) and obligatory (invasive and destructive growth, systemic effect on the organism) ones. The idea that the biochemical dedifferentiation of malignant tissue (anaplasia) is, in fact, a secondary optional trait in tumorigenesis was confirmed in a series of experiments on the hormone-induced synthesis of tyrosine aminotransferase and tryptophan pyrrolase in rats with Zajdela's hepatoma. (5 refs)

78-3035 **The Urothelium: A Battleground for Oncogenesis.** (Eng) Melicow, M. M. (Uropathology Res., Columbia Univ. Coll. Physicians and Surgeons, New York, NY). *J Urol* 120(1): 43-47; 1978.

The urothelium, a term that should replace the label transitional cell epithelium, bears the brunt of attacks by various carcinogens in the urine. It is postulated that the attack is on the asymmetric unit membrane and the Golgi complex or other coding units and that it results in the destruction of the mechanism for specialization of the urothelial cell. The anaplastic cancer cell is a return to the unicellular state. Cancers in which a deficiency of the specialization function has been observed or is suspected are listed. (24 refs)

78-3036 **Problems Concerning the Morphogenesis of Colorectal Cancer.** (Rus) Pozharisskii, K. M. (N. N. Petrov Res. Inst. Oncology, USSR Ministry Public Health, Leningrad, USSR). *Arkh Patol* 40(5): 76-84; 1978.

Analysis of the literature shows that there is no reliable evidence of an association between intestinal adenomatous polyps and the morphogenesis of colorectal cancer. On the other hand, villous and glandular-villous tumors often undergo malignant transformation. The growth pattern as well as the histological, ultrastructural, histochemical, immunological, and other characteristics of these tumors are similar to those of adenocarcinomas. Therefore, villous tumors without invasion can be referred to as carcinoma in situ or superficial cancer. (80 refs)

78-3037 **The Epidemiology of Cervical Cancer.** (Eng) Barberis, L. (Direzione Sanitaria, Ospedale Maggiore di Bergamo, Bergamo, Italy); Bertocchi, C. *Minerva Ginecol* 30(4): 291-298; 1978.

Recent epidemiological and etiological studies of carcinoma of the uterine cervix are reviewed. Early sexual activity and sexual promiscuity have been shown to increase the incidence of cervical carcinoma. Barrier contraceptives and circumcision are believed to have no effect on this incidence. With respect to the latter, the lower incidence among Jewish women may be due to racial or genetic factors. The carcinogenicity of sex hormones has been proved in animals, but not in humans. Statistics show that there is a relationship between herpes simplex virus type 2 and cervical carcinoma, but an etiological role for oral contraceptives is uncertain. Microbial inflammations may be a cause of precancerous conditions or of a predisposition to cervical neoplasia. (20 refs)

78-3038 **Epidemiological Approaches to Determining Whether Herpesvirus Is the Etiological Agent of Cervical Cancer.** (Eng) Melnick, J. L. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX, 77030); Adam, E. *Prog Exp Tumor Res* 21: 49-69; 1978.

Evidence suggestive of an etiological role for herpes simplex virus type 2 (HSV-2) in cervical cancer is reviewed, and epidemiological approaches to further investigations are proposed. HSV-2 infection and cervical cancer affect women with similar sexual attributes, suggesting a venereal transmission of the virus. Immunofluorescence techniques have demonstrated the presence of herpesvirus antigens in cells exfoliated from lesions of squamous cell carcinoma of the cervix. Antigens were also reported in cells from lesions of carcinoma in situ (CIS) and lesser degrees of atypia. Infectious HSV-2 was also recovered from a cell line derived from a CIS lesion. The prevalence of HSV-2 antibodies in patients with invasive cancer varied from 31%-100%; the range in controls was 13%-67%. In CIS patients, antibodies were present in 32%-100% vs 14%-53% in controls. In subjects with dysplasia, the range was 24%-95% vs 14%-44% in controls. The relative risk of invasive carcinoma of the cervix in women with HSV-2 antibodies ranged up to 10 times that in women without antibodies. Serological data indicate that: (1) HSV-2 infection may be etiologically associated with only some cervical neoplasia cases, suggesting multiple etiologies; (2) there may be geographical differences in conditions for the sequence of infections with different types of herpesvirus; (3) antigenically different HSV-2 strains may have a different geographic distribution; and (4) the association of HSV-2 infection and cervical neoplasia is not an etiologic one. In case control studies of antibodies to nonvirion antigens, antibodies to the soluble antigen isolated from cancer cells of the lower genital tract of 95% of patients and 25% of controls reacted with HSV-2 antisera prepared in guinea pigs. Cervical cancer patients also had a greater serum reactivity to VP134 antigen than patients with breast cancer and controls. Antibodies to HSV-2-induced specific antigen were found in 91% of invasive carcinoma, 68% of CIS, and 35% of atypia patients and 9% of controls. The data support an etiological relationship of HSV-2 infection to at least some cervical neoplasias. (69 refs)

78-3039 **The Genital Herpes-Cervical Cancer Hypothesis--10 Years Later.** (Eng) Nahmias, A. J. (Dept. Pediatrics, Emory Univ. Sch. Medicine, Atlanta, GA, 30303); Sawanabori, S. *Prog Exp Tumor Res* 21: 117-139; 1978.

Studies related to the hypothesis that genital herpes simplex virus (HSV) infection is in some way associated with cervical cancer are reviewed. The various topics include the epidemiological aspects, the temporal relationship, clinicopathological aspects, HSV strain differences, the oncogenic potential of HSV, HSV markers in neoplastic cells, and seroepidemiological studies and assays for cell-mediated immunity. The results that have been obtained are extremely variable. Although this may indicate that the association between viral infection and cervical cancer is a spurious one, it may also indicate the complexity of the problem and of the methodologies employed. (120 refs)

78-3040 **Viruses and Human Cancer: The History and Current Status of ESP-1.** (Eng) Dmochowski, L. (Dept. Molecular Carcinogenesis and Virology, Univ. Texas System Cancer Center, Texas Medical Center, Houston, TX, 77030); Bowen, J. M. *Prog Exp Tumor Res* 21: 160-195; 1978.

Current knowledge of ESP-1 virus, isolated from the pleural effusion cells (ESP-1 cells) from a 5-yr-old boy with American-type Burkitt's lymphoma, is reviewed. Electron microscopy of cells from the 10th tissue culture passage revealed the presence of C-type virus particles in varying stages of development. Human leukocyte antigen (HLA) studies of the ESP-1 cells indicated that the HLA types were entirely compatible with those of the parents; ie, that the tissue culture cells contained surface antigens shared with the mother and father. Sera of 265 cancer patients and 180 normal individuals were tested by indirect immunofluorescence assays for antibodies to ESP-1 cell-associated antigens. The highest frequencies of positive reactions were observed with sera from patients with Burkitt's lymphoma (African and American), reticulum cell sarcoma, and rhabdomyosarcoma; only 2/180 normal donor sera were positive. ESP-1 virus has the typical morphology of mammalian C-type viruses, with the mean diameter of the mature virion being 100 nanometers. The buoyant density of the ESP-1 virions in isopycnic gradients is 1.14-1.17 g/cm^3; the virion RNA has a mean sedimentation value of 68S, and it separates into subunits of 28S and 35S upon heating. The virus contains a reverse transcriptase with the characteristic properties of a C-type virus enzyme. However, thus far, it has demonstrated a total lack of infectivity for other cells in tissue culture. The major identifiable antigenic determinants of ESP-1 virus are murine. (78 refs)

78-3041 **An Assessment of the Possible Role of Viruses in the Aetiology of Burkitt's Lymphoma.** (Eng) Epstein, M. A. (Dept. Pathology, Univ. Bristol Medical Sch., University Walk, Bristol BS8 1TD, England). *Prog Exp Tumor Res* 21: 72-99; 1978.

The role of Epstein-Barr virus (EBV) in the etiology of Burkitt's lymphoma (BL) is reviewed. Different techniques have demonstrated that the DNA and antigens of EBV are present in the cells of almost every African BL and in a minority of sporadic BL. Furthermore, the absence of virus particles in the cells in vivo and the integration of at least some virus DNA molecules into the host cell genome follow the pattern seen with known animal oncogenic DNA viruses in relation to the cells of the tumors they cause. African BL has long been recognized as a monoclonal disease, and the presence of the virus DNA in every tumor cell indicates that the original cell undergoing malignant transformation must have been infected at the outset. Antibody responses in African BL and some cases of sporadic BL fulfill criteria for a putative human tumor virus in being both always present and greatly raised in titer in the tumor patients compared with controls. Cell lines arising either by the exposure of lymphocytes from seronegative donors to EBV or by a two-step mechanism from the B cells of seropositive donors differ from BL-derived lines in cell morphology, growth characteristics, and karyotype. These observations indicate that changes brought about by the virus when conferring the power of unlimited proliferation on virus-free lymphocytes in vitro resemble those of malignant transformation by known oncogenic animal viruses. The finding that EBV causes fatal, malignant reticuloproliferative disease on inoculation into South American subhuman primates fulfills the last criterion for a putative human tumor virus. Although this finding does not prove that the virus plays an etiological role in BL, it does show that it can be directly carcinogenic in vivo. (186 refs)

78-3042 **Do Normal Human Cells Express RNA Tumor Virus-related Information?** (Eng) Gerard, G. F. (Inst. Molecular Virology, Saint Louis Univ. Sch. Medicine, St. Louis, MO, 63110); Green, M. *Prog Exp Tumor Res* 21: 250-269; 1978.

Normal human cells were examined for the presence of RNA tumor virus-related information, and the results are discussed along with those found in the literature. In general, attempts to detect RNA tumor virus-related components in normal human cells and tissues have been unsuccessful. Particle-associated reverse transcriptase (RT) has been detected in the cytoplasm of several kinds of human tumors, and a new DNA polymerase present at 1% of the level of DNA polymerase γ has been detected in cultured human cancer cells. Several observations are compatible with a role for RT-containing particles in specialized cellular functions. C-type particles have been visualized in normal human placenta, but attempts to detect RT have failed. Particles with some of the morphological and biochemical properties of RNA tumor viruses and enzyme activity similar to RT have been found in normal human milk. Recently, an endogenous DNA polymerase with RT-like activity has been found in human sperm. Nucleic acid hybridization and immunological studies, respectively, have produced evidence compatible with: (1) endogenous human genetic information distantly related to baboon C-type virus and (2) widespread infection with viruses that cross-react with two groups of subhuman primate C-type viruses. The relationship of putative endogenous human 'virogenes' and unidentified exogenously spread human viruses to human neoplasia is unknown. (76 refs)

78-3043 **Evidence for the Presence of RNA Tumor Virus Components in Human Leukemia.** (Eng) Gallo, R. C. (Lab. Tumor Cell Biology, Div. Cancer Treatment, NCI, NIH, Bethesda, MD, 20014); Meyskens, F. L. *Prog Exp Tumor Res* 21: 216-249; 1978.

Evidence for the involvement of RNA tumor viruses in human myelogenous leukemia is reviewed. Components related to murine and primate (woolly monkey and baboon) RNA tumor viruses have often been identified in the peripheral WBC and tissues of untreated patients with acute myelogenous leukemia and chronic myelogenous leukemia in the acute phase. These components are: (1) a reverse transcriptase with significant homology to woolly monkey (simian sarcoma virus; SiSV) and partial homology to murine (Rauscher murine leukemia virus; R-MuLV) reverse transcriptase; (2) viruslike intracellular particles that fractionate isopycnically at 1.17 g/ml in sucrose and can in some cases be converted to 1.23-g/ml cores, have a sedimentation coefficient of 550S, have a diameter of 500-1,000 A, can generate virus-related complementary DNA sequences in an endogenous reaction, and have nucleic acids with viruslike properties; (3) R-MuLV-, baboon endogenous virus (BaEV)-, and SiSV-related sequences, which have been detected in the DNA of some leukemic tissue but not in that of normal tissue; (4) viruslike particles that have demonstrated homology to SiSV and BaEV; (5) a virus, isolated several times from a single patient, that contains both SiSV- and BaEV-related components. (126 refs)

78-3044 **Transport Alterations in Virus-transformed Cells.** (Eng) Parnes, J. R. (Dept. Medicine, Harvard Medical Sch., Boston, MA, 02114); Isselbacher, K. J. *Prog Exp Tumor Res* 22: 79-122; 1978.

Studies of the changes in transport that occur with the transformation of mammalian cells are reviewed, especially those in which membrane vesicles were used. The data show that viral transformation results in the enhanced transport of specific nutrients, but it is not known whether these changes play a primary or secondary role in transformation. (164 refs)

78-3045 **Virus-Virus Cocarcinogenesis and Other Aspects of Virus Interaction.** (Rus) Mazurenko, N. P. (Cancer Res. Center, Moscow, USSR); Merekalova, Z. I.; Iakovleva, L. S.; Seniuta, N. B.; Kiselev, F. L.; Pavlovskaia, A. I.; Topol', L. Z.; Plakhov, I. V.; Lavrov, S. V.; Nikitin, A. M.; Stepina, V. N.; Zakharova, O. S. *Vestn Akad Nauk SSSR* (5): 65-73; 1978.

Various aspects of viral cocarcinogenesis and the interaction between oncogenic and infectious viruses are summarized. The phenomenon of virus-virus cocarcinogenesis was first demonstrated in 1955, when 20% of CC57Br mice (low incidence of leukemia) inoculated with smallpox virus developed leukemia. In another study, smallpox virus was isolated from primary or continuous cultures of diffuse polyposis cells from human large intestine. The ability of some infectious viruses to activate latent oncogenic viruses and induce the expression of viral proteins was further confirmed in studies of the superinfection of BALB/c embryonal fibroblast cultures infected with Friend leukemia virus (FLV) or Moloney sarcoma virus. Superinfection with Sendai virus, influenza virus or herpesvirus significantly increased the production of the leukemia and sarcoma viruses. Simultaneous cultivation of FLV with Sindbis virus inhibited production of the latter; the inhibition was probably due to the FLV-induced production of interferon. Specific interaction was also discovered between the attenuated and pathogenic variants of Marek's disease virus. (25 refs)

78-3046 **Membrane Proteins of Cells Neoplastically Transformed by Simian Virus 40.** (Eng) Schmidt-Ullrich, R. (Therapeutic Radiology Dept., Radiobiology Div., Tufts-New England Medical Center, 171 Harrison Ave., Boston, MA, 02111); Wallach, D. F. *Prog Exp Tumor Res* 22: 151-189; 1978.

Studies of membrane-protein anomalies in cells neoplastically transformed by simian virus 40 (SV40) and of the possible role of SV40-specific proteins for abnormal membrane structure and function are reviewed. The findings are consistent with the possibility that it is the insertion of the SV40-specific membrane proteins that modifies the collective thermotropic behavior of both membrane proteins and membrane lipids, although nonspecific mechanisms are also possible. (83 refs)

78-3047 **Recombination Events Between Simian Virus 40 and the Host Genome.** (Eng) Winocour, E. (Dept. Virology, Weizmann Inst. Science, Rehovot, Israel); Oren, M.; Lavi, S.; Vogel, T.; Gluzman, Y. *Miami Winter Symp* 14: 181-196; 1977.

During the replication of simian virus 40 (SV40) in monkey cells, large portions of the viral genome are replaced by covalently linked cellular DNA sequences. The passage history of three independently generated substitute SV40 populations, CVB-1-P-4, CVB-4-P8, and CVG-1-P10, is reviewed with emphasis on the isolation of a clonal CVB-1-4 variant (F161-F). The structure of the F161-F DNA was investigated by restriction endonuclease mapping and hybridization techniques. The results indicate that 61% of the total DNA sequences are of host origin and are interspersed between four repeated viral units. Preliminary data on the four host segments suggest they share common sequences and that they code for only 16,000-18,000 daltons of protein. The use of marker rescue experiments to study recombination events between endogenous and exogenous SV40 genes is also discussed. In these experiments, the DNA of marker-rescued virus was analyzed by restriction endonuclease mapping to determine whether it contained segments of both the endogenous virus (SV40 strain 777) genome of transformed cells and the exogenous superinfecting temperature-sensitive (*ts*) mutant. Virus rescue was observed only with *tsA* mutants, but the level was very low. This result is consistent with the concept that the endogenous SV40 genome is defective in the function required for initiation of viral DNA synthesis. (20 refs)

78-3048 **Cell Surface Markers.** (Eng) Cantor, H. (Harvard Medical Sch.-Farber Cancer Center, Boston, MA, 02115). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 323-326; 1978.

The importance of T cells to tumor immunity is reviewed. The specific topics include the cell-surface phenotypes of normal and leukemic T and B cells, interactions among lymphocyte sets, and characteristics of the immune response to murine sarcoma virus-induced lymphomas and leukemias. (6 refs)

78-3049 **Immunosuppressive Phenomena in Experimental Tumor Systems.** (Fre) Reme, T. X. (Dept. d'Immunologie Clinique et Experimentale, Centre Paul Lamarque, Hopital St-Eloi, 34033 Montpellier, France). *Pathol Biol (Paris)* 25(10): 735-740; 1977.

The immunosuppressive role of T lymphocytes and macrophages and/or their secretions in the growth of various spontaneous, chemically induced, or virally induced animal tumors is reviewed. Numerous studies have established the ability of T lymphocytes from tumor-bearing animals to block tumor rejection in syngeneic immunized animals. In vitro studies have demonstrated the suppressive effect of T lymphocytes from tumor-bearing animals on cell-mediated immune reactions. Splenocytes, thought to be macrophages, from rats carrying nonviral tumors do not respond to stimulation by phytohemagglutinin (PHA). Other studies have investigated the immunosuppressive activity of serum from tumor hosts without determining the exact nature of the complex responsible for the immunosuppression or its relation to T cells or macrophages. The mechanism of immunosuppression is discussed with respect to the production of antigens by tumor cells, whether these antigens are virus-related, and the relationship between the antigens and the reaction of suppressor cells to stimulation by PHA and other mitogens. (80 refs)

78-3050 **Etiology of Human Leukemias.** (Rus) Vorob'ev, A. I. (Dept. Hematology, Central Inst. Advanced Training Physicians, Moscow, USSR); Brilliant, M. D. *Ter Arkh* 50(7): 3-10; 1978.

Problems related to the etiology of human leukemias are discussed. External factors that may lead to the development of chronic and acute myeloid leukemia include ionizing radiation and chemical mutagens (including some cytostatic antitumor drugs). Severe immunodepression caused by treatment with cytostatics, total irradiation, or antilymphocyte serum increases the incidence of lymphosarcomas markedly. (76 refs)

78-3051 **Some Thoughts on Moles and Melanomas.** (Eng) Whiting, D. A. (Section Dermatology, Univ. Texas Health Science Center at Dallas, 5323 Harry Hines Blvd., Dallas, TX, 75235). *Int J Dermatol* 17(6): 485-487; 1978.

Moles on the trunk may be more susceptible to malignant change than moles elsewhere. In one study, a correlation was found between melanoma on the trunk and the presence of nevus cells in the lesion. Most of the nevus cell-containing melanomas were presumed to have developed in junctional nevi, which have a predilection for the trunk. Speculations are made on the role of UV light in the development of melanomas. (5 refs)

78-3052 **The Time Factor in Carcinogenesis. The 1977 Sievert Lecture.** (Eng) Mayneord, W. V. (7 Downs Way Close, Tadworth, Surrey, KT20 5DR, England). *Health Phys* 34(4): 297-309; 1978.

The time factor in radiation carcinogenesis is discussed in relation to Sievert's concept that the latent period depends on the radiosensitivity of the cell and the speed of reconstitution of essential cell ingredients. Dose-response curves for chemical carcinogens and radiation are compared, and examples are given of the lognormal distribution of time delay and cancer. Experiments with low linear-energy-transfer radiation suggest that the dose response of tumor-resistant tissue is curvilinear or sigmoid, but that of sensitive tissue is linear. Sensitive tissue corresponds to a low median dose (or time) and a large variance (ie, population heterogeneity). Because a probability density function can be assumed for the latent period, a combination of cell survival and probable tumor incidence can be used to predict the peak response. The possibility that the latent period may be reduced by noncarcinogenic promotors or viruses is noted. (23 refs)

78-3053 **A Current Literature Report on the Carcinogenic Properties of Ionizing and Non-ionizing Radiation. I. Optical Radiation.** (Eng) Cunningham-Dunlop, S. (Franklin Inst. Res. Lab., Science Information Services, Philadelphia, PA, 19103); Kleinstein, B. H. (Washington: U.S. Government Printing Office): DHEW (NIOSH) Publication No. 78-122, 90 pp.; 1977.

Carcinogenic hazards of occupational exposure to UV, visible, and infrared radiation are presented based on a review of the literature published since 1970. The types of cancer induced by optical radiation in man and laboratory animals, proposed radiocarcinogenic mechanisms, dose-response relationships, wavelength dependence, and synergistic effects are covered. The risk of excess radiation-induced cancer under commonly encountered conditions of occupational exposure to UV is estimated. Specific gaps in the present knowledge of radiation carcinogenesis are identified, and specific areas in need of further investigation are recommended. (137refs)

78-3054 **Histogenesis of Chemically and Radiation-induced Carcinogenesis of the Respiratory Tract (Meeting Abstract).** (Eng) Smith, D. M. (No affiliation given). *Am J Med Technol* 44(5): 491; 1978. (no refs)

78-3055 **Xeroderma Pigmentosum: Genetic and Environmental Influences in Skin Carcinogenesis.** (Eng) Cleaver, J. E. (Lab. Radiobiology, Univ. California Medical Sch., San Francisco, CA, 94143). *Int J Dermatol* 17(6): 435-444; 1978.

The biochemical, cellular, and clinical characteristics of xeroderma pigmentosum, an excision repair-defective disease, are reviewed to provide a better understanding of one kind of carcinogenesis, in which an accumulation of genetic damage is theorized to be part of the underlying mechanisms. If damage in some critical region(s) of the genome is not repaired or is repaired inaccurately before DNA replication occurs, the region may become the site of a somatic mutation. (47 refs)

78-3056 **Fetal Patterns in Adult Rat Liver Undergoing Reparative and Neoplastic Changes (Meeting Abstract).** (Eng) Uriel, J. (Institut de Recherches sur le Cancer, Villejuif, France). *Digestion* 16(3): 280; 1977. (no refs)

78-3057 **Relation of Lectin-induced and Spontaneous Adhesion to Tumorigenicity.** (Eng) Wright, T. C. (Dept. Pathology, Harvard Medical Sch., 25 Shattuck St., Boston, MA, 02115); Ukena, T. E.; Karnovsky, M. J. *Prog Exp Tumor Res* 22: 1-27; 1978.

Studies of intercellular adhesive interactions mediated by ligands such as plant lectins and spontaneous aggregation occurring between cells in suspension are reviewed, and differences in the adhesion of normal and transformed cells are surveyed. Transformation-related membrane changes, which affect cell-cell and cell-substratum adhesiveness, appear to be one of the most clear-cut systems for demonstrating surface differences between the two cell types. (98 refs)

78-3058 **A Single Protein Key to Cancer?** (Eng) Durham, A. (Institut de Biologie Moleculaire et Cellulaire, CNRS, Strasbourg, France). *New Sci* 77(1096): 860-862; 1978.

A hypothetical mechanism by which a transforming protein may affect cellular transformation is proposed. In this model, the oncoprotein is integrated into the cellular membranes and acts directly or indirectly as an ion channel. Altered ion concentrations cause more oncoprotein to be made and the cell is trapped in a cycle of positive feedback. Under these conditions, the virus would fluorish but the cell would fare badly. (no refs)

78-3059 **Polyamines in Rapid Growth and Cancer.** (Eng) Janne, J. (Dept. Biochemistry, Univ. Helsinki, SF-00170 Helsinki 17, Finland); Poso, H.; Raina, A. *Biochim Biophys Acta* 473(3/4): 241-293; 1978.

Studies of the role of the natural aliphatic polyamines putrescine, spermidine, and spermine in cell proliferation and neoplastic processes are reviewed. Topics covered include: the relation of polyamine synthesis and accumulation to cell cycle and proliferative activity, the relation of the biological effects of polyamines to their postulated physiological functions, the occurrence of polyamines in rapidly growing animal tissues during developmental and compensatory growth, the effect of growth-promoting hormones on the synthesis and metabolism of polyamines in various tissues, polyamine synthesis and accumulation in experimental animal tumors, and the presence of polyamines in human tissues and fluids in various types of cancer. Embryonic and other rapidly growing tissues, both normal and neoplastic, contain high concentrations of polyamines, and the experimental evidence at present indicates that natural polyamines play an essential role in cellular metabolism and in cell proliferation in particular. The use of selective inhibitors of polyamine synthesis as specific antiproliferative agents may have practical therapeutic applications, and measurements of polyamines in physiological fluids may serve as useful markers of human malignancies. (426 refs)

78-3060 **Cancer and Mental Illness (Letter to Editor).** (Eng) Roppel, R. M. (Biomechanics Dept., Michigan State Univ., East Lansing, MI, 48824). *Science* 201(4354): 398; 1978.

The fact that mental patients have a greatly reduced incidence of cancer (vs the general population) may be due to a breakdown of cybergenetic mechanisms, a psychotherapy agent with antitumor effects, a common immunological factor, a factor in the mental hospital environment, or a genetic factor. There is no evidence that favors one of these explanations over the others. (8 refs)

78-3061 **An Evaluation of 6 Short-Term Tests for Detecting Organic Chemical Carcinogens.** (Eng) Purchase, I. F. (Imperial Chemical Industries Ltd., Central Toxicology Lab., Alderley Park, Cheshire, England); Longstaff, E.; Ashby, J.; Styles, J. A.; Anderson, D.; Lefevre, P. A.; Westwood, F. R. *Br J Cancer* 37(6): 873-903; 1978.

The ability of the following six short-term tests to predict the carcinogenicity of 120 organic chemicals was compared: mutation of *Salmonella typhimurium*, cell transformation, degranulation of endoplasmic reticulum, sebaceous gland suppression, tetrazolium reduction, and sc implant. The chemicals included 50 carcinogens and 62 noncarcinogens, and they were divided into four broad chemical classes. All tests but the tetrazolium test showed some ability to discriminate between carcinogens and noncarcinogens, with the cell transformation and bacterial mutation tests being the most accurate (94% and 93%, respectively). The use of cell transformation and bacterial mutation together provided an advantage over the use of either test alone, but the inclusion of the other four tests in a screening battery resulted in a great increase in overall inaccuracy and loss of discrimination. False-negative and false-positive results can be minimized by using appropriate chemical class controls to identify the test that is optimal for the class in question. Structural analogy may play a part in the rapid detection of environmental carcinogens, and some general guidelines for its use are given. (39 refs)

78-3062 **The Tetrazolium-Reduction Test.** (Eng) Westwood, F. R. (Imperial Chemical Industries Ltd., Central Toxicology Lab., Alderley Park, Cheshire, England). *Br J Cancer* 37(6): 949-953; 1978.

A modified tetrazolium-reduction technique was evaluated for its ability to predict the potential carcinogenicity of 120 organic chemicals. Seven drops of a benzene soln of each compound were applied to the clipped dorsolumbar region of male Swiss-derived Alderley Park mice. Differences in tetrazolium reducton that would occur as a result of using hirsute rather than hairless mice were reduced by using 6- to 8-wk-old animals rather than 12- to 15-wk-old animals. The skin was sampled at various times after treatment, and a 2-day interval was found to be optimum. A compound was considered positive (carcinogenic) if there was a significant increase in the amount of formazan deposited in the skin, and a compound was considered negative if there was a significant decrease in formazan deposition or no significant change from control. (The amount of formazan deposited is directly proportional to the oxygen consumption of tissues or cells). The results agreed with those predicted from the literature for only 66/120 compounds. False-negative results were obtained with 36/58 carcinogens and false-positive results were obtained with 18/62 noncarcinogens. (12 refs)

78-3063 **The Degranulation Test.** (Eng) Lefevre, P. A. (Imperial Chemical Industries Ltd., Central Toxicology Lab., Alderley Park, Cheshire, England). *Br J Cancer* 37(6): 937-943; 1978.

The effectiveness of the degranulation test for predicting the carcinogenic potential of 120 organic chemicals was determined. The use of membranes containing radiolabeled RNA for monitoring ribosome loss was also evaluated. Portions of rough and smooth endoplasmic reticulum (RER and SER, respectively) from male Wistar rats were incubated with the test compounds, and the extent of degranulation was measured. More than 90% of the radioactive label administered in vivo was associated with the RNA in both RER and SER, and the ribosomal RNA was uniformly labeled. The results confirm that radioactivity can be equated with RNA content. Of the 58 carcinogens tested, 41 gave a positive result in the degranulation test, and a correct negative result was obtained with 44/62 noncarcinogens tested. There was no apparent correlation between the extent of degranulation and carcinogenic potency, and the test was not specific for liver carcinogens. It is necessary to choose dose levels with care in degranulation assays, because increasing the carcinogen concentration above a certain level decreases the degranulation. (21 refs)

78-3064 **Comparative Cytological and Histological Investigations on Experimentally Induced Thyroid Tumors in the Rat (Meeting Abstract).** (Ger) Schafer, R. (Wurzburg, W. Germany); Muller, H. A. *Zentralbl Allg Pathol* 122(1/2): 146-147; 1978. (no refs)

78-3065 **Molecular Probes for Genetically Toxic Chemicals: Exploitable Features of DNA Damage.** (Eng) Hewitt, R. R. (Dept. Biology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX); Harless, J.; Lloyd, R. S.; Love, J. D. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 7-8 pp.; 1978. (no refs)

78-3066 **Differences in Metabolism of Vinylidene Chloride Between Mice and Rats.** (Eng) Jones, B. K. (Central Toxicology Lab., Imperial Chemical Industries, Alderley Park, Cheshire SK10 4TJ, England); Hathway, D. E. *Br J Cancer* 37(3): 411-417; 1978.

The metabolism of vinylidene chloride (1,1-dichloroethylene, DCE) was investigated in male Alderley Park rats and male Alderley Park mice. Following a single po dose of 50 mg/kg, pulmonary excretion of unchanged DCE accounted for 28% of the dose in rats, but for only 6% in mice. This suggested that the efficiency of DCE metabolism follows the known activity of cytochrome P-450 in the organs of mice, that the real exposure (expressed as amount of DCE metabolized) is relatively higher for orally dosed mice than rats, and that DCE is more likely to be carcinogenic in mice than rats. The mice metabolized DCE similarly to rats, but differences were noted. Qualitatively, mice, but not rats, excreted a small amount of N-acetyl-S-(2-carboxymethyl)cysteine. Quantitatively, (1) the relative proportions of the N-acetyl-S-cysteinylacetyl derivative that were formed in mice and rats paralleled the activity of liver glutathione-S-epoxide transferase, and (2) there were marked differences in the proportions of DCE metabolites belonging to the chloroacetic acid branch of the metabolic pathway. A tracer study verified the β-thionase hydrolysis of thiodiglycollic acid and the biogenesis of the N-acetyl-S-cysteinylacetyl derivative. It is suggested that the metabolites 1,1-dichloroethylene oxide and chloroacetyl chloride may be important in murine DCE carcinogenicity. (19 refs)

78-3067 **Pulmonary Tumors Induced in Mice by Vinyl Chloride Monomer.** (Eng) Suzuki, Y. (Environmental Science Lab., Mount Sinai Sch. Medicine, Fifth Ave. and 100th St., New York, NY, 10029). *Environ Res* 16(1/3): 285-301; 1978.

The incidence of lung tumors due to vinyl chloride (VC) inhalation was studied in 27 CD1 Charles River male mice, and precancerous changes and the resulting tumors were characterized ultrastructurally. The mice were exposed to 2,500 or 6,000 ppm VC 5 hr/day, 5 days/wk, for 5 or 6 mo and then sacrificed 2, 6, or 37 days later. Pulmonary tumors were found in 26/27 mice but in none of the 16 controls. The tumors were round, whitish, multiple, and variable in size from 1 to 5 mm in diameter. The tumors were arranged in tubulopapillary or adenomatous formations. There were no metastases to regional lymph nodes or other organs, parenchymal fibrosis, or fibrotic adhesions of the pleura, although occasional mitotic divisions and invaginations in the bronchiolar lumen were noted. Under the electron micr scope, the characteristic features of the neoplastic cells we short microvilli, tight junctions between two adjacent cell osmiophilic lamellar bodies, large irregularly shaped mit chondria, well-developed Golgi complexes, continuous discontinuous basement membranes, occasional appearan of sequestration and of crystalloids, and a lack of cilia an mucous secretory granules. Some of the cells were poorl differentiated and were equipped with poorly developed o ganoids, without the formation of osmiophilic lamellar b dies. The gross anatomical and histological aspects of th tumors indicated that they were alveologenic tumors. Th neoplastic cells were assumed to have been transformed fro type II alveolar epithelium via its hyperplastic form, becaus of the ultrastructural similarities between normal type II cell and the neoplastic cells. It is concluded that the mouse lun is a sensitive indicator of the oncogenicity of VC. (40 refs

78-3068 **Mutagenic Effects of Vinyl Chloride on *Droso phila melanogaster* with and Without Pretreat ment with Sodium Phenobarbiturate.** (Eng) Magnusson, J (Environmental Toxicology Unit, Wallenberg Lab., Univ Stockholm, Stockholm, Sweden); Ramel, C. *Mutat Res* 57(3) 307-312; 1978.

Exposure of wild-type Karsnas 60 Drosophila males (0- days old) to 10,000, 100,000 or 200,000 ppm vinyl chlorid (VC) gas increased the number of complete and mosai sex-linked recessive lethals. A threshold effect was observe that may be due to a limit of the mixed-function oxidas activity. In additional experiments, Drosophila were pretreated with a 1% soln of phenobarbital sodium (PB) dissolve in water containing 1% sucrose for 24 hr, followed immediately by exposure to 1,000 or 100,000 ppm VC gas (Grou 1). Group 2 was treated with either concentration of VC alone, and Group 3 was not treated. PB pretreatment enhanced the number of recessive lethals over the numbers produced by Groups 2 and 3. Compared with Groups 2 (corresponding group) and 3, the total number of recessive lethals produced by PB + 10,000 ppm VC was significant, but not the number produced by PB + 100,000 ppm VC. In Groups 1 and 2, the higher VC dose did not produce a higher frequency of recessive lethals than the lower dose; again, there was a lack of a dose-response effect. The results indicate that the mixed-function oxidase system can be induced in Drosophila in the same way as in mammals. (12 refs)

78-3069 **Ten Cases of Angiosarcoma of the Liver in Shawinigan, Quebec.** (Eng) Delorme, F. (Dept. Pathology, Centre Hospitalier Regionale de la Maurice, Shawinigan, Quebec, Canada); Theriault, G. *J Occup Me* 20(5): 338-340; 1978.

f the 10 cases of angiosarcoma of the liver diagnosed in hawinigan, Quebec, since 1955, all occurred in men who orked in a vinyl chloride (VC) polymerizing plant. All 10 en were very light drinkers, and only 4 smoked one or more acks of cigarettes per day. The time between the first expo- re of VC monomer and the diagnosis of angiosarcoma nged from 11 to 29 yr, with an av of 20.5 yr. Exposure to C monomer in the plant, although not documented, is be- eved to have been very high in the past. The benign liver sions found in these workers included portal and erisinusoidal fibrosis. (15 refs)

8-3070 **Mutagenicity of Vinyl Chloride. External Chromosome Studies on Persons with and With- ut VC Illness, and on VC Exposed Animals.** (Eng) Fleig, . (Dept. Occupational Medicine and Health Protection, ASF Aktiengesellschaft, 6700 Ludwigshafen, W. Ger- any); Thiess, A. M. *J Occup Med* 20(8): 557-561; 1978.

hromosome analysis was performed on lymphocyte cultures rom 6 workers with an estimated degree of exposure to vinyl hloride (VC), 4 workers whose degree of exposure was moni- ored, 20 workers showing symptoms of VC illness after un- nown degrees of exposure, and 1 patient with VC-induced ngiosarcoma. Bone marrow cells from Chinese hamsters ex- osed to VC by inhalation (2,500 or 5,000 ppm for 4 hr/day 5) or ip injection (300 or 600 mg/kg/day x 5) were also nalyzed. The frequency of chromosome aberrations was not ncreased relative to controls in the exposed workers with no igns of VC illness. The frequency of aberrant metaphases in he workers with VC illness was 5.2% excluding gaps and 1.2% including gaps; these values were significantly higher han the control values of 2.1% and 5.5%, respectively. The ate of aberrant metaphases in the angiosarcoma patient was .3% excluding gaps and isogaps and 16.6% including gaps nd isogaps. The frequency of structural abnormalities in the C-treated hamster cells was also significantly higher than hat in the control cells, inclusive and exclusive of gaps. (15 efs.)

8-3071 **Irreversible Binding of Chlorinated Ethylenes to Macromolecules.** (Eng) Bolt, H. M. (Inst. oxicology, Univ. Tubingen, Wilhelmstrasse 56, D-7400 Tu- ingen-1, W. Germany); Filser, J. G. *Environ Health Perspect* 1: 107-112; 1977.

he binding of vinyl chloride (VC) and trichloroethylene TCE) to male Wistar rat macromolecules was investigated nd compared to that of carbon tetrachloride (CCl_4). Satura- ion of the rat metabolizing systems was achieved at 250 ppm C, 150 ppm TCE, and 250 ppm CCl_4. Data on the uptake f the compounds, the urinary excretion of metabolites, and xhalation after exposure indicated that the chlorinated ethy- enes were metabolized much faster than CCl_4. The metabo- ites of all three compounds bound irreversibly to tissue pro- eins, mainly in the liver. Other sites of binding included the idneys, small intestine, lung, and spleen. Irreversible binding of these compounds ranged within the same order of magnitude when related to the amount of the compound that had been absorbed. No differences in the relative portion of irreversibly bound metabolites were found after exposure of the rats to different atmospheric concentrations of the three compounds. In vitro studies indicated that TCE metabolism by rat liver microsomes in the presence of an NADPH-regenerating system leads to irreversible protein binding, mainly to albumin; similar findings have been reported for VC. Unlike VC, however, TCE metabolites also bind to non-SH proteins such as γ-globulin and concanavalin A. Thus, TCE metabolites irreversibly bind not only to SH groups of a protein, but also to NH_2 groups. (28 refs)

78-3072 **Mutagenicity of the Halogenated Olefin, 2-Bromo-2-chloro-1,1-difluoroethylene, a Presumed Metabolite of the Inhalation Anesthetic Halothane.** (Eng) Garro, A. J. (Dept. Microbiology, Mount Sinai Sch. Medicine, City Univ. New York, New York, NY, 10029); Phillips, R. A. *Mutat Res* 54(1): 17-22; 1978.

The mutagenicity of 2-bromo-2-chloro-1,1-difluoroethylene (BCD), a presumed urinary metabolite of halothane, was tested in the *Salmonella typhimurium* auxotroph reversion assay and in an assay in which a transformable strain of *Bacillus subtilis* was used to score the formation of mutagenic lesions produced by interaction of BCD with isolated transforming DNA. BCD induced both base-substitution and frameshift mutations reverting Salmonella strains TA92, TA98, and TA100. Since these are R factor-carrying strains, the induction of both types of mutations may be the results of error-prone repair of BCD-damaged DNA. Toxicity and mutagenicity were increased concomitantly at higher concentrations (10^{-3}-10^{-1} M), which may reflect BCD-mediated damage to cellular membranes, resulting in increased permeability. In the *B. subtilis* assay, there was a dose-dependent increase in the number of asporagenous mutants among the cells transformed to rifampin resistance with BCD-treated DNA. Inactivation of transforming activity also was observed, but it is not known whether the two effects are due to the same type of lesion in the DNA. Microsomal activation was not required for mutagenesis in either assay. The mutagenicity of BCD may result from direct reaction with nucleophilic sites on DNA bases. The biological significance of BCD mutagenicity with respect to the morbidities noted in epidemiological surveys of anesthesia personnel is not known. A note added in proof states that the BCD was actually only 63% pure; repetition of the experiments revealed that in the *Salmonella* assay, 99% pure BCD induced histidine-independent revertants, but only at toxic concentrations. (26 refs)

78-3073 **Butadiene Diepoxide Activation of Guanylate Cyclase.** (Eng) Vesely, D. L. (Div. Endocrinology and Metabolism, Dept. Medicine, Univ. Miami Sch. Medicine, Miami, FL, 33152); Levey, G. S. *Enzyme* 23(2): 140-143; 1978.

The effect of butadiene diepoxide (BD) on guanylate cyclase activity in various male Sprague-Dawley rat tissues was determined in vitro. BD markedly increased guanylate cyclase activity in all tissues tested, with the degree of stimulation ranging from 2- to 17-fold. The resultant increases in cyclic 3′,5′-guanosine monophosphate secondary to guanylate cyclase activation were significant in all tissues. In terms of max responsiveness to BD in vitro: bladder > kidney > heart > liver > spleen > stomach > pancreas > colon. Max stimulation of hepatic guanylate cyclase was found at a concentration of 100 millimole(mmol)/liter (20-fold increase); a 5-fold increase was noted at 1 mmol/liter and no significant increase at 0.1 mmol/liter. The precise mechanism of guanylate cyclase activation is not understood. (20 refs)

78-3074 **Mutagenic Interaction of Caffeine on Ethyleneimine-induced Chromosomal Damage in a Reconstructed Karyotype of Barley.** (Eng) Gecheff, K. I. (Inst. Genetics and Plant Breeding, Sofia 1113, Bulgaria). *Mutat Res* 50(1): 77-83; 1978.

The effect of caffeine (CF: 5 x 10^{-3} M for 75 min) on the intrachromosomal distribution of ethyleneimine (EI)-induced aberrations was studied in resting (dry) seeds of the barley translocation line T-1586. EI alone (0.04% for 75 min) increased the spontaneous aberration frequency markedly (up to 0.01/cell). The aberrations included isochromatid breaks, duplication deletions, intercalary deletions, chromatid translocations, and triradials. With the dry seeds, the peak of the aberration-frequency curve typical for the mutagens with ‘delayed’ action was not clearly pronounced. CF enhanced the frequency of chromosome aberrations by a factor of 6.2 when applied immediately after EI treatment. The results indicate that CF exerts an effect on the cells long before the beginning of DNA synthesis. CF specifically influenced the distribution pattern of EI-induced aberrations in that it decreased the relative frequencies of centromeric aberrations. Some segments (15, 18, 20, 42, 50, and, especially, 30 and 53) showed a greater sensitivity to the CF enhancement of EI-induced damage. Also, certain chromosome segments revealed a specific involvement in certain types of aberration. It is possible that the capacity of the centromere regions to be involved in aberration formation is restricted during CF enhancement of EI-induced damage. (14 refs)

78-3075 **Enhancement by Caffeine of Sister-Chromatid Exchange Frequency in Lymphocytes from Normal Subjects after Treatment by Mutagens.** (Eng) Faed, M. J. (Cytogenetics Lab., Univ. Dept. Pathology, Ninewells Hosp. and Medical Sch., Dundee, DD2 1UB); Mourelatos, D. *Mutat Res* 49(3): 437-440; 1978.

A study was made of the enhancing effect of caffeine on sister chromatid exchange (SCE) frequency in mutagen-treated lymphocytes from normal subjects. Phytohemagglutinin (PHA)-stimulated cultures of whole blood were grown in the presence of 12 μM bromodeoxyuridine (BUdR). 8-Methoxypsoralen (8-MOP) and UV-A (0.74 joule/cm^2) were administered prior to the addition of PHA and BUdR, but all other agents tested and caffeine were given with the BUdR at 18 hr after culture initiation. Caffeine (5 x 10^{-4}) induced a small but significant increase in SCE rates. However, when caffeine was added to cultures exposed to mitomycin C, 8-MOP/UV-A, 4-nitroquinoline 1-oxide or trenimon, at doses that induce SCE, the effect of the caffeine was synergistic. N-Methyl-N′-nitro-N-nitrosoguanidine (MNNG: 6.6 x 10^{-5} M) had a small effect on SCE rates, but further increases in concentration to 10^{-3} M had no further effect. Post treatment of MNNG-treated cultures with caffeine was also without effect. The effect of posttreatment with caffeine on SCE rates in human lymphocytes following treatment with mutagens is the reverse of that reported for cultured rodent cells. It is suggested that the effect of caffeine on SCE levels in any particular cell type may be determined by the proportion of responsibility for repair carried by excision or postreplication mechanisms. (19 refs)

78-3076 **The Inhibitory Effect of Cysteine on the Mutagenic Activities of Several Carcinogens.** (Eng) Rosin, M. P. (Cancer Res. Centre, Univ. British Columbia, Vancouver, British Columbia, Canada V6T 1W5); Stich, H. F. *Mutat Res* 54(1): 73-81; 1978.

The Salmonella/microsome mutagenesis assay was used to determine the effect of the reducing agent cysteine (α-amino-β-mercaptopropionic acid) on the mutagenicity of several carcinogens: N-methyl -N′- nitro -N- nitrosoguanidine (MNNG), N-acetoxy -2- acetylaminofluorene, N-hydroxy-2-acetylaminofluorene, 4-nitroquinoline 1-oxide methyl methanesulfonate, 5-nitro-2-furaldehyde semicarbazone (nitrofurazone, NF), 2-(2-furyl) -3- (5-nitro -2-furyl)acrylamide, aflatoxin B_1 and the nitrosation products of methylurea and methylguanidine. *Salmonella typhimurium* strains TA98, TA100, and TA1535 were used. At 2.5-10 mM, cysteine inhibited the mutagenic effects of MNNG and the methylguanidine nitrosation products completely; the mutagenicity of all the remaining compounds except NF was inhibited to various extents. Cysteine concentrations of 20-200 mM were required to inhibit NF-induced mutagenesis. The extent of inhibition varied with the particular carcinogen assayed and with the doses of cysteine and carcinogen. It is theorized that reducing agents could act as nucleophiles competing with nucleophilic sites on DNA for the electrophilic ultimate mutagen. Thus, the mutagenic potency of a compound may be related to the rate at which its active radicals are formed and to their electrophilic strength. An increase in these variables would increase he mutagenic potency of a compound as well as the amount f trapping agent required to inhibit its mutagenicity. Other heories are also proposed. (34 refs)

8-3077 **Carcinogenicity of Flower Stalks of *Petasites japonicus* Maxim. in Mice and Syrian Golden amsters.** (Eng) Fushimi, K. (Dept. Pathology, Gifu Univ. ch. Medicine, Gifu-City 500, Japan); Kato, K.; Kato, T.; atsubara, N. *Toxicol Lett* 1(5/6): 291-294; 1978.

he carcinogenicity of the yellow flower stalks of *Petasites aponicus* Maxim. was investigated in ddN, Swiss, and 57BL/6 mice and Syrian golden hamsters. The flower stalks ere dried, milled, and mixed with a basal diet to a concenration of 4%. The animals were placed on the diet for 480 ays. Animals were autopsied at death or when sacrificed ecause of a moribund condition, or at the termination of the xperiment. Of 45 ddN mice, 1 had a hemangioendothelial arcoma of the liver, 4 had reticulum cell neoplasms of the iver, 24 had pulmonary adenomas, 6 had adenocarcinomas f the lung, and 3 had other miscellaneous tumors. No signifiant tumor incidence was noted in any of the other animals tudied. However, proliferative changes in the bile duct, bile uct epithelium, and sinusoidal endothelium of the liver were bserved in all animals, particularly in the C57BL/6 ice and the hamsters. The species and strain difference n susceptibility to the carcinogenicity of these flower talks may be due to differences in ability to metabolize he carcinogenic alkaloid petasitenine in the liver. 8 refs)

8-3078 **Expression of a Bacterial Gene Turned on by a Potent Carcinogen.** (Eng) Levine, A. (Section e Radiobiologie Cellulaire, Laboratoire d'Enzymologie, .N.R.S., 91190, Gif-sur-Yvette, France); Moreau, P. L.; edgwick, S. G.; Devoret, R.; Adhya, S.; Gottesman, M.; as, A. *Mutat Res* 50(1): 29-35; 1978.

method is described whereby a potential carcinogen can e detected within 2 hr by measuring the induction of galacokinase synthesis. A strain of *Escherichia coli* K12 s used in which the galactose operon is integrated nto an early transcribed region of prophage λ and is hus directly under the negative control of the λ reressor. Activated aflatoxin B_1 (AFB_1) induced galactoinase synthesis in the tester strain SA 1512, whereas ure AFB_1 alone did not. A linear dose-response curve as obtained between 0.5 and 25 μg/ml of activated FB_1, enzyme production being enhanced about 0 times over background levels by 25 μg/ml. Using tester train SA 1511, which is similar to SA 1512 but carries a utant gene (*cI857*) that directs the synthesis of a thermolaile repressor, the amount of galactokinase produced at 42 declined at AFB_1 concentrations > 10 μg/ml. This showed the toxic effect of AFB_1 metabolites on the cells. The synthesis of galactokinase was not induced at 32 C by 25 μg/ml of AFB_1 because the λ*cI857* repressor was not inactivated at that temperature. (28 refs)

78-3079 **Aflatoxin Detected by Fluorescence Microscopy During Experimental Hepatocarcinogenesis in Rats and in Primary Hepatomas in Inhabitants of Zaire.** (Fre) Stora, C. (Faculte de Medecine de Nice, Chemin de Vallombrose, 06034 Nice Cedex, France). *Bull Acad Natl Med* 162(4): 287-291; 1978.

Female Sprague-Dawley rats were fed a normal diet or a diet containing 1 or 5 ppm aflatoxin B_1 (AFB_1), or they were inoculated ip with 1 μg of AFB_1 while on a normal diet. Animals were killed every 2 days for 20 days, and their organs were examined for the mycotoxin by immunofluorescence microscopy. Also, biopsy specimens of the livers of Zairians with hepatocarcinomas or cholangiomas who were known to have consumed food contaminated with aspergillus flavus were examined for AFB_1. All of the AFB_1-treated rats had evidence of the mycotoxin in the liver and in all organs beginning on the second day of examination. Initially, the AFB_1 was found only in the cytoplasm of the hepatocytes, but on days 6-15, immunofluorescence was observed in the nucleus. The nuclei of glands such as the maxillary and adrenal glands also contained AFB_1. In the human hepatocarcinoma biopsy specimens, healthy tissue surrounding the tumor showed intense AFB_1 immunofluorescence. The tumor was also very fluorescent, particularly the nuclei. The cholangiomas were much less fluorescent than the hepatocarcinomas, and there was evidence of aflatoxin G as well. (no refs)

78-3080 **Metabolic Activation of Aflatoxins Related to Their Mutagenicity.** (Eng) Gurtoo, H. L. (Dept. Experimental Therapeutics, Roswell Park Memorial Inst., New York State Dept. Health, Buffalo, NY, 14263); Dahms, R. P.; Paigen, B. *Biochem Biophys Res Commun* 81(3): 965-972; 1978.

Rat hepatic microsome-mediated DNA binding and mutagenesis to *Salmonella typhimurium* strain TA 100 by various aflatoxins and some mixed-function oxygenase (MFO)-mediated metabolites of aflatoxin B_1 (AFB_1) were studied. Hepatic microsomes isolated from 3-methylcholanthrene-treated C57BL/6J mice were used to prepare AFM_1, and hepatic microsomes from phenobarbital-treated rats and mice were used to prepare AFQ_1 and AFP_1, respectively. Hepatic microsomes from phenobarbital-treated rats were used in both the DNA-binding assay and the mutagenesis assay. In the range 28-112 μM, the DNA-binding activity of AFB_1 was linear with the substrate concentration. AFB_1 was shown to be a potent mutagen. AFB_2, a congener of AFB_1, that lacks the C_2-C_3 double bond, did not bind to DNA and was $< 1\%$ as mutagenic as AFB_1. AFG_1 was 18% as active as AFB_1 in the DNA-binding assay and 8%-10% as potent in the mutagenesis assay. AFG_2, a congener of AFG_1 that lacks the C_2-C_3 double bond, was inactive in binding to DNA and was as weak as AFB_2 in the mutagenesis assay. These data suggest that there is a good correlation between in vitro DNA binding and mutagenesis and that the critical site in the molecule is the C_2-C_3 double bond. In microsome-mediated DNA binding and mutagenesis to *S. typhimurium* TA100, AFM_1, AFQ_1, and AFP_1, were $< 50\%$ as active as AFB_1, indicating that MFO-mediated metabolism at sites other than the C_2-C_3 double bond in the AFB_1 molecule represents a deactivation step. (30 refs)

78-3081 **Quantitative Determination of Aflatoxins B_1, B_2, G_1, and G_2 in Foods.** (Ger) Muller, H. (Institut fur Forschung und Entwicklung, Maizena Gesellschaft mbH, Knorstrasse 1, D-7100 Helbronn, W. Germany); Siepe, V. *Dtsch Lebensm Rundsch* 4: 133-136; 1978.

A chromatographic-spectrophotometric method using methylene chloride as the extraction agent and solvent instead of chloroform, a suspect carcinogen, was developed for the determination of aflatoxins in foods. The aflatoxin B_1, B_2, G_1, and G_2 levels found in peanuts, hazelnuts, almonds, oat, rice, buckwheat, and wheat, barley, and corn products ranged from 75 to 108 ppb. (5 refs)

78-3082 **High Pressure Liquid Chromatographic Determination of Aflatoxins in Peanut Products.** (Eng) Pons, W. A. (U.S. Dept. Agriculture, Science and Education Admin., Southern Regional Res. Center, New Orleans, LA, 70179); Franz, A. O. *J Assoc Off Anal Chem* 61(4): 793-800; 1978.

A rapid, precise, and sensitive high-pressure liquid chromatographic method for determining aflatoxins in all types of peanut products is based on acidified aqueous methanol extraction, partition of aflatoxins into dichloromethane, and purification of the extract on silica gel. Accurate and sensitive detection of aflatoxins B_1, B_2, G_1, and G_2 was made at levels as low as 0.3-1.0 μg/kg. (18 refs)

78-3083 **Morphology, Growth and Activation of Chemical Carcinogens by Liver Cells in Culture (Meeting Abstract).** (Eng) Tompa, A. (Eppley Inst. Res. Cancer, Omaha, NB, 68105); Freed, H.; Huberman, E.; Langenbach, R. *In Vitro* 14(4): 337; 1978. (no refs)

78-3084 **Uptake of [^{14}C]-Aflatoxin B_1 by Liver and Kidney Slices of Different Animal Species.** (Eng) Prasanna, H. R. (Dept. Biochemistry, Vallabhbhaipatel Chest Inst., Univ. Delhi, Delhi-110007, India); Gupta, S. R.; Viswanathan, L.; Venkitasubramanian, T. A. *Z Lebensm Unters Forsch* 166(3): 162-163; 1978.

The respective uptake of [^{14}C]-aflatoxin B_1 (AFB_1) by liver and kidney slices of different animal species was compared. For rat, mouse, guinea pig, rabbit, and monkey, the uptake (cpm/100 mg tissue) in liver and kidney slices was 641 and 612, 598 and 561, 653 and 992, 398 and 682, and 882 and 735, respectively; while the LD_{50} (mg/kg) was 7.0, 60.0, 1.0, 0.5 and 2.2, respectively. Thus, the transport of the toxin into liver or kidney is not the major cause for the variation in species susceptibility to AFB_1. (10 refs)

78-3085 **7,8-Benzoflavone Stimulates the Metabolic Activation of Aflatoxin B_1 to Mutagens by Human Liver.** (Eng) Buening, M. K. (Dept. Biochemistry and Drug Metabolism, Hoffmann-LaRoche Inc., Nutley, NJ, 07110); Fortner, J. G.; Kappas, A.; Conney, A. H. *Biochem Biophys Res Commun* 82(1): 348-355; 1978.

The addition of 7,8-benzoflavone (7,8-BF) to a monooxygenase system from human liver markedly stimulated the metabolic activation of aflatoxin B_1 (AFB_1) to reactive intermediates that caused mutations in *Salmonella typhimurium* strains TA98 and TA100. When 5×10^{-5} M 7,8-BF was added to this monooxygenase system, the amount of AFB_1 needed for a mutagenic response was decreased by twenty- to fortyfold. 7,8-BF did not enhance the metabolic activation of AFB_1 to mutagens when rat liver was used as the source of monooxygenase. The results suggest that 7,8-BF may promote the formation of the carcinogenic metabolite AFB_1-2,3-oxide in human liver. (21 refs)

78-3086 **Nasopharyngeal Carcinoma Associated with Long-Term Arsenic Ingestion.** (Eng) Prystowsky, S. D. (Letterman Army Inst. Res., San Francisco, CA); Elfenbein, G. J.; Lamberg, S. I. *Arch Dermatol* 114(4): 602-603; 1978.

A 41-yr-old white woman with psoriasis who had been treated with Fowler's soln (potassium arsenite) for > 20 yr developed a metastatic nasopharyngeal carcinoma (NPC). Laboratory analysis indicated a serum antibody titer of 1:400 to Epstein-Barr virus. Palmar and plantar keratoses were present. Although the role of the virus in the development of this cancer cannot be excluded, it is suggested that NPC may be associated with long-term arsenic treatment in patients who are not genetically predisposed or residing in endemic regions the world. Long-term arsenic ingestion may be additive or nergetic with other causal factors in oncogenesis. (24 refs)

3-3087 **Cadmium Content of Lung, Liver and Kidney in Rats Exposed to Cadmium Oxide Fumes.** (Eng) oisset, M. (I.N.S.E.R.M. U. 122--Laboratoire de Toxicolo-ie, U.E.R. des Sciences Pharmaceutiques et Biologiques, ue J.B. Clement, F-92290 Chatenay-Malabry, France); Gi-ırd, F.; Godin, J.; Boudene, C. *Int Arch Occup Environ lealth* 41(1): 41-53; 1978.

Iale Sprague-Dawley rats were exposed to air containing .43 mg/m^3 cadmium oxide 30 min/day for 5 consecutive ays and then sacrificed at various times over a 3-mo period) determine the effects on the lungs, liver, and kidneys. The me-course effect on the relative wt of the lungs was biphasic, ith an increase noted immediately after exposure and again om days 42 to 56. Macroscopic examination of the pulmo-ary lobes of exposed rats sacrificed on days 42, 56, and 84 evealed areas of scar formation. Liver wt increased after 14 ays, but the increase was significant only when it was ex-ressed as percent of body wt. A second significant rise was bserved during the third month of observation. Little effect n kidney wt was noted except for a slight increase after 14 ays. Cd deposits in the lungs were cleared slowly, and the stimated half-life was 56 days. There was also a slight but ignificant accumulation of Cd in the liver that reached a max fter 2 mo. Cd concentrations in the liver never exceeded 0.63 ıg/g. Significant amounts of Cd accumulated in the kidney, ut only during the first month of observation. No mortality vas noted in the exposed groups. The body wt of exposed rats lecreased during the first 2 wk postexposure but then re-urned to normal. The slow clearance rate could be the result f impaired phagocytic ability of the pulmonary macro-hages. (35 refs)

'8-3088 **Cytotoxic Effects of Hexavalent and Trivalent Chromium on Mammalian Cells In Vitro.** (Eng) Levis, A. G. (Inst. Animal Biology, Univ. Padova, Via Lore-dan n. 10, 35100 Padua, Italy); Bianchi, V.; Tamino, G.; Pegoraro, B. *Br J Cancer* 37(3): 386-396; 1978.

The cytotoxic effects of potassium dichromate ($K_2Cr_2O_7$; hex-avalent Cr) and chromium chloride ($CrCl_3$; trivalent Cr) on cultured BHK 21 hamster fibroblasts and HEp 2 human epi-theliallike cells were determined. $K_2Cr_2O_7$ (10^{-5} to 10^{-3} M) stimulated the uptake of labeled thymidine (TdR) into the soluble intracellular pool, but Cr^{3+} (10^{-2} M) exerted an in-hibitory action. DNA synthesis was inhibited by treatment with both chromium compounds, but especially by $K_2Cr_2O_7$. Furthermore, 10^{-2} M $CrCl_3$ reduced the sensitivity of DNA and RNA to hydrolysis with per-chloric acid. Treatment of the cells with $K_2Cr_2O_7$ in balanced salt soln, in which Cr^{6+} reduction is less marked, induced more pronounced cytotoxic effects than treatments in complete growth medium. HEp cells were more sensitive to hexavalent Cr than BHK fibroblasts: in the former line, TdR uptake was less stimulated, and DNA synthesis and cell survival were more affected. Survival of BHK cells to $K_2Cr_2O_7$ indicated a multihit mechanism of cell inactivation, with about 10 independent Cr-induced events being necessary for cell death. The cytotoxic effects of Cr were attributed to the action of Cr^{6+} at the plasma membrane level on the mechanisms involved in nucleoside uptake and to the interaction of Cr^{3+} at the intracellular level with nucleophilic targets on the DNA molecule. (36 refs)

78-3089 **Bronchopulmonary Carcinomas in Two Chromium-exposed Workers Employed in the Same Section of an Electroplating Factory.** (Fre) Michel-Briand, C. (Inspection medicale du travail, 23, rue de la Republique, 25000 Besancon, France); Simonin, M. *Arch Mal Prof* 38(12): 1001-1013; 1977.

Bronchopulmonary carcinoma occurred in two men (41 and 47 yr old) employed in the same electroplating section of a factory. One patient was exposed to toxic chromium vapors for 9 yr, and the second was exposed to chromic acid for > 5 yr; both men had a history of smoking. Changes in the working conditions had increased the risk over the years. It is hypothesized that these two carcinomas were related to the inhalation of chromic acid. (22 refs)

78-3090 **In Situ Mutagenic and Other Effects Associated with Lead Smelting.** (Eng) Lower, W. R. (Dept. Family and Community Medicine, Environmental Trace Substances Res. Center, Columbia, MO, 65201); Rose, P. S.; Drobney, V. K. *Mutat Res* 54(1): 83-93; 1978.

The applicability of several plant systems, Zea (corn), Tradescantia (Trad: spiderwort), and Glycine (soybean), to in situ mutagenesis monitoring was tested; the mutagenic impact on the environment of a large lead-smelting operation in Southeastern Missouri, whose major effluents are Pb, Cd, Cu, Zn, and S, was assessed; and biochemical and physiological tests were used to determine any nonmutagenic toxicity to indigenous mammals and test plants. The latter included assays of Hb synthesis in Peromyscus (white-footed mouse), N fixation of Glycine root tissue and determination of Zea height, Zea pollen abortion, and day of first flowering of Trad. The total concentration of metals in the soil decreased with distance from the smelter. Levels of plant-available concentrations of Pb, Cd, Cu, and Zn were lower than the total metal levels, but the arithmetic correlations between the two were high. Mutation frequency and pollen abortion increased in Zea and Trad, and urinary δ-aminolevulinic acid excretion increased in the mice. Zea height, Trad flowering time, and Glycine root N fixation decreased. The change in the germinal mutation frequency of Zea and the somatic mutation frequency of Trad was not a linear function of distance from the smelter. The highest frequencies were at 1.7 km and not at 0.3 m. Greenhouse experiments using soil from these sites confirmed the findings that more moderate exposure can result in an increase in mutation frequency. However, with all five toxicity parameters, the most toxic conditions were at 0.3 km compared with the other sites (to 14.7 km) and control sites. This same level of toxicity and the resulting physiological damage may differentially mask mutagenesis at 0.3 km in Zea and Trad. Evidence is given that shows that the smelter operation, the only industry in an otherwise undeveloped area, was responsible for these effects. (21 refs)

78-3091 **Investigation of Exposure to Nickel and Lung Cancer Mortality: Case Control Study at Aircraft Engine Factory.** (Eng) Bernacki, E. J. (Dept. Lab. Medicine, Univ. Connecticut Sch. Medicine, Farmington, CT, 06032); Parsons, G. E.; Sunderman, F. W. *Ann Clin Lab Sci* 8(3): 190-194; 1978.

A case-control mortality study was performed at an aircraft engine factory to assess the possible association between exposure to nickel-containing compounds and death from lung cancer. Forty-two nonsalaried male workers who died of lung cancer from 1966 to 1976 were identified from a chronological registry of death certificates maintained by the company's medical department. Each of the decedents was matched with the next two nonsalaried decedents in the registry who were of the same sex and comparable age (±10 yr) and who died of causes other than cancer. Without knowledge of the workmen's identities or causes of death, one of the authors classified the deceased workmen as exposed or not exposed to nickel compounds during their employment. The proportion of workmen who died of lung cancer and were exposed to nickel (11/42) was equal to the proportion of workmen who died of noncancerous causes and were exposed to nickel (22/84). The results indicate that chronic occupational exposure to low atmospheric concentrations of nickel compounds did not increase the relative risk of mortality from lung cancer prior to retirement. (21 refs)

78-3092 **Microbiological Mutagenicity Studies of Pesticides In Vitro.** (Eng) Carere, A. (Istituto Superiore di Sanita, Rome, Italy); Ortali, V. A.; Cardamone, G.; Torracca, A. M.; Raschetti, R. *Mutat Res* 57(3): 277-286; 1978.

The mutagenic activity of 14 pesticides as pure compounds was compared in two in vitro microbial genetic assay systems. The pesticides were tested for the induction of point mutations in four strains of *Salmonella typhimurium* (TA1535, TA1536, TA1537, and TA1538) in the presence and absenc of rat liver microsome fractions and for the induction of resistance to low concentrations (3-5 $\mu g/ml$) of streptomyci in the filamentous bacterium *Streptomyces coelicolor*. In mos cases, the spot test technique was used: triangles of steril absorbent paper saturated with a soln of the test compoun were applied to the surface of the agar medium. The pesticides investigated were: aminotriazole, Benomyl, Captafol Captan, Dichlorvos, Dalapon-Na, Dinobuton, Dodine, Ioxynil, Mecoprop, Neburon, Picloram, Triallate, and Trichlorphon. In Salmonella, Captan and Triallate were mutageni for TA1535; Dichlorvos and Trichlorphon were negative i the spot test, but mutagenic after incubation in liquid culture of TA1535. In the *S. coelicolor* forward-mutation test, aminotriazole, Dichlorvos, Picloram, Trichlorphon, and Triallat were mutagenic in the spot test, and Captan showed weal mutagenicity with a plate incorporation technique. The results are compared with those in the literature. It is suggeste that the *S. coelicolor* test will prove useful for the study o environmental mutagens. (27 refs)

78-3093 **The Induction of II-III Translocations by Tris-(2,3-dibromopropyl) Phosphate in Drosophila.** (Eng) Berkowitz, S. (Dept. Zoology, Univ. Wisconsin Madison, WI, 53706). *Mutat Res* 57(3): 385-387; 1978.

The capacity of the flame retardant tris(2,3-dibromopropyl) phosphate (TBPP) to induce chromosome rearrangements was determined in Drosophila. Seven-day-old adult Canton-S males were fed for 48 hr on a 1.0% glucose soln containing 1,000 ppm (1.43 x 10^{-3} M) or 10,000 ppm (1.43 x 10^{-2} M) TBPP. Exposed males were mated immediately after treatment to brown ebony virgins in culture bottles containing 20 males and 40 females. Every 2 days, the males were provided with fresh vials of virgins to sample a variety of treated germ cell stages. F_1 males were singly mated in vial cultures to virgins, and F_2 cultures were scored for II-III translocations. The results showed that TBPP was capable of causing reciprocal translocations in Drosophila, with 8 translocations being found out of 8,000 tests. The translocation recovery at 10,000 ppm was not significantly different from that found at 1,000 ppm, a result that could be due to an enzyme saturation effect, as TBPP requires metabolic activation. The higher mutation rate found for early spermatids and/or spermatocytes relative to mature sperm is consistent with the view that these early stages are more mutable than later stages. (7 refs)

78-3094 **Mutagenicity of Derivatives of the Flame Retardant Tris(2,3-dibromopropyl) Phosphate: Halogenated Propanols.** (Eng) Carr, H. S. (Dept. Microbiology, New York Medical Coll., Valhalla, NY, 10595); Rosenkranz, H. S. *Mutat Res* 57(3): 381-384; 1978.

'he mutagenicity of some mono- and dihalopropanols, erivatives of the flame retardant tris(2,3-dibromopropyl) hosphate (TBPP), was tested in the *Salmonella typhimuri-* *m* assay. Both the bacteria and the chemicals were incor- orated into the agar overlay. Tester strains TA100, TA1535, 'A1538 were used in the presence and absence of metabolic ctivation (microsomal fraction derived from Araclor-in- uced rat livers). Specific mutagenicities (revertants/nano- iole) were calculated from the linear portion of the ose-response curve obtained with TA1535 in the absence of iicrosomal activation. All of the halogenated propanols test- d, except for 3-chloro-1-propanol, exhibited direct-acting ase-substitution activity, as evidenced by their mutagenicity or TA100 and TA1535 but not TZ1538. Even with microso- ial activation, 3-chloro-1-propanol remained genetically in- rt. All bromo analogs were more mutagenic than the corre- ponding chloro compounds except for the bromo analog of ,3-dichloro-1-propanol. The mutagenicity of some of the romine-containing chemicals was further increased in the resence of rat liver microsomes. (14 refs)

8-3095 **Present and Future Uses of Mutagenicity Tests for Assessment of the Safety of Food Additives.** Eng) Green, S. (Dept. Pharmacology, Howard Univ. Coll. ledicine, Washington, DC, 20059). *J Environ Pathol Toxicol* (2): 49-54; 1977.

'he tier approach to investigating the potential mutagenicity f food additives and substances generally recognized as safe GRAS substances) is described. This approach is used to set esting priorities. The first tier, which is a prescreen to deter- iine whether a chemical has the potential to cause genetic amage, is composed of tests using *Salmonella typhimurium* 'A1535, TA1537, TA1538, TA98, and TA100, with and vithout metabolic activation, for the detection of gene muta- ions. Tier 2 is used to confirm the mutagenic activity detect- d at tier 1 and is comprised of tests in mouse lymphoma .5178Y cells and in *Drosophila melanogaster* and the rat ominant lethal assay. Tier 3 attempts to define the genetic isk to humans that is associated with use of a substance onfirmed as a mutagen in tiers 1 and 2. The specific locus ssay and the heritable translocation test are used at tier 3. nformation assembled in 1974 from tests of 51 GRAS sub- tances was compared, and the results showed that there is ery little correlation among the short-term mutagenicity ests or between the short-term and animal tests. It is con- luded that mutagenicity tests in the future will probably be ised (1) as a prescreen for in vivo carcinogenicity studies and 2) as a means of identifying materials that may produce heri- able genetic damage in humans. (5 refs.)

'8-3096 **Chromosomal Damage and Hair Dyes (2 Letters to Editor).** (Eng) Kirkland, D. J. (British Indus- rial and Biological Res. Assoc., Carshalten, Surrey, England); Kirkland, D. J.; Lawler, S. D.; Venitt, S. *Lancet* 2(8083): 271-272; 1978.

Data from a previous study of the effects of hair dyes on chromosomes are interpreted to mean that women who dye their hair are unlikely to suffer greater chromosomal damage than women who have had recent exposure to x-rays or virus infection. In a response, the original authors state that the x-ray exposures referred to were high-dose diagnostic x-rays, and the virus exposures were recent influenza, chicken pox, heavy vaccination, or glandular fever. In fact, the data showed that there was a statistically significant excess of chromosomal damage (mainly chromatid breaks) in women with dyed hair. (no refs)

78-3097 A Mutation in *Drosophila melanogaster* that **Generates Ovarian Tumors After a Latent Period (Meeting Abstract).** (Eng) Gollin, S. M. (Northwestern Univ., Evanston, IL); King, R. C. *Genetics* 88(4, part 2, Suppl): s31-s32; 1978. (1 ref)

78-3098 **Carcinogenesis Studies on Organ Cultures of Animal and Human Respiratory Tissues.** (Eng) Saffiotti, U. (Experimental Pathology Branch, Carcinogenesis Res. Program, DCCP, NCI, NIH, Bethesda, MD, 20014); Harris, C. C. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research, held in Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute. (Houston, Texas): pp. 2-4; 1978.

Organ culture models of respiratory tract carcinogenesis are described. Preneoplastic epithelial lesions induced by chemical carcinogens in cultured human bronchi were identifiable by light and electron microscopy. Autoradiography was used to identify the cellular localization of the bound carcinogens and to measure changes in the cell kinetics of the respiratory epithelium. In biochemical studies, procarcinogens (eg, polynuclear aromatic hydrocarbons, N-nitrosamines, and mycotoxins) were enzymatically activated by human tissues to electrophiles that formed adducts with DNA. Human tissue-mediated mutagenesis was demonstrated when human bronchi explants were cocultivated with Chinese hamster V-70 cells in the presence of procarcinogens. (no refs)

78-3099 **Unscheduled DNA Synthesis Testing of Pesticides (Meeting Abstract).** (Eng) Mitchell, A. D. (Stanford Res. Inst., Menlo Park, CA, 94025); Robinson, D.

E.; Sterling, G. P.; McAfee, P. A.; Waters, M. D. *Mutat Res* 53(1): 109; 1978. (no refs)

78-3100 Carcinogenic Activity of Compounds Eluting from Elastic Resins into Food. (Rus) Medvedev, V. I. (All-Union Res. Inst. Hygiene and Toxicology Pesticides, Polymers and Plastics, Kiev, USSR); Rodionov, G. A. *Vopr Onkol* 24(6): 94-99; 1978.

The potential carcinogenic activity of the compounds that can elute from elastic resins into food products was tested in C57BL mice. Extracts from two types of resins (I and II) were administered either intragastrically (0.024 and 0.068 μg/kg, respectively, 3x/wk, for 6 mo) or sc (0.2 μg/kg, 2x/wk, for 8 mo). The animals were sacrificed after 18 mo. Intragastric administration of resins I and II induced tumors in 11/46 and 3/37 mice, respectively. The tumors included pulmonary adenomas (5 and 2), papillomas of the cardiac stomach (2 and 1), and tumors of other location (lipoma of the small intestine, liver adenoma, and renal fibroma, which were detected only in mice exposed to type I). The sc application of type I resin induced tumors in 11/48 mice (6 skin papillomas, 4 pulmonary adenomas, and 1 adenocarcinoma of the stomach). (12 refs)

78-3101 Absence of Chromosome Damage in the Bone Marrow of Rats Fed Detergent Actives for 90 Days. (Eng) Hope, J. (Environmental Safety Div., Unilever Res. Lab., Colworth House, Shanbrook, Bedford, England). *Mutat Res* 56(1): 47-50; 1977.

Four detergent actives, sodium lauryl sulfate, DOBS 055 (nominally dodecyl benzene sulfonate, but with a chain length of 10-15 carbon atoms), Dobanol 25 sulfate LCU, and Dobanol 25 sulfate HCB, were tested for their potential to induce chromosome damage in vivo after chronic administration in the diet to rats. The detergent actives were fed to Colworth/Wistar rats for 90 days at the max tolerated dose, 1.13% active ingredient in each case. Sodium lauryl sulfate and DOBS 055 were also fed at 0.56%. Two hours before sacrifice, the rats were inoculated ip with colchicine. Chromosome preparations were made from the bone marrow and scored for the presence of rearrangements, chromatid gaps and breaks, and isochromatid gaps and breaks. The four detergent actives had no effect on the chromosomes of rat bone marrow cells. The absence of chromosome damage suggests that these detergent actives are harmless. (3 refs)

78-3102 Experiments on the Carcinogenic Effect of Ortho-Toluol-sulfonamid (OTS). (Eng) Schmahl, D. (Inst. Toxicology and Chemotherapy, German Canc Res. Center, Im Neuenheimer Feld 280, D-6900 Heidelber W. Germany). *Z Krebsforsch* 91(1): 19-22; 1978.

The carcinogenicity of O-toluenesulfonamide (OTS), an e sential impurity in saccharin prepared by the Remser Fahlberg method, was investigated in Sprague-Dawley rat The OTS was mixed with the food and fed to the rats at dail doses of 200 and 20 mg/kg; the diets were continue for life. The max dose given amounted to 17 g/kg. No life shortening effects were observed, and the incidence of malig nant tumors in the OTS-treated animals was identical to tha in a control group. However, one animal on the high-dos diet developed a urinary bladder carcinoma; four benign tu mors were noted in four other animals in this group. Thre animals in the low-dose group developed bladder papillomas Although these results do not prove that OTS is carcinogenic it is suggested that saccharin used in future experiments b kept as pure as possible. (8 refs)

78-3103 Analysis of Aryl Hydrocarbon Hydroxylase Ac tivity in Human Lung Tissue, Pulmonary Mac rophages, and Blood Lymphocytes. (Eng) McLemore, T. L (Dept. Medicine, Infectious Diseases Section, Baylor Coll Medicine, 1200 Moursund Ave., Houston, TX, 77030). *Cancer* 41(6): 2292-2300; 1978.

Aryl hydrocarbon hydroxylase (AHH) levels were measured in pulmonary alveolar macrophages (PAM) freshly lavaged from the lung, in fresh surgically excised lung tissue, and in cultured mitogen-stimulated peripheral blood lymphocytes Samples were obtained from seven patients without cancer (controls) and seven patients with untreated primary lung cancer; all were cigarette smokers. AHH levels in the PAM and lymphocytes from individual control patients, but not from individual lung cancer patients, were correlated positively. AHH levels in freshly excised lung tissue and in the PAM correlated strongly in the controls but only weakly in the cancer patients. AHH activity in freshly excised lung tissue was also positively correlated with AHH fold-induction ratios in cultured lymphocytes from individual controls; there was no such correlation in individual cancer patients. In the controls, but not in the cancer patients, there was an excellent positive correlation among AHH activity levels in fresh PAM and in fresh lung tissue and fold-induction of AHH in cultured lymphocytes. These data indicate that the capacity for AHH induction is similar in the fresh lung tissue, fresh PAM, and cultured mitogen-stimulated lymphocytes of cigarette smokers without evidence of lung cancer, but that AHH levels in similar tissues from individual lung cancer patients are not correlated positively. (42 refs)

78-3104 Aryl Hydrocarbon Hydroxylase Activity in Subpopulations of Peripheral Blood Mononu-

clear Cells. (Eng) Jett, J. R. (Dept. Pathology and Anatomy, Mayo Clinic/Foundation, Rochester, MN, 55901); Stobo, J. D.; Moses, H. L. *Cancer Res* 38(7): 1979-1982; 1978.

The activity of aryl hydrocarbon hydroxylase (AHH) was measured in peripheral blood mononuclear cell (PMC) subfractions consisting of T-cell-enriched, T-cell-depleted, and monocyte-depleted populations. Fluorometrically and radiometrically, the T-cell-enriched populations consistently showed enhanced AHH activity compared with the total PMC population. This enhanced activity was observed by the sheep RBC rosette method when the T-cell-enriched populations were isolated either before or after 96 hr of lymphocyte culture or by the nylon wool column technique before lymphocyte culture. T-cell-depleted populations (B-cell-enriched) obtained by the sheep RBC rosette method had diminished AHH activity. Monocytes were shown to contribute to the total PMC AHH activity through an indirect technique, in which monocytes were first depleted from PMC by the carbonyl iron method. The monocyte-depleted populations had less AHH activity than the total PMC population after both 24 and 96 hr of culture. The greatest amount of AHH activity was present in PMC populations with their native number of monocytes when cultured for 96 hr in the presence of mitogens. These results suggest that studies of AHH activity in patients with different cancers should take into account the relative composition of PMC and the degree of mitogen stimulation. (29 refs)

78-3105 **The Interaction of Asbestos Exposure and Smoking on Respiratory Health.** (Eng) Rossiter, C. E. (MRC Pneumoconiosis Unit, Llandough Hosp., Penarth, Glamorgan CF6 1XW, Wales); Berry, G. *Bull Eur Physiopathol Respir* 14(2): 197-204; 1978.

The association between asbestos exposure, smoking, and the development of asbestosis was investigated in workers at the Royal Navy Dockyards (RND) and in workers at an asbestos textile factory. The presence of small opacities on chest radiographs and crepitations that are not cleared on coughing were studied because of their importance in the diagnosis of asbestosis. A total of 195 asbestos workers and 1,005 other workers at the RND formed the basis for the first study. Among the asbestos workers who smoked, the prevalence of small opacities rose markedly for those with >20 yr exposure to asbestos; this finding did not hold for exsmokers and nonsmokers, who had prevalence rates compatible with a base rate of approx 3%. Crepitations that did not clear upon coughing also showed higher prevalence rates for smoking asbestos workers with >20 yr exposure to asbestos. Prevalence rates among exsmokers and nonsmokers did not vary significantly from the av of 8%. In the second study, the presence of crepitations and asbestosis was determined in nonsmokers, exsmokers, and people who smoked 1->25 cigarettes/day in the textile factory. The av cumulative exposure to asbestos was similar for all smokers. Crepitations were never heard in nonsmokers or in men who smoked <5 cigarettes/day; the prevalence in the heavier smokers averaged 19%. The presence of small opacities in the lung was also greater in heavier smokers than nonsmokers. These findings indicate that among asbestos workers, smokers have a higher risk of developing asbestosis than nonsmokers. A review of the pertinent literature suggested that a synergism between asbestos exposure and smoking occurs at an early age and can lead to premature death. (14 refs)

78-3106 **Pleuropulmonary Disease among Asbestos Workers in Relation to Smoking and Type of Exposure.** (Eng) Weiss, W. (Div. Occupational Medicine, Dept. Medicine, Hahnemann Medical Coll. and Hosp., 6401 New Coll. Building, 230 N. Broad St., Philadelphia, PA, 19102); Theodos, P. A. *J Occup Med* 20(5): 341-345; 1978.

The chest roentgenograms of employees aged 40+ in two asbestos manufacturing plants were read by consensus without knowledge of the plant or smoking habits. Plant A used only chrysotile asbestos throughout its history, Plant B used amosite as well as chrysotile, the amosite being limited to the period 1950-1964. The incidence of pulmonary disease was 10/40 in Plant A and 16/48 in Plant B. The incidence of pleural thickening was 18% in Plant A and 35% in Plant B. Neither age nor work duration were factors in these differences. In Plant A, there was no obvious relationship between smoking and pleuropulmonary disease. In Plant B, there was a definite relationship of smoking to pulmonary disease and perhaps to pleural thickening. Both types of asbestos and smoking habits appear to be determinants of pleuropulmonary disease in asbestos workers. (11 refs)

78-3107 **Differential Response in the Male and Female Tracheal Epithelium Following Exposure to Tobacco Smoke.** (Eng) Hayashi, M. (Thorndike Lab., Dept. Medicine, Harvard Medical Sch., Boston, MA); Sornberger, G. C.; Huber, G. L. *Chest* 73(4): 515-518; 1978.

Histochemical quantification of goblet cells and measurements of epithelial thickness were made in the trachea of male and female CD rats exposed for 30 consecutive days (3-10 min exposures/day) to fresh whole smoke from standard 2R1 Kentucky reference unfiltered cigarettes. Smoke exposure increased the total goblet cell count in both sexes, with the female rats differentially responding to a greater degree than the male rats. There was a shift from PAS-positive to alcian blue-positive goblet cells, an increase in the number of both 'large' and 'small' goblet cells, and an increase in epithelial thickness. PAS-positive 'small' cells, the major glycoprotein-reactive cell in the rat tracheal epithelium, increased in the female rats but showed no increase in the male rats.

However, male rats contained a substantially higher proportion of alcian blue-positive cells than did female rats, both before and after smoke exposure. It is not known whether these differences in the morphologic response of the tracheal epithelium to experimental exposures to smoke exist in humans and whether the differences contribute to the differential risk of respiratory disease following tobacco smoking in men and women. (24 refs)

78-3108 Smoking and Lung Cancer: A Cohort Data Study of Men and Women in England and Wales 1935-70. (Eng) Townsend, J. L. (Univ. Essex, Colchester, England). *J R Statist Soc A* 141(Part 1): 95-107; 1978.

In 1965, Pike and Doll postulated that the risk of death from lung cancer among doctors was linearly related to the level and duration of cigarette smoking. A generalization of the Doll and Pike model gave a very good approximation to male mortality from lung cancer over the period 1935-1970, and it explained 98.9% of the variation in excess mortality. The fit for women was not as close as that for men, but the model accounted for 93.8% of the variation between age groups and over time. The model predicted the fall in 1973 mortality rates in men aged 35-65 and the rise in mortality among women aged 45-80. In the period 1966-1970, smoking accounted for 94.4% of lung cancer mortality in men and 71.2% of the mortality in women. Cigarette smoking affected the mortality risk equally, whether the smoking took place early or late in life. Pipe and cigar smoking appeared to have an insignificant effect on mortality. (13 refs)

78-3109 Carboxyhaemoglobin Levels and Inhaling Habits in Cigarette Smokers. (Eng) Wald, N. (Dept. Regius Professor Medicine, Univ. Oxford, Radcliffe Infirmary, Oxford OX2 6HE, England); Idle, M.; Bailey, A. *Thorax* 33(2): 201-206; 1978.

Carboxyhemoglobin (COHb) levels were determined in 520 male cigarette smokers, aged 35-64 yr, and the results were compared with those obtained for 1,891 nonsmokers. The mean COHb was 0.7% among the nonsmokers and 5.3% among the smokers. The level among smokers that had not smoked on the day of the test was 1.1%. The mean COHb level, standardized for the number of cigarettes smoked before the test on the day of the test was 4.0% for self-described noninhalers, 5.2% for slight inhalers, 5.3% for moderate inhalers, and 5.6% for deep inhalers. The increasing trend in COHb levels with extent of inhalation was small but statistically significant. These findings indicate that smokers who say they do not inhale nearly always inhale enough smoke to cause their COHb level to be well above that seen in nonsmokers; and that there is a weak but statistically significant correlation noted between COHb levels and the self-described level of inhaling. They also may help to explain the epidemiolog cal findings that the risk of lung cancer is higher amon self-described noninhalers than nonsmokers and th the association between inhaling and lung cancer i cidence is weak among light smokers. (18 refs)

78-3110 Smoking, Chronic Bronchitis, Bronchiolar Obstruction, and Bronchial Carcinoma. (Ger Scherrer, L. (Pneumologische Abteilung, Medizinische Universitatsklinik, Inselspital, CH-3010 Bern, Switzerland Zeller, C.; Scherrer, M. *Schweiz Med Wochensch* 108(15): 556-559; 1978.

Correlation among smoking, chronic bronchitis, and bronchiolar obstruction were studied in 396 patients (mostly men aged 33-80 yr) with lung carcinoma. Chronic bronchitis (cough and expectoration for > 5 yr) was found in 221 patients (Group 1), but not in the remaining 175 patients (Group 2). The number of heavy (6-20 cigarettes/day) and very heavy (>20 cigarettes/day) smokers and of patients with severe bronchiolar obstruction was significantly higher in Group 1 than in Group 2 ($p < 0.0005$). For both groups combined, however, there was no correlation between the first-minute forced expiratory volume and smoking. In Group 1, patients with severe obstruction included light ($<$ 5 cigarettes/day), heavy, and very heavy smokers in about identical proportions. There were 123 heavy smokers in Group 1: 54 of them had severe bronchiolar obstruction, 69 light obstruction. There were 65 heavy smokers in Group 2: 7 of them had severe obstruction, 58 slight obstruction. The findings suggest that heavy and very heavy smoking for decades seems to protect certain lung cancer patients from chronic bronchitis and bronchiolar obstruction. (16 refs)

78-3111 Effect of a Nitrate Test Meal on Salivary and Gastric Nitrite Levels in Humans. (Fre) Klein, D. (Departemente de Nutrition et des Maladies Metaboliques, Universite de Nancy I, 40, rue Lionnois, 54 000 Nancy, France); Gaconnet, N.; Poullain, B.; Debry, G. *Food Cosmet Toxicol* 16(2): 111-115; 1978.

The effect of a test meal containing 112 mg sodium nitrate on the levels of salivary and gastric nitrite and nitrate was studied in 10 normal subjects (3 men, 7 women: av age 25 yr). The test meal contained 10 g glucose, 5 g corn oil, 10 g powdered milk, 2.2 g polyethylene glycol (PEG), and 25 mg sodium nitrate. Salivary nitrite and nitrate was measured at half hour intervals for 6 hr after ingestion of the meal. Five of the 10 subjects had their saliva aspirated to prevent its passage to the stomach. Gastric samples withdrawn by a sump pump were tested for nitrite and nitrate in both the aspirated and nonaspirated groups. The levels of salivary nitrite rose rapidly during the first half hour after the meal and then slowly attained a max that varied markedly among the bjects. The max level of salivary nitrate was reached 1 hr ter the test meal; it declined thereafter, but at 6 hr had not reached the premeal level. Gastric levels of trate decreased in both groups but the rate of de- ease was slower than that of the PEG (used as a arker of gastric emptying). Gastric nitrite concen- ations did not change in the aspirated group, but in e nonaspirated group they increased rapidly. The ow of salivary nitrite was not sufficient to explain this crease, which may have been due to nitrate reduction by al microflora during esophageal transit. (18 refs)

3-3112 **Mutagenic and Chromosome-Breaking Effects of Azide in Barley and Human Leukocytes.** Eng) Sander, C. (Dept. Plant Pathology, Washington State Jniv., Pullman, WA, 99164); Nilan, R. A.; Kleinhofs, A.; ig, B. K. *Mutat Res* 50(1): 67-75; 1978.

xtensive chromosome analyses in barley were conducted to etermine whether azide mutagenicity could be increased by ncreasing the germination time before mutagen treatment. n addition, a preliminary analysis of the effect of azide on uman WBC chromosomes was made. Azide-induced M_2 arley seedling mutations increased with increasing germina- ion time prior to treatment up to a max mutation frequency t 8 hr (75%) and 16 hr (78%) on a percent mutant events asis or 12 hr (19.7%) on a percent mutant seedling basis. he peak of seedling mutations suggested an azide-sensitive tage between 8 and 16 hr germination. Azide-induced em- ryo lethality and/or ovule sterility, as measured by M_2 seedlings per M_1 spike, increased up to 12 hr and hen decreased with increasing germination. Azide did ot increase the frequencies of chromosome aberrations, centric fragments, or dicentric chromosomes in em- ryonic barley shoots pregerminated for 8 or 12 hr, nicrospores of barley, or human WBC obtained from ormal volunteers. Although azide did have an effect n the general morphology of the human chromosomes, t may have been a physiologic rather than a genetic ffect. The results suggest that azide may act only on eplicating DNA and that it may act only as a point- nutation mutagen. (21 refs)

8-3113 **Effects of Carcinogens on Nuclear Protein Composition and Metabolism. Alterations in Chromatin Structure and Activity as Related to Postsynthet- c Modification of Histones and to Dispositions of DNA-Binding Proteins.** (Eng) Allfrey, V. G. (Rockefeller Jniv., New York, NY); Boffa, L. C.; Vidali, G. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). pp. 29-30; 1978. (no refs)

78-3114 **The Toxicities and Mutagenic Properties of Ethylidene Gyromitrin and N-Methylhydrazine with *Escherichia coli* as Test Organism.** (Eng) Von Wright, A. (Food Res. Lab., Technical Res. Centre Finland, Biologinkuja 1, SF-02150 Espoo 15, Finland); Niskanen, A.; Pyysalo, H. *Mutat Res* 56(2): 105-110; 1977.

The toxicities and mutagenic properties of ethylidene gyromitrin (EG), the main poisonous substance of the false morel (*Gyromitra esculenta*), and N-methylhydrazine (NMH), the possible metabolic end product of EG, were studied using *Escherichia coli* as the test organism. NMH (20 μg) was strongly bacteriocidal, but EG showed no toxicity. In liquid culture, 3 μg NMH/ml nutrient broth caused growth inhibition; EG had no effect. Tests with repair-deficient strains indicated that they were more sensitive to NMH than repair-proficient strains. In revertant tests, NMH caused a dose-dependent increase in revertants at concentrations of 5-20 μg/ml. No increase in revertant frequency was noted at EG concentrations of up to 250 μg/ml. These results suggest that NMH could be responsible for the toxic effects of EG. (9 refs)

78-3115 **The Effect of Expression Time on the Frequency of Carcinogen-induced Mutants at the TK and HGPRT Loci in L5178Y Mouse Lymphoma Cells (Meeting Abstract).** (Eng) Brown, M. M. (Univ. North Carolina, Chapel Hill, NC, 27514); Clive, D. *Mutat Res* 53(1): 116-117; 1978. (no refs)

78-3116 **Effect of Schedule of 1,2-Dimethylhydrazine Administration on Its Carcinogenicity.** (Rus) Pozharisskii, K. M. (N. N. Petrov Res. Inst. Oncology, Leningrad, USSR); Vokhmianin, V. N.; Kulakov, N. A.; Balanski, R. M.; Likhachev, A. Ia.; Pemrov, A. C., Shaposhnikov, Ia. D. *Biull Eksp Biol Med* 85(3): 337-340; 1978.

The effect of the schedule of 1,2-dimethylhydrazine (DMH) administration on its carcinogenicity was studied in random-bred albino rats. The animals were divided into three groups: Group 1 received daily sc injections of DMH (3 mg/kg/injection), Group 2 received DNH 1 x/wk (21 mg/kg/injection) and Group 3 received DMH 2 x/wk (42 mg/kg/injection). Animals in all three groups received approx equal total doses of DMH (528, 514, and 524 mg/kg, respectively). Mice from Group 1 developed tumors in the large intestine alone, but mice from Groups 2 and 3 developed multiple tumors in various segments of the intestine (the extent of tumor dissemination was less pronounced in Group 3). The cytochrome P450 level in the liver microsomes was decreased significantly in Group 1 (2.48 IU, compared to 5.32 IU in untreated controls and 5.1 IU in Group 2). (9 refs)

78-3117 **Inhibition of 1,2-Dimethylhydrazine-induced Neoplasia of the Large Intestine in Female CF$_1$ Mice by Carbon Disulfide: Brief Communication.** (Eng) Wattenberg, L. W. (Dept. Laboratory Medicine and Pathology, Medical Sch., Univ. Minnesota, Box 609, Mayo Memorial Building, Minneapolis, MN, 55455); Fiala, E. S. *J Natl Cancer Inst* 60(6): 1515-1517; 1978.

The effect of carbon disulfide (CS$_2$) on 1,2-dimethylhydrazine (DMH)-induced carcinogenesis was studied in female CF$_1$ mice. Mice were given either 5.0 or 2.5 mg CS$_2$ in corn oil or corn oil only by gastric intubation. A fourth group did not receive anything at this time. Four hours later, all mice received 0.4 mg DMH sc. This dose was repeated at 1-wk intervals for 16 wk, except that upon the last eight administrations, the dose was increased to 0.6 mg. The experiment was ended 40 wk after the first DMH dose. Of 20 mice that had received DMH only, half had tumors of the large intestine or anus. Of 19 that had received the corn oil plus DMH, 10 had tumors of the large intestine and anus. In contrast, none of the 20 given 2.5 mg CS$_2$ + DMH and none of the 19 given 5.0 mg CS$_2$ + DMH had large intestinal or anal tumors. These results indicate that CS$_2$ inhibits DMH-induced tumors of the large intestine and anus. (14 refs)

78-3118 **Formylhydrazine Carcinogenesis in Mice.** (Eng) Toth, B. (Eppley Inst. Res. in Cancer, Univ. Nebraska Medical Center, Omaha, NB, 68105). *Br J Cancer* 37(6): 960-964; 1978.

The tumorigenicity of formylhydrazine (FH) was studied in Swiss mice who were given 0.125% FH in their drinking water for life beginning at 6 wk of age. Compared with untreated controls, survival was shortened significantly in the treated mice. Of the treated females, 47 (94%) developed 531 lung tumors: 149 adenomas in 23 mice and 331 adenomas and 51 adenocarcinomas in 24 mice. The first tumor was observed during wk 43, and the av age at death was 68 wk. Fifty (100%) of the treated males developed 400 lung tumors: 114 adenomas in 26 mice and 237 adenomas and 49 adenocarcinomas in 24 mice. The first tumor was seen during wk 37, and the av age at death was 64 wk. The incidence of lung tumors in the untreated controls was 15% in females and 22% in males; the difference in tumor incidence between the treated and untreated mice was highly significant ($p < 0.0001$). The treatment had no detectable tumorigenic effect in other organs. (20 refs)

78-3119 **Early Proliferative Defects Induced by Six Weekly Injections of 1,2-Dimethylhydrazine in Epithelial Cells of Mouse Distal Colon.** (Eng) Deschner, E. E. (Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY, 10021). *Z Krebsforsch* 91(2): 205-216; 1978.

Early histological and kinetic response of the colonic mucosa of CF$_1$ mice to 1,2-dimethylhydrazine (DMH) have been followed daily after just one injection and then weekly after 5 additional treatments. DMH was injected subcutaneously at a dose of 20 mg/kg body wt. To compensate for dramatic cell loss at 48 hr, marked elevation of the ^{3}H(TdR) labeling index with widening of the proliferation zone to the luminal surface of crypts occurred 2-4 days after initial treatment. At the fourth post-injection day, the regenerating mucosa showed decreased mucin production and structural alterations in crypts such as bifurcations and budding. Recovery of normal crypt histology, crypt cell number, mitotic frequency and labeling index was completed by 7 days. Focal atypia restricted to single isolated crypts were present afte 3 injections of the carcinogen. Replacement of lethall damaged cells 2-4 days after initial injection involve DNA synthesis in an increased number of epithelia cells in the mid portion of colonic crypts with som activity of cells in the upper crypt. This expansio of the proliferative compartment in the mid and upper portions of the crypts which has previously been noted in large bowel mucosa and some patients with a history of colorectal cancer was again evident a wk following the fourth, fifth and sixth DMH injections, indicating the existence of more permanent defect in the regulatory control of cell proliferation at a time when areas of focal atypia were increasing in number. (no refs)

78-3120 **Diet and Its Effect on Enzymes Linked to Colon Cancer (Meeting Abstract).** (Eng) Goldin, B. R. (Tufts Univ. Sch. Medicine, Boston, MA); Gorbach, S. L. *Digestion* 16(3): 240; 1977. (no refs)

78-3121 **Agents Promoting Carcinogenesis in Dimethylhydrazine-initiated Colonic Cancer in Rats (Meeting Abstract).** (Eng) Lewin, M. R. (Surgical Unit, Univ. Coll. Hosp. Medical Sch., London, England); Cruse, J. P.; Ngui, C. F.; Clark, C. G. *Br J Surg* 65(5): 369 1978. (no refs)

8-3122 **Malignant Degeneration of Experimental Ulcerative Colitis (Meeting Abstract).** (Ger) nipper (Hamburg, W. Germany); Dahm; Mitschke *entralbl Chir* 103(6): 377-378; 1978. (no refs)

78-3123 **Studies on N-Nitroso Derivatives of N-Methylcarbamate Insecticides (III). Mutagenicity of N-Nitroso Derivatives of 3-Methylphenyl N-Methylcarbanate, 3,4-Dimethylphenyl N-Methylcarbamate and Naphhyl N-Methylcarbamate for *Salmonella typhimurium*.** Eng) Sakai, A. (Natl. Inst. Hygienic Sciences, 18-1, amiyoga 1-chome, Setagaya-ku, Tokyo, Japan); Yoshiawa, K.; Kurata, H.; Tanimura, A. *J Food Hyg Soc Jpn* 9(1): 122-125; 1978.

3-Methylphenyl N-methylcarbamate, 3,4-dimethylphenyl N-methylcarbamate, and naphthyl N-methylcarbamate and heir N-nitroso derivatives were tested for mutagenicity in *Salmonella typhimurium* strains TA100 and TA98. In the absence of a metabolic activation system (a rat liver microsome preparation, S-9 mix), the nitroso derivatives were mutagenic for strain TA100 but not for strain TA98, indicating that the mutations were due to base-pair substitutions. The S-9 mix inactivated the mutagenicity of the nitroso derivatives for TA100. With or without the S-9 mix, the N-methylcarbamate insecticides were nonmutagenic for TA100 and TA98. (17 refs)

78-3124 **Changes in Liver Nuclear Protein Metabolism after a Single Dose of Urethane to Suckling Mice.** (Eng) Gronow, M. (Cancer Res. Unit, Dept. Biology, Univ. York, Heslington, York Y01 5DD, England); Lewis, F. A. *Chem Biol Interact* 19(3): 327-338; 1977.

The rate of synthesis and the composition of liver nuclear proteins were examined for up to 24 hr after the sc injection of a single carcinogenic dose (1.2 mg/g body wt) of urethane into 8- to 9-day-old C57BL/A mice. There was an immediate decrease in liver DNA synthesis that reached a max 16-18 hr after injection and returned to normal after 48 hr. When the nuclear proteins were radiolabeled, the nonhistone protein (NHP) fraction showed a significant decrease in specific activity 8-18 hr after injection of urethane and a slight increase in specific activity after 24 hr. Histone and residual proteins did not show any significant change. Analysis of liver NHP by electrophoresis showed no distinctive differences following urethane injection. Isoelectric focusing, however, indicated that urethane induced some changes in protein content and synthesis, the most noticeable of which were an increase in a protein component having an isoelectric point (pI) of 7.35 and the appearance of new bands at pI's of 7.85 and 5.55 in the 18-hr-treated livers. The ^{3}H tryptophan labeling pattern indicated that the changes were not due to an increased synthesis of these components. Twenty-four hours after urethane injection, there appeared to be an increased rate of NHP synthesis, particularly the protein component with a pI of 5.65. No differences were detected by electrophoresis in the histone and residual protein fractions of treated and control livers. (27 refs)

78-3125 **Studies on N-Butyl-N-nitrosourethan-induced Cancers of the Esophagus and the Forestomach of Rats.** (Eng) Yasumoto, T. (Second Surgical Div., Yamaguchi Univ. Sch. Medicine, Ube, Umaguchi 755, Japan). *Arch Jpn Chir* 47(2): 156-168; 1978.

Donryu rats were treated with N-butyl-N-nitrosourethan (BNUR: 0.04% in the drinking water for 20-40 wk) and then with copper-containing bleomycin (BLM: 250 mg/kg as a single im injection, or 2 mg/kg im 2x/wk for 8 wk), and the distribution of BLM was studied in the esophagus and in the resulting squamous cell carcinomas. BNUR alone induced multiple papillomas in the esophagus and forestomach in all treated rats and squamous cell carcinomas in 26/29 rats. Twenty-four of the cancers were well-differentiated squamous cell carcinomas, and 2 were poorly differentiated. One of the most prominent features of these cancers, none of which metastasized, was the occurrence of multiple tumors extending from the esophagus to the forestomach. In rats given a single dose of BLM, the compound was found in greater concentrations in the cancer lesions than in the normal esophagus or papillomas; the distribution in the well- and poorly differentiated carcinomas did not differ. Under the polarizing microscope, the tonofibrils in the cells glittered. The tonofibrils in the well-differentiated carcinomas, but not in the poorly differentiated tumors, had the same appearance as those in normal squamous epithelium. Multiple BLM injections were fairly effective on the BNUR-induced carcinomas, although vacuolation and pyknosis of the nucleus and eosinophilic changes in the cancer cells were observed. The BLM-treated cancer pearls glittered more than the untreated cancer pearls under the polarizing microscope. (27 refs)

78-3126 **Effect of Implantation of Embryonal Liver Cells on Antibody Formation and Urethane-induced Carcinogenesis.** (Rus) Kraskovskii, G. V. (Inst. Genetics and Cytology, Minsk, USSR); Kagan, L. F. *Dokl Akad Nauk BSSR* 22(2): 181-184; 1978.

The effect of embryonal liver cell implantation on urethane-induced pulmonary carcinogenesis and antibody formation was studied in Af mice (urethane-sensitive) and (Af x C3H/Sn)F1 mice (C3H/Sn line is urethane-resistant). The mice were inoculated ip with urethane (1 mg/g) and, 1 day later, with 50 x 10^6 Af or C3H/Sn embryo liver cells. On the next day, animals were immunized with sheep RBC (100 x 10^6). Although the liver cell injection normalized urethane-inhibited antibody formation (hemagglutinin titer was 7.0 vs 5.6 in animals treated with urethane alone), it did not prevent urethane-induced pulmonary carcinogenesis. The av number of adenomas per mouse was 9.5, compared with 9.3 in mice inoculated with urethane alone. (10 refs)

78-3127 **Increased Activity of Rat Liver Messenger RNA and of Albumin Messenger RNA Modulated by Thioacetamide.** (Eng) Chakrabartty, P. K. (Lab. Pathophysiology, NCI, NIH, Bethesda, MD, 20014); Schneider, W. C. *Cancer Res* 38(7): 2043-2047; 1978.

The daily ip administration of 0.2 ml thioacetamide, a hepatocarcinogen, to male Sprague-Dawley rats increased the activity of liver messenger RNA, as determined by the wheat germ protein synthesis assay. The synthesis of total protein was increased by about 2.5-fold after 4 days of treatment. The treatment also increased the relative quantity of polyadenylic acid-containing RNA. Immunoprecipitation studies showed that albumin synthesis directed by messenger RNA was increased disproportionately, reaching a level five to six times the control level after 4 days of thioacetamide administration. (24 refs)

78-3128 **Carcinogens Inhibit DNA Synthesis with Isolated DNA Polymerases from *Escherichia coli.*** (Eng) Berthold, V. (Max-Planck-Institut fur medizinische Forschung, Abteilung Molekulare Biologie, Jahnstrasse 29, 6900 Heidelberg, W. Germany); Thielmann, H. W.; Geider, K. *FEBS Lett* 86(1): 81-84; 1978.

The effects of N-acetoxy-2-acetylaminofluorene (N-AAF) and N-methyl-N-nitrosourea (MNU) on DNA synthesis were determined using DNA polymerases isolated from *Escherichia coli.* DNA polymerase III holoenzyme was inhibited by 85% when RNA-primed single-stranded fd DNA was preincubated with N-AAF for 1 hr. Only 12% inhibition was observed when carcinogen, DNA polymerase III, and deoxynucleoside triphosphates were added simultaneously. Preincubation of the single-stranded DNA with increasing concentrations of N-AFF resulted in increasing inhibition of DNA synthesis. DNA polymerase III was inhibited 50% with 5 μM N-AAF; DNA polymerase I was inhibited less at the same concentration. Complex formation of fd DNA with the DNA-binding protein I prior to addition of 15 μM N-AAF reduced DNA synthesis by only 7%, compared with 75% with free DNA. Preincubation of the RNA-primed DNA template in 10 mM NMU for 10 min led to 70% inhibition; 26% inhibition was noted when polymerization was allowed to proceed after MNU addition. DNA polymerase III holoenzyme was slightly more sensitive to MNU than DNA polymerase I. (20 refs)

78-3129 **The Sebaceous-Gland Test.** (Eng) Longstaff, (Imperial Chemical Industries Ltd., Centr Toxicology Lab., Alderley Park, Cheshire, England). *Br Cancer* 37(6): 944-948; 1978.

The sebaceous gland test was evaluated as a short-term te for predicting carcinogenic potential. One hundred an twenty test compounds were dissolved singly in dimethyl su foxide and applied bid for 3 days to the shaved dorsolumb skin of male specific-pathogen-free Swiss-derived Alderl Park mice. A statistically significant reduction in the rat of sebaceous glands to hair follicles was taken to indica carcinogenic potential. A comparison of the percentage glands to follicles of control and treated skins demonstrat that the sebaceous glands of mice were sensitive to the actic of some chemicals. The test was 65% accurate in predictin carcinogenicity. When compounds giving false results we retested using a range of doses, only cyclophosphamid 4-aminobiphenyl, and 2-acetylaminofluorene could be adde to the list of identifiable carcinogens. Mouse skin sebaceo glands seemed insensitive to the action of benzidin o-dianisidine, dimethylnitrosamine, hexamethylphosphor mide, and urethane. The predictive value of the test was pa ticularly good for polycyclic aromatic hydrocarbons. Howe er, of 29 compounds causing >50% suppression of th glands, six were false-positives. It is concluded that this tec nique is not valid for comparing chemicals of different class for potency. (8 refs)

78-3130 **Loss of Adenylate Cyclase Hormonal Sensitivi ty in Chemically Transformed Epithelial Cell** (Eng) Makarski, J. S. (Dept. Surgery, Boston Univ. Sc Medicine, Boston, MA, 02118); Niles, R. M. *Exp Cell Re* 114(1): 191-195; 1978.

The hormonal sensitivity of adenylate cyclase from a norma rat liver epithelial cell line (K16) and it N-acetoxy-N-acetylaminofluorene-transformed derivativ (WB) was compared. Intact normal rat liver cells ha markedly increased cAMP levels after brief exposure to epi nephrine, isoproterenol, norepinephrine, or prost glandin E_1. The cAMP level rose very rapidly a reached a peak around 15 min, declining slightly 30 min. In contrast, the cAMP levels of chemical transformed cells were relatively unaffected by the same compounds, even after incubation for 3 hr. T basal adenylate cyclase activity of broken K-16 ce was six times higher than that of broken W-8 cells. T response to NaF was similar in the two cell lines, suggestin that the catalytic portion of the W-8 enzyme is not signi cantly altered. The response to catecholamines and prost glandins paralleled the intact cell studies. These data sugge that the W-8 cells have fewer or altered hormone bindin sites. It is also suggested that alteration in the function adenylate cyclase may be one way by which cells escape exte nal growth regulation. (11 refs)

8-3131 **Studies on the Transformation of Rat Embryo Cells of Low Passage by Carcinogenic Fluore- ylhydroxamic Acids and Their Acetate Esters.** (Eng) Kur- epa, H. (Lab. Cancer Res., Veterans Admin. Hosp., Min- eapolis, MN, 55417); Gutmann, H. R.; Malejka-Giganti, D.; rauss, A.; Cervenka, J.; Vosika, G. J.; Rydell, R. E. *In Vitro* 4(3): 261-270; 1978.

istar-Furth rat embryo cells of low passage were treated ith a single dose of certain carcinogenic fluorenylhydroxam- c acids and their respective acetate esters and examined for igns of in vitro transformation. Dishes of 10^4 cells ere exposed to 50 or 24 nanomoles (nmol) N-hydroxy- -fluorenylacetamide (N-hydroxy-2-FAA), 25 or 12 mol N-hydroxy-2-fluorenylbenzamide (N-hydroxy-2- BA), 50 or 25 nmol N-hydroxy-3-FAA, 50 or 25 mol N-acetoxy-2-FAA, 25 or 12 nmol N-acetoxy- -FBA, or 50 or 25 nmol N-acetoxy-3-FAA. All treated ell lines showed at least some signs of transformation, such s changes in phenotype, growth in soft agar, and agglutina- ion with concanavalin A. Furthermore, some changes in aryotype and loss of diploidy were observed. Electron mi- roscopy of the cells following chemical treatment revealed o viruslike particles; reverse transcriptase assay confirmed hese findings. Cells from these lines were then injected either c into newborn Wistar-Furth rats or im into weanling rats f the same strain. None of the inoculations resulted in tumor ormation. These findings suggest that malignant transforma- ion is a multistep process and that certain criteria of transfor- ation of rat embryo cells are associated with the initial tage(s) in which the cells are transformed without eing tumorigenic. (34 refs)

78-3132 **In Vitro Metabolism and Mutagenic Activation of 2-Acetylaminofluorene by Subcellular Liver Fractions from Cotton Rats.** (Eng) Schut, H. A. (Div. Cancer Treatment, NCI, NIH, Bethesda, MD, 20014); Thorgeirsson, S. S. *Cancer Res* 38(8): 2501-2507; 1978.

Biochemical parameters associated with in vivo 2-acetylaminofluorene (AAF) carcinogenesis and in vitro AAF mutagenesis in Salmonella were evaluated in subcellular liver fractions of the Cotton rat, a species resistant to AAF carcinogenesis. Low concentrations (1 μg/plate) of AAF or N-hydroxy-AAF (N-OH-AAF) were highly mutagenic (> 1,000 colonies/plate) in the presence of low concentrations (0.4 mg/plate) of microsomal protein. Deacetylation and mutagenicity of AAF and N-OH-AAF were partially inhibited by 0.001-0.1 μM paraoxon and completely inhibited by 100 μM, indicating involvement of a microsomal deacetylase in the mutagenic activation of AAF and N-OH-AAF. The activity of N-OH-AAF sulfotransferase in Cotton rat livers was one-third that of the Sprague-Dawley rat and it decreased cytosol-mediated N-OH-AAF mutagenesis 45%, indicating that this enzyme is not important in the mutagenic activation of N-OH-AAF. AAF mutagenesis with microsomes from untreated animals was much more sensitive to inhibition by 1-10 μM α-naphthoflavone (ANF) than that with microsomes from 3-methylcholanthrene (MC)-treated animals, and at 50 μM ANF this process was inhibited completely in both cases. AAF N-hydroxylase from untreated animals also exhibited a greater sensitivity to ANF (1-10 μM) than that of MC-treated animals, but the difference was much less pronounced; at 500 μM ANF, this difference was reversed, with the enzyme from MC-treated animals showing greater inhibition (90% vs 60%). Inhibition of aryl hydrocarbon hydroxylase required ANF concentrations > 10 μM, and the enzyme from MC-treated animals was more sensitive to this agent (60% inhibition) than that of untreated animals (35%). The Cotton rat appears to possess all the known enzyme systems to activate AAF into an ultimate carcinogen or mutagen. Exploration of other biochemical properties, such as interaction of AAF with subcellular macromolecules or in vivo AAF metabolism, may provide further insight into the resistance of this species to AAF carcinogenesis. (48 refs)

78-3133 **Gangliosides of Liver Tumors Induced by N-2-Fluorenylacetamide. II. Alterations in Biosynthetic Enzymes.** (Eng) Merritt, W. D. (Section Cytology, Yale Univ. Sch. Medicine, New Haven, CT, 06510); Morre, D. J.; Keenan, T. W. *J Natl Cancer Inst* 60(6): 1329-1337; 1978.

Activities of the enzymes of the monosialoganglioside pathway were studied in male Wistar rats fed a low-protein basal diet with or without 0.05% N-2-fluorenylacetamide. Livers, hyperplastic nodules, and hepatomas were analyzed throughout a 23-wk period of tumorigenesis. An early twofold elevation of monosialoganglioside G-M3 levels in precancerous tissues and nodules correlated with an early twofold increase in the specific activity of cytosine monophosphate-N-acetylneuraminic acid:lactosylceramide sialyltransferase of G-M3 biosynthesis. This increase was followed by increases in specific activities of the galactosyltransferases and sialyltransferases of G-M1 and G-D1a biosynthesis in well-differentiated hepatomas and in well-circumscribed, poorly differentiated hepatomas. The result was a thirtyfold increase in the products of the monosialoganglioside pathway (G-M1 + G-D1a) relative to the sum of the products of the disialoganglioside pathway (G-D3 + G-X). The property of invasiveness may be associated with this increasing ratio. (23 refs)

78-3134 **Toxicity and Frequency of Sister Chromatid Exchanges Produced by N-hydroxy-2-acetylaminofluorene (N-OH-AAF) in Human Lymphocytes (Meeting Abstract).** (Eng) Altenburg, L. C. (Univ. Texas Health Science Center, Medical Genetic Center, Houston, TX, 77030); Ray, J. H.; Jacobs, M. M. *Mutat Res* 53(1): 74-75; 1978. (no refs)

78-3135 **Unscheduled DNA Synthesis in Spermatogenic Cells of Mice Treated In Vivo with the Indirect Alkylating Agents Cyclophosphamide and Mitomen.** (Eng) Sotomayor, R. E. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Sega, G. A.; Cumming, R. B. *Mutat Res* 50(2): 229-240; 1978.

The effects of cyclophosphamide (CP) and mitomen (2,2′-dichloro-N-methyldiethylamine N-oxide: DMO) on DNA repair were studied in meiotic and postmeiotic germ cells of male (C3Hf x 101)F_1 mice using an in vivo unscheduled DNA synthesis (UDS) assay. Ethylmethanesulfonate (EMS), a compound that does not require metabolic activation, was also studied for comparison. Both CP and DMO (200 and 40 mg/kg ip, respectively) induced UDS in the meiotic through early-to-midspermatid stages, but no UDS was detected in late spermatids and mature sperm. EMS (200 mg/kg ip) produced a max UDS response in the germ cells immediately after treatment; CP and DMO did not produce a max response until approx 0.5-1.0 hr after injection. This delay is attributed to the time required for CP and DMO to be enzymatically activated to alkylating metabolites. Unlike the results found with EMS, mutation frequencies (dominant lethals, translocations, and specific-locus mutations) following CP treatment were not noticeably reduced in the germ cell stages in which UDS occurred. With DMO, mutations were induced only in mature spermatozoa, and these germ cell stages represented only a fraction of those in which no UDS was detected. These findings do not resolve the ambiguity of the relationship between DNA repair and the differential spermatogenic response of mice to genetic damage. (31 refs)

78-3136 **Does Cyclophosphamide Induce Bladder Cancer?** (Eng) Pearson, R. M. (Dept. Urology, Univ. Tennessee Center for the Health Sciences, Memphis, TN, 38163); Soloway, M. S. *Urology* 11(5): 437-447; 1978.

The case report of a 40-yr-old man with malignant lymphoma who subsequently developed adenocarcinoma of the urinary bladder is presented. Treatment of the lymphoma included the administration of 3 g of cyclophosphamide; this is the first reported case of an adenocarcinoma following cyclophosphamide therapy. The 13 previously reported cases of bladder tumors following cyclophosphamide therapy are reviewed. (91 refs)

78-3137 **Chromosome Aberrations Induced by Monomeric Acrylamide in Bone Marrow and Germ Cells of Mice.** (Eng) Shiraishi, Y. (Dept. Anatomy, Kochi Medical Univ., Nangoku City, Kochi 781-51, Japan). *Mutat Res* 57(3): 313-324; 1978.

The chromosomes of the marrow, spermatogonia, and primary spermatocytes of male DDY mice were examined quantitatively after the mice were treated with acrylamide (AA) po (500 ppm of diet for 1, 2, or 3 wk) or ip (single dose of 100, 150, or 200 mg/kg). Samples were taken at various times after treatment. A comparatively high frequency of chromatid exchanges and breaks was induced in the spermatogonia of the 3-wk po mice only (19% vs 2.4% in controls). However, the frequency of chromosome aberrations was not increased appreciably in the marrow cells of any of the mice. The number of mitotic cells was reduced significantly in the spermatogonia of all treated mice, especially 12 and 24 hr after the ip injections. Thus, it was not possible to examine the acute cytogenetic effects of AA on spermatogonia. The frequency of aneuploid and polyploid cells increased with time after treatment in both the marrow and spermatogonia cells. AA induced a significant number of chain quadrivalents, ring quadrivalents, fragments, and univalents in the primary spermatocytes, especially in ip mice. There was no noticeable increase in the sister chromatid exchange frequency over control levels in the marrow and spermatogonia cells. AA also reduced the testis wt significantly, which suggests that there is a correlation between AA-induced chromosome aberrations and cell killing. The findings of a high frequency of rearrangements and univalents in the primary spermatocytes is significant, but it is not known whether these aberrations are transmitted to the next generation. (21 refs)

78-3138 **Positive Interferant in the Chemiluminescent Detection of Nitrosamines.** (Eng) Gough, T. A. (Lab. Government Chemist, Stamford St., London SE1 9NQ, England); Webb, K. S. *J Chromatogr* 154(2): 234-237; 1978.

Although chemiluminescence is highly selective in detecting N-nitroso compounds, it is possible to observe a response from other (interfering) compounds at the retention times of volatile nitrosamines. Therefore, the chemiluminescence findings should be confirmed by mass spectrometry. (13 refs)

78-3139 **Structure-Activity Studies on Mutagenicity of Nitrosamines Using Molecular Connectivity.** (Eng) Kier, L. B. (Dept. Pharmaceutical Chemistry, Medical Coll. Virginia, Virginia Commonwealth Univ., Richmond, VA, 23298); Simons, R. J.; Hall, L. H. *J Pharm Sci* 67(5): 725-726; 1978.

The relationship between molecular structure and mutagenicity in the Ames test was investigated using 15 nitrosamines, including alkyl, guanidino, and ureido derivatives. The structure-activity relationships were examined by the molecular connectivity method, which allows the evaluation of structural features influencing physical property values and biological activity. There was a good relationship etween a molecular connectivity description of structure and nutagenic potency. This finding could be used to develop a heoretical prediction of mutagenic potential in discrete hemical classes of molecules. (11 refs)

8-3140 **The Carcinogenic Effect of β Oxidized Dipropylnitrosamine in Mice. II. 2-Hydroxypropyl-N-propylnitrosamine and 2-Oxo-propyl-N-propylnitrosamine.** (Eng) Dickhaus, S. (Abteilung fur Experimentelle Pathologie, Medizinische Hochschule, Hannover, Karl-Wiechert-Allee 9, D-3000, Hannover 61, Germany); Reznik, G.; Green, U.; Ketkar, M. *Z Krebsforsch* 91(2): 189-193; 1978.

The carcinogenic effects of 2-hydroxypropyl-N-propylnitrosamine (2-HPPN) and 2-oxo-propyl-N-propylnitrosamines (2-OPPN) were studied in female NMRI mice. The chemicals were given sc at 0.2, 0.1, and 0.05 of the LD$_{50}$ once weekly for life beginning at 6 wk of age. With 2-HPNN, survival time decreased with increasing dosage, and tumor incidence increased (from 84% to 100%) with increasing survival period. Adenomas, papillomas, adenocarcinomas, and epidermoid carcinomas occurred in the nasal cavities; alveogenic adenomas and adenocarcinomas arose in the lungs; and hemangioendotheliosarcomas, cholangiomas, and one mixed tumor arose in the liver. Survival and tumor multiplicity increased with increasing dose of 2-OPPN. Tumor incidence was 86%-92%. Papillomas and adenocarcinomas arose in the nasal and paranasal cavities; intrabronchial papillomas, alveogenic adenomas, and adenocarcinomas arose in the lungs; and all liver tumors were hemangioendotheliosarcomas, except for two cholangiomas and one mixed tumor. Neither 2-HPPN nor 2-OPPN induced tumors in the nasopharyngeal duct or larynx. The carcinogenicity of the β-metabolites of dipropylnitrosamine cannot be unequivocally associated with the carcinogenic effect of the parent compound. (12 refs)

78-3141 **Detection of N-Nitrosodimethylamine by Gas Chromatography-Methane Chemical Ionization Mass Spectrometry with Single Ion Monitoring.** (Eng) Mach, M. H. (Chemistry and Physics Lab., Ivan A. Getting Labs., Aerospace Corp., El Segundo, CA, 90245); Pallos, A. *Anal Lett* 11(1): 97-102; 1978.

Gas chromatography-methane chemical ionization mass spectrometry with single-ion monitoring was used to detect N-nitrosodimethylamine (NDMA) at concentrations <50 ppb. In water, concentrations as low as 25 ppb could be detected; similar findings were obtained with NDMA in hexane. This method is intended for relatively pure samples known to contain the compound and not for the analysis of NDMA in unknowns. (18 refs)

78-3142 **Lipoperoxidative Effects on Liver Rough Microsomal Membranes and the Ribosome-Membrane Complex.** (Eng) Shires, T. K. (Dept. of Pharmacology, The Toxicology Center, Coll. of Med., Univ. of Iowa, Iowa City, IA, 52242). *Lab Invest* 38(6): 693-702; 1978.

A direct and quantitative assay of the loss of radioactively labeled ribosomes from the membranes of rough microsomal preparations from rat liver was undertaken after the membranes were incubated with carbon tetrachloride (CCl$_4$), dimethylnitrosamine (DMNA), or aflatoxin B$_1$. Incubations were carried out in the presence of NADPH and/or varying concentrations of K^+. None of these compounds fostered the detachment of ribosomes from the membrane in vitro under conditions that supported their metabolism. Lipoperoxidation caused by CCl$_4$ or NADPH did, however, stimulate detachment in the presence of elevated K^+ concentrations (750 mM). Rough membranes isolated from the liver of rats given lethal doses of CCl$_4$, but not DMNA, also responded to high K^+ concentrations with increased ribosome detachment. Three changes in the polysome-membrane complex were elicited by lipoperoxidation: an apparent loosening of the linkage involving peptidyl transfer RNA and the membrane; an inactivation of the ion-sensitive binding sites for the ribosome, as shown by the decreased acceptance of exogenous ribosomes by stripped lipoperoxidized rough membranes; and a site stabilization by stripped ribosome occupation of the binding site at the time of lipoperoxidation. The disappearance of rough endoplasmic reticulum in the livers of animals ingesting compounds such as CCl$_4$ seems to arise from the failure of polysome assembly on membranes of the endoplasmic reticulum rather than from the detachment of active ribosomes from the membranes. (26 refs)

78-3143 **Hydroxyurea-sensitive Thymidine Incorporation in Rat Liver and Kidney Following Administration of Dimethylnitrosamine.** (Eng) Stewart, B. W. (Sch. Pathology, Univ. New South Wales, P.O. Box 1, Kensington, New South Wales, Australia 2033); Brian, M. J. *Cancer Lett* 4(3): 155-161; 1978.

The sensitivity of ^{3}H-thymidine incorporation to inhibition by hydroxyurea (HU) was determined in the livers and kidneys of female Wistar rats treated with dimethylnitrosamine (DMN: 30 or 40 mg/kg ip). In control animals, HU (50-250 mg/kg ip) inhibited thymidine incorporation by 70%. Twelve hours after DMN administration, hepatic thymidine incorporation was unaffected by 50 mg/kg HU, but a progressive reduction in incorporation occurred as the dose of inhibitor was increased. In the kidney, HU-insensitive thymidine incorporation was observed only in rats conditioned by a diet that increased the renal metabolism of DMN. At 72 hr after DMN treatment, thymidine incorporation in both liver and kidney was inhibited $>70\%$ by 50 mg/kg HU. The results support the use of HU to study DNA repair in intact animals. (19 refs)

78-3144 **RNA Metabolism and Poly(A) Distribution in Mouse Liver Following Administration of**

Dimethylnitrosamine. (Eng) Hultin, T. (Wenner-Gren Inst., Univ. Stockholm, S-113 45 Stockholm, Sweden); Eriksson, B.; Nygard, O.; Von Der Decken, A. *Chem Biol Interact* 21(1): 45-58; 1978.

Dimethylnitrosamine (DMNA: 1% soln, 25-75 mg/kg) was administered by stomach tube to NMRI mice, and the effects of the compound on RNA metabolism and on poly(A) distribution in the liver were determined. DMNA inhibited RNA synthesis in the liver markedly, but the intracellular nucleotide pattern, rate of nucleotide synthesis, and phosphorylation ratio were unaffected. In addition, an unidentified, probably nonnucleotide, component in the acid-soluble liver fraction was reduced selectively, and the RNA polymerase activity of isolated liver nuclei was decreased within 45 min. Both Mg^{2+}- and $Mn^{2+}/(NH_4)_2SO_4$-stimulated polymerase activity was decreased. The inhibition of RNA synthesis involved the entire complement of RNA, including poly(A)-containing nuclear and cytoplasmic RNA and isolated RNase-resistant poly(A) sequences. The transfer of labeled RNA from the nucleus to the cytoplasm was not impaired. There was no detachment of poly(A)-containing RNA from the microsomes, and the proportion of tightly membrane-bound microsomal RNA and poly(A) sequences was not reduced, as determined by a flotation technique. Sedimentation analysis showed that there was no breakage or shortening of the poly(A) chains. Even at the high DMNA levels, there was no decrease in the proportion of poly(A)-containing RNA and poly(A) sequences tightly bound to endoplasmic membranes and no evidence of poly(A) degradation. (31 refs)

78-3145 **Qualitative Alterations in Plasma Esterases in BALB/c Mice Following the Administration of Diethylnitrosamine.** (Eng) Davidson, K. A. (Biology Div., Oak Ridge Natl. Lab., P.O. Box Y, Oak Ridge, TN, 37830); Tyndall, R. L.; Clapp, N. K. *Chem Biol Interact* 21(1): 19-27; 1978.

Female BALB/c mice were treated with dimethylnitrosamine (DEN: in the drinking water for 7 wk), and their plasma esterases and those of untreated mice were analyzed qualitatively. Selected nonspecific plasma esterases, separated by polyacrylamide gel electrophoresis, were altered following DEN treatment. These alterations included a decreased preference for naphthyl butyrate as a substrate relative to naphthyl acetate; a decreased sensitivity to enhancement by divalent cations such as Ca^{2+}, Mg^{2+}, and Mn^{2+}; and an increased sensitivity to inhibition by eserine. All the affected esterase species were also altered quantitatively, and some were testosterone-dependent. The differential response of the plasma esterases of the treated mice to Mn^{2+}, eserine, and substrate are interesting in view of the fact that their electrophoretic mobilities were not altered. (23 refs)

78-3146 **Singlet Oxygen Formation During Peroxidase Catalyzed Degradation of Carcinogenic N-Nitrosamine.** (Eng) Duran, N. (Dept. Biochemistry, Universidade de Sao Paulo, Instituto de Quimica, C.P. 20780, Sao Paulo, Brazil); Faljoni, A. *Biochem Biophys Res Commun* 83(1): 287-294; 1978.

Evidence is presented for the formation of singlet oxygen and chemiluminescence during the decomposition of nitrosamines catalyzed by peroxidase. The reaction medium contained 40 mM diisopropyl-N-nitrosamine and 3 μM horseradish peroxidase in 0.1 phosphate buffer, pH 7.4. The singlet oxygen traps 2,5-diphenylfurane and 1,3-diphenylisobenzofurane were oxidized to *cis*-benzoylethylene and *o*-dibenzoylbenzene during the decomposition. Singlet oxygen quenchers such as butylated hydroxytoluene and 3,5-ditertbutyl-4-hydroxybenzylalcohol inhibited this conversion and also the chemiluminescence accompanying the catalyzed reaction. Chemiluminescence was enhanced by 1,4-diazobicyclo(2.2.2)octane, fluorescein, eosin, rhodamine B, and rose bengal, but little effect was detected with 9,10-dibromoanthracene-2-sulfonate, 9,10-diphenylanthracene-2-sulfonate, and anthracene-2-sulfonate. In the absence of an enhancer, an emission spectrum in the region 560-600 nanometers was observed. It is suggested that singlet oxygen formation could play an important role in nitrosamine carcinogenesis. (27 refs)

78-3147 **Changes of the APUD Cell System During Nitrosamine-Induced Pulmonary Carcinogenesis in a Syrian Golden Hamster (Meeting Abstract).** (Ger) Reznik-Schuller, H. (Hannover, W. Germany). *Zentralbl Allg Pathol* 122(1/2): 139-140; 1978. (no refs)

78-3148 **Effect of L-Tryptophan on Diethylnitrosamine and 3′-Methyl-4-N-dimethylaminoazobenzene Hepatocarcinogenesis.** (Eng) Evarts, R. P. (Carcinogen Metabolism and Toxicology Branch, NCI, Bethesda, MD 20014); Brown, C. A. *Food Cosmet Toxicol* 15(5): 431-435; 1977.

The effect of L-tryptophan (TP) on hepatocarcinogenesis in male Wistar rats fed either 3′-methyl-4-N-dimethylaminoazobenzene (3′-MeDAB) or diethylnitrosamine (DENA) was investigated. There were six groups of rats. Group I (20 rats) received no treatment; Group 2 (20 rats) received a diet containing 1% TP; Group 3 (25 rats) received a 0.002% solution of DENA in the drinking water; Group 4 (29 rats) received DENA + TP; tryptophan; Group 5 (29 rats) received 0.05% 3′-MeDAB in the diet; and Group 6 (30 rats) received 3′-MeDAB + TP. DENA was administered for 128 days, 3′-MeDAB for 96 days. Thereafter, basal or 1% TP was given until day 166. No tumors were found in the first two groups. Group 3 contained 22 animals with tumors; 13 anaplastic (A) and 4 well-differentiated

(W-D) hepatocellular tumors and 5 hemangioendothelial sarcomas, plus 5 metastases. Group 4 had 17 animals with tumors: 16 hepatocellular tumors (8A, 8W-D) and 1 hemangioendothelioma sarcoma, plus 6 metastases. Group 5 had 24 animals with hepatocellular tumors, 21 A and 2 W-D, plus 10 metastases. Group 6 had 12 animals with tumors: 10 hepatocellular tumors (5A, 5 W-D) and 2 cholangiomas plus 4 metastases. These findings indicate that TP decreases the incidence of 3′-MeDAB- and DENA-induced liver tumors in rats, but it has no effect on the location and incidence of esophageal tumors in DENA-fed animals. (26 refs.)

78-3149 **Liver Cell-mediated Mutagenesis of Mammalian Cells by Liver Carcinogens.** (Eng) Langenbach, R. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NB, 68105); Freed, H. J.; Huberman, E. *Proc Natl Acad Sci USA* 75(6): 2864-2867; 1978.

A cell-mediated mutagenesis assay was developed in which primary cultures of rat liver cells were used to activate two classes of liver carcinogens, nitrosamines and aflatoxins, and Chinese hamster V79 cells were used to detect their mutagenic metabolites. Mutations in the V79 cells were characterized by resistance to ouabain (oua-r). When the liver and V79 cells were cocultivated in the presence of N-nitrosodimethylamine (DMN), N-nitrosodiethylamine, and aflatoxin B_1, oua-r mutants were induced in the V79 cells. When the liver cells were absent, the carcinogens did not induce oua-r mutants, which suggests that the reactive intermediate(s) of these carcinogens is stable enough to be transferred from the liver cells to the V79 cells. The analogs N-nitrosomethyl-tert-butylamine and aflatoxin G_2 were not mutagenic. Mutation frequency increased with increasing doses of the carcinogens and with increasing numbers of liver cells seeded. Liver cell-mediated mutagenesis may be more sensitive than liver microsome-mediated mutagenesis: with 2×10^6 attached liver cells from noninduced animals, a DMN concentration of only 1.4 mM was required to enhance mutation frequency 90-fold, whereas in other studies, 50 mM DMN was required to obtain a similar increase in the mutation frequency of V79 cells with microsomes from induced animals. The liver cell-mediated assay may be a sensitive assay for screening chemical carcinogens. (37 refs)

78-3150 **Heritable Translocation Test on Random-bred Mice after Prolonged Triethylenemelamine Treatment.** (Eng) Sheu, C. W. (Genetic Toxicology Branch, Div. Toxicology, Food and Drug Admin., Washington, DC, 20204); Moreland, F. M.; Oswald, E. J.; Green, S.; Flamm, W. G. *Mutat Res* 50(2): 241-250; 1978.

Heritable translocation and dominant lethal tests were performed with random-bred Swiss albino male mice that had received triethylenemelamine (TEM) at concentrations of 0.0125, 0.025, or 0.050 mg/kg/day in the drinking water for 4 wk. Following treatment, the mice were mated for 3 successive wk for analysis of dominant lethality and production of F_1 progeny. The dominant lethal effect was statistically significant for offspring of the first 2 wks of mating. Potential translocation carriers among F_1 males were selected after two breedings and confirmed by cytogenetic analysis. Translocation heterozygotes were obtained in offspring from the TEM-treated groups, but not in offspring from control groups. In F_1 males produced from the first week of mating, the frequencies of translocations were 0%, 1.7%, 6.2%, and 10% for the control and 0.0125-, 0.025-, and 0.050-mg/kg/day groups, respectively. The frequencies in F_1 males produced in the third week of mating were 0% and 2.1% for the control and 0.050-mg/kg/day group respectively. F_1 males from the second week of mating were not studied. TEM-induced dominant lethality and heritable translocations were most prominent in the first week of mating after 4 wk of treatment. Heritable translocations appeared to be a more sensitive end point than dominant lethal mutations for measuring the mutagenic effects of TEM. (10 refs)

78-3151 **Effect of Cholecystoduodenostomy and Choledochostomy in Pancreatic Carcinogenesis.** (Eng) Pour, P. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB, 68105); Donnelly, T. *Cancer Res* 38(7): 2048-2051; 1978.

The pancreatic carcinogen N-nitrosobis(2-oxopropyl)amine (BOP) was administered to Syrian golden hamsters after cholecystoduodenostomy and choledochostomy to investigate a possible bile reflux mechanism as a factor in pancreatic carcinogenesis. Ten animals received weekly sc injections of BOP (5 mg/kg) for 20 wk, beginning 1 wk after surgery. Another three hamsters received 5 mg/kg BOP as a single injection at the 11th wk after surgery, and seven hamsters served as untreated controls. All 13 treated hamsters developed pancreatic neoplasms that were classified as adenomas, intraductal carcinomas, and adenocarcinomas. The lesions were similar in morphology, multiplicity, and distribution to those of animals in other studies in which the above surgery was not performed. Consequently, the effect of bile can be excluded as a mediating factor in pancreatic carcinogenesis. In this as well as in previous experiments, most of the induced pancreatic tumors were of ductular origin, and many seemed to develop within or around the islets. The preference for the peri- and intrainsular ductules as sites of cancer induction could be due to a greater exposure of the responsive ductular cells to the blood-borne carcinogen because of a marked vascularity of the islets. This would explain why limited or single BOP doses in previous studies and in this experiment primarily affected the peri- and intrainsular ductules. (9 refs)

78-3152 **Carcinogenicity of Acetoxymethyl-methyl-nitrosamine after Subcutaneous, Intravenous and**

Intrarectal Applications in Rats. (Eng) Habs, M. (Institut Toxikologie und Chemotherapie, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany); Schmahl, D.; Wiessler, M. *Z Krebsforsch* 91(2): 217-221; 1978.

In comparison to the known carcinogenic properties of acetoxymethyl-methyl-nitrosamine (AMMN) after oral or intraperitoneal application the dimethylnitrosamine derivative was tested by subcutaneous, intravenous and intrarectal route in male Sprague-Dawley or Wistar rats. AMMN proved to be primarily a locally acting carcinogen. However, a second mode of action is indicated by systemic carcinogen properties found after iv and sc applications. The lung and heart, and to a less extent the kidney and ear duct were found as target organs of distant carcinogenic response. (no refs)

78-3153 **Urinary Bladder Neoplasms in Syrian Hamsters after Administration of N-Nitroso-N-methyl-N-dodecylamine.** (Eng) Althoff, J. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB, 68105); Lijinsky, W. *Z Krebsforsch* 90(3): 227-231; 1977.

The carcinogenic effect of N-nitroso-N-methyl-N-dodecylamine (NMDA) was examined in Syrian hamsters. NMDA was given intragastrically once a week for life to three groups of hamsters at dose levels corresponding to 0.05, 0.025, and 0.0125 of the LD$_{50}$. Tumors were observed 36 wk after the start of the treatment, and the incidence showed a positive dose-response relationship. The urinary bladder was the main target organ, followed by the lungs. Lung tumors occurred only in female hamsters, whereas males more frequently had neoplasms of the nasal cavity and digestive tract. It appears questionable as to whether urinary metabolites of NMDA can be specifically linked to urinary bladder carcinogenesis. A urogenic mode of action would also not explain the simultaneous occurrence of neoplasms in organs other than the urinary bladder. (13 refs)

78-3154 **Carcinogenicity of Subcutaneously Injected N-Nitrosoheptamethyleneimine in European Hamsters.** (Eng) Reznik, G. (Abteilung fur Experimentelle Pathologie, Medizinische Hochschule Hannover, Karl-Wiechert-Allee 9, 3000 Hannover 61, W. Germany); Mohr, U.; Lijinsky, W. *J Natl Cancer Inst* 61(1): 239-243; 1978.

The carcinogenicity of sc injected (once weekly for life) N-nitrosoheptamethyleneimine (NHMI) was studied in male and female European hamsters (45 of each sex). The hamsters were divided into three groups that received 20%, 10%, or 5% of the LD$_{50}$ (330 mg/kg for males and 220 mg/kg for females). Av survival times decreased with increasing dose levels of NHMI. A high percentage (96%-100%) of lung tumors was observed in all dose groups. They were histologically diagnosed as adenomas, adenocarcinomas, squamous cell carcinomas, or mixed cell carcinomas, with the squamous cells lying centrally in the tumor. Nasal cavity tumors developed in 100% of all treated males and females; 78% of these tumors were malignant (squamous cell carcinomas), and they frequently infiltrated and destroyed the nasal and maxillary bone. As many as 53% of the treated animals developed small squamous cell papillomas in the forestomach. Five animals had squamous cell carcinomas of the forestomach. One tracheal papilloma and one laryngeal squamous cell carcinoma were seen in two males from the group given the highest dose of NHMI. These results together with those of earlier investigations suggest an influence of the mode of application of NHMI on the tumor response in European hamsters. (10 refs)

78-3155 **Mutagenicity of Nitrosourea Compounds for *Salmonella typhimurium*: Brief Communication.** (Eng) Auletta, A. E. (JRB Associates, Inc., 8400 Westpark Dr., McLean, VA, 22101); Martz, A. G.; Parmar, A. S. *J Natl Cancer Inst* 60(6): 1495-1497; 1978.

The nitrosourea derivatives 1,3-bis(2-chloroethyl)-1-nitrosourea (BCNU), 1-(2-chloroethyl)-3-cyclohexyl-1-nitrosourea (CCNU), 1-(2-chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea (Me-CCNU), and 1-methyl-1-nitrosourea (MNU) were tested for mutagenicity in vivo and in vitro using *Salmonella typhimurium* strains G46 and TA1530. In vitro tests of these compounds at concentrations of 1-1,000 μg/plate indicated that all four were mutagenic; MNU, CCNU, and Me-CCNU were also mutagenic in the host-mediated assay in Swiss albino mice. 3-(1-Adamantyl) -1- (2-chloroethyl) -1- nitrosourea, 3-(1-adamantyl)-1-(2-fluoroethyl) -1- nitrosourea, 1,1′- (4,4′-biphenylylenedimethylene)-bis[3- (2-chloroethyl)] -3-nitrosourea, and 1,1′-(1,4-cyclohexylenedimethylene)bis[3-(2-chloroethyl)] -3-nitrosourea (NSC-120337) were also tested for in vitro mutagenicity at concentrations of 1-1,000 μg/plate using strain G46. All compounds were mutagenic except for NSC-120337. However, this may have been an indication of the instability of NSC-120337 in soln rather than a true reflection of its mutagenic potential. All eight compounds were then tested in a repair test with *S. typhimurium* strains TA1978 and TA1538. The results of this test were negative, reflecting the insensitivity of these strains to ethylating and methylating agents. The insensitivity of the host-mediated assay is also discussed. (16 refs)

78-3156 **Modulation of Carcinogen Chromatin-DNA Interaction by Polyamines.** (Eng) Rajalakshmi, S. (Dept. Pathology, Univ. Toronto, Medical Sciences Building, Toronto, Ontario M5S 1A8, Canada); Rao, P. M.; Sarma, D. S. *Biochem Biophys Res Commun* 81(3): 936-940; 1978.

The effect of polyamines on the methylation of chromatin DNA in vitro by N-methyl-N-nitrosourea (MNU) was investigated. Hepatic chromatin was isolated from male Fischer rats and exposed to 4 ml of a soln containing 2 micromoles (μmol)/ml MNU for 2 hr. The addition of 0.0125, 0.025, and 0.05 μmol spermine/ml incubation mixture containing 150-200 μg of chromatin DNA inhibited DNA methylation by 27%, 56%, and 86%, respectively. Similar results were obtained when rat liver DNA was substituted for liver chromatin. Spermidine had a similar effect, but putrescine was less effective (50% inhibition at 0.1 μmol/ml). At about 86% inhibition, methylation at the N^3 position of adenine was inhibited by 100% and that at the N^7 or O^6 position of guanine was inhibited by 80%. However, spermine did not inhibit the methylation of 2-deoxy-5′-guanilic acid included as an internal control. Further studies indicated that the inhibition of MNU-induced methylation of DNA by polyamines was not due to a suppression of the release of active methyl groups from MNU during its decomposition in the incubation mixture. The polyamines modulate carcinogen-DNA interaction by altering the net charge and/or the conformation of DNA. (15 refs)

78-3157 **The Effect of Chloramphenicol on the Toxic and Therapeutic Activity of N-Nitrosomethylurea.** (Rus) Bogush, T. A. (Lab. Chemical Carcinogenesis, Cancer Res. Center, Moscow, USSR); Konopleva, I. A.; Shabad, L. M. *Vopr Onkol* 23(12): 66-70; 1977.

The effect of the antibiotic chloramphenicol (CAP) on the toxicity and therapeutic activity of N-nitrosomethylurea (NMU) was studied in CBA mice. Two series of experiments were carried out. In the first series, mice were given CAP (1 mg/g, po, for 5 days); 2 hr after the last CAP administration, animals were inoculated sc with NMU (0.05-0.1 mg/g). All control mice (treated with NMU alone) died by day 14 after injection; NMU + CAP provided 65% survival. In the second series, mice were inoculated ip with 2.5 x 10^6 Ehrlich ascites carcinoma cells; on the next day, some of the animals started to receive CAP (1 mg/g/day, po). Five days later all animals were inoculated with NMU (0.05-0.075 mg/g, sc). The av duration of survival of mice treated with NMU alone and with NMU + CAP was increased by 2-3 days, compared with untreated controls. CAP administration decreased the number of tumor cells in the ascites fluid: on day 6 after carcinoma transplantation, mice treated with NMU + CAP had approx 1.5 x 10^6 tumor cells, compared with 3.5 x 10^6 cells in mice treated with NMU alone and approx 4 x 10^6 cells in untreated controls. (13 refs)

78-3158 **Measurements of DNA Damage in Chinese Hamster Cells Treated with Equi-toxic and Equi-mutagenic Doses of Nitrosoureas (Meeting Abstract).** (Eng) Erickson, L. C. (Lab. Molecular Pharmacology, Developmental Therapeutics Program, DCT, NCI, NIH, Bethesda, MD, 20014). *Proc Am Assoc Cancer Res* 19: 210; 1978. (2 refs)

78-3159 **Genetic Basis of Susceptibility for Development of Neoplasms Following Treatment with N-Methyl-N-nitrosourea (MNU) or X-Rays in the Playfish/-Swordtail System.** (Eng) Schwab, M. (Genetisches Institut der Justus Liebig-Universitat Giessen, Heinrich-Buff-Ring 58-62, D-6300 Giessen, W. Germany); Haas, J.; Abdo, S.; Ahuja, M. R.; Kollinger, G.; Anders, A.; Anders, F. *Experientia* 34(6): 780-782; 1978.

The susceptibility of 65 defined genotypes, including nonhybrids, F$_1$ hybrids, and backcross hybrids, of xiphophorine fish to tumor induction by x-rays or N-methyl-N-nitrosourea (MNU) was analyzed to determine the role of genetic factors. Both 6-wk- and 6-mo-old fish were exposed in tanks to an aqueous soln (10^{-3} M) of MNU for 1 hr at 2-wk intervals for a total of four treatments. Additional fish underwent whole-body irradiation (1,000 R for 45 min, 3 treatments at 6-wk intervals). Tumor development was age-dependent, with the younger group being much more susceptible. Benign and malignant melanoma, neuroblastoma, and fibrosarcoma were the most prevalent tumor types. Other tumor types included squamous cell carcinoma, epithelioma, adenocarcinoma of the kidney and thyroid, hepatoma, rhabdomyosarcoma, and lymphosarcoma. The neoplasms developed almost exclusively in the backcross hybrids. Certain genotypes carrying specific chromosomes, which could be identified by phenotypic markers, were susceptible to tumor development, but others lacking these chromosomes were not susceptible. This suggests that the susceptibility has a genetic basis. The most likely mechanism is a somatic mutation. (35 refs)

78-3160 **Inhibition of Post-replication Repair by Isonicotinic Acid Hydrazide.** (Eng) Klamerth, O. L. (Institut fur Humangenetik, Universitat Heidelberg, D-69 Heidelberg, W. Germany). *Mutat Res* 50(2): 251-261; 1978.

The cell survival rate and mitotic index were determined in human peripheral lymphocytes exposed to various concentrations of isonicotinic acid hydrazide (INH) and N-methyl-N-nitrosourea (MNH), and the effect of INH on excision and postreplication repair in Chinese hamster ovary (CHO) cell cultures was determined. INH, in concentrations < 1 mM, was harmless to human cells. In the presence of MNU, however, even low concentrations of INH led to a pronounced reduction in cell survival and mitosis; this reduction was greater after 96 hr than after 72 hr. At > 5 mM, INH alone markedly depressed the cell number and the mitotic index. In CHO cells, incorporation of thymidine was almost totally inhibited by 10 mM INH, but up to 1 mM had no appreciable effect. No appreciable thymidine incorporation into the DNA of CHO cells, either with or without INH, was detected after treatment of these cells with NMU following blockage of normal semiconservative replication with hydroxyurea. A small increase over control value was consistently observed shortly after pulse labeling for MNU concentrations < 0.1 mM when semi-conservative replication was not inhibited. INH inhibited post-replication repair, possibly because it binds to exposed single-stranded segments of DNA in MNU-alkylated cells after strand breakage and/or excision of apurinic sites in parental DNA, followed by insertion into the daughter strand, but before sealing by ligase. INH had no effect on excision repair in these cells. (27 refs)

78-3161 **Dose Response to Intrarectal Administration of N-Methyl-N-nitrosourea and Histopathologic Evaluation of the Effect of Two Retinoids on Colon Lesions Induced in Rats.** (Eng) Ward, J. M. (Tumor Pathology Branch, Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NCI, NIH, PHS, U.S. Dept. Health, Education and Welfare, Bethesda, MD, 20014); Sporn, M. B.; Wenk, M. L.; Smith, J. M.; Feeser, D.; Dean, R. J. *J Natl Cancer Inst* 60(6): 1489-1493; 1978.

Male F344 rats (8 wk of age) were given twice weekly intrarectal administrations of 2, 1, or 0.5 mg N-methyl-N-nitrosourea (NMU) for 8 wk. Five days after the last NMU instillation, the rats were placed on one of three diets: chow with gelatin beadlets, chow with beadlets containing 0.024% 13-cis-retinoic acid, or chow and beadlets with 0.006% of the trimethylmethoxyphenyl analog of retinoic acid ethylamide. Groups of rats were killed 22-26 wk after the first carcinogen treatment. The number of rats with colon carcinoma and the number of tumors per rat were dose-related. Tumors were noted in 75% of the high-dosed, 45% of the middle-dosed, and 25% of the low-dosed rats. Furthermore, pathologic investigation of four predesignated colon locations indicated a dose-related incidence of microscopic preinvasive and invasive colon carcinomas. The feeding of the different diets did not significantly alter tumor incidence or the mean histopathologic score at the predesignated locations for preinvasive or invasive neoplastic lesions. Over 90% of the colon tumors induced were invasive tubulopapillary adenocarcinomas. The diameters of the tumors correlated significantly with the degree of invasion of the colon. Only one tumor (a signet ring carcinoma) metastasized to the peritoneal cavity, and only 2/300 NMU-treated rats had tumors at sites other than the colon. (12 refs)

78-3162 **Mutagenicity of BCNU and Related Chloroethylnitrosoureas in Drosophila.** (Eng) Kortselius, M. J. (Dept. Radiation Genetics and Chemical Mutagenesis, Sylvius Labs., State Univ. Leiden, Wassensarseweg 72, Leiden, Netherlands). *Mutat Res* 57(3): 297-305; 1978.

To determine whether or not the mutagenicity of the chloroethylnitrosourea (CNU) group is influenced by N-1 substitutions, 1,3-bis(2-chloroethyl)-1-nitrosourea (BCNU) and 10 related CNU's were tested for their ability to induce sex-linked recessive lethals in mature Drosophila spermatozoa. All CNU's tested were found to be potent mutagens. Among the compounds with one CNU group, chlorozotocin, BCNU, and 1-(2-methanesulfonyloxyethyl)-3-(2-chloroethyl)-3-nitrosourea had the strongest mutagenic effects. Two hydroxyalkyl CNU's, CNU-ethanol [1-(ω-hydroxyethyl) -3- (2-chloroethyl)-3-nitrosourea] and CNU-propanol, also behaved as potent mutagens, although their mutation frequencies were one order of magnitude lower. Among the substances with two CNU groups, bisCNU-diphenylmethane [1,1-diphenylmethylenebis-3-(2-chloroethyl)-3-nitrosourea] and bisCNU-ethane were most active. When the interconnecting polymethylene chain was elongated from two (bisCNU-ethane) to six methylene groups (bisCNU-hexane), mutagenicity decreased by a factor of 2. The mutagenic activity of polymethylene bisCNU's with connecting chains of intermediate length was not different from that of bisCNU-hexane. The different mutagenic responses probably do not result from different reaction mechanisms for these compounds, but rather from differences in dose at the genetic target. Small structural changes in the nonmutagenic part of the molecules can result in differences in transportability, differences in actual doses at the target cells, and, thus, differences in mutagenicity. (25 refs)

78-3163 **Immunological Detection of O^6-Methylguanine in Alkylated DNA.** (Eng) Briscoe, W. T. (Dept. Biochemistry, School Medicine, Oral Roberts Univ., Tulsa, OK); Spizizen, J.; Tan, E. M. *Biochemistry* 17(10): 1896-1901; 1978.

Antibodies to O^6-methyldeoxyguanosine were produced in rabbits and used in a radioimmunoassay to detect this nucleoside at picomole levels. The specificity of the antibodies was demonstrated by the use of nucleoside analogs as inhibitors in the radioimmunoassay. The antibodies cross-reacted with O^6-methylguanosine, O^6-methylguanine, and O^6-ethylguanosine. There was 10^4-10^6 times less sensitivity to inhibition by deoxyadenosine, deoxyguanosine, and guanosine than by O^6-methyldeoxyguanosine. The radioimmunoassay also detected O^6-methylguanine in DNA alkylated by agents known to produce O^6-methylguanine, such as N'-methyl-N-nitrosourea. DNA alkylated with dimethyl sulfate, which does not produce O^6-methylguanine in DNA, cross-reacted with the antibodies to a very limited extent. Subsequent purification of the antibodies and further refinement of the assay system will enhance its sensitivity to a more practical level r use with biological systems. The system would be a useful ·obe for detecting O^6-methylguanine in DNA treated with :rtain alkylating agents, including carcinogens, and would so be useful in following the repair of such lesions. (26 refs)

3-3164 **Electron Spin Resonance Study of the Role of Nitrosyl-Heme in the Activation of Guanylate yclase by Nitrosoguanidine and Related Agonists.** (Eng) eRubertis, F. R. (Veterans Admin. Hosp., University Drive , Pittsburgh, PA); Craven, P. A.; Pratt, D. W. *Biochem iophys Res Commun* 83(1): 158-167; 1978.

artial purification of soluble guanylate cyclase (GC) from at liver results in loss of responsiveness of the enzyme to everal N-nitroso carcinogens, nitric oxide, nitrite, and nitrorusside (NP). Responsiveness to these agonists is restored y the addition of 0.25-50 μM of free or protein-bound heme lus a reducing agent, but not by other free or chelated forms f iron. The formation of nitrosyl-heme from the GC agonists I-methyl-N′-nitro-N-nitrosoguanidine (MNNG), NO, nirite, and NP was examined by electron spin resonance (ESR) pectroscopy under different incubation conditions and corelated with enzyme activation. The capacity of preformed itrosyl-Hb to stimulate partially purified enzyme activity 'as also assessed. In the presence of heme plus a reducing gent (1 mM dithiothreitol, cysteine, ascorbate, or glutathone), each enzyme agonist formed paramagnetic nitroyl-heme complexes, as shown by ESR spectroscopy. The apacity of MNNG, NO, nitrite, and NP to activate purified nzyme activity correlated with their capacity to generate itroso-heme under different incubation conditions. Preormed nitrosyl-Hb activated purified GC in the absence of oth heme and reducing agents, and max effects were noted t a concentration of 10 μM. It is concluded that nitroyl-heme is an active intermediate in the expression of the ctions of N-nitroso compounds, NO, nitrite, and NP on GC. 31 refs)

'8-3165 **Carcinogen-induced Damage to DNA.** (Eng) Strauss, B. S. (Dept. Microbiology, Univ. Chicao, Chicago, IL). *In: Carcinogens: Identification and Mechnisms of Action. Proceedings of the Thirty-First Annual Symosium on Fundamental Cancer Research Held at Houston, 'exas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 14-15 pp.; 1978. no refs)

'8-3166 **Mechanisms of Inhibition by Ascorbate of Microbial Mutagenesis Induced by N-Nitroso Compounds.** (Eng) Guttenplan, J. B. (Dept. Biochemistry, New York Univ. Dental Center, New York, NY, 10010). *Cancer Res* 38(7): 2018-2022; 1978.

The effects of ascorbate on the mutagenesis induced in *Salmonella typhimurium* TA1530 by N-methyl-N′-nitro-N-nitrosoguanidine (MNNG), dimethylnitrosamine (DMN), and N-methyl-N-nitrosourea (MNU) were investigated. Ascorbate inhibited the mutagenesis induced by MNNG and DMN, but it had no effect on that induced by MNU. Inhibition of MNNG-induced mutagenesis resulted from a reaction between ascorbate and MNNG that led to consumption of MNNG. The rate of this reaction was considerably enhanced by catalytic amounts of Cu(II) and Fe(III). No direct reaction between DMN and ascorbate was detectable, but relatively high concentrations of Cu(II) enhanced the inhibition of DMN-induced mutagenesis by ascorbate. However, since DMN does not appear to be directly deactivated by ascorbate, a metabolite of DMN or even an activated oxygen species generated by the mixed-function oxidase system may be reduced by ascorbate. Added protein reduced the effectiveness of Cu(II) as a catalyst of the reaction between ascorbate and MNNG, suggesting that the microsomal protein necessary to activate DMN may reduce the concentration of free Cu(II) and thereby lower its catalytic efficiency. (18 refs)

78-3167 **In Vitro Transformation of Epidermal Cells from Newborn Mice.** (Eng) Slaga, T. J. (Biology Div., Oak Ridge Natl. Lab., P.O. Box Y, Oak Ridge, TN, 37830); Viaje, A.; Bracken, W. M.; Buty, S. G.; Miller, D. R.; Fischer, S. M.; Richter, C. K.; Dumont, J. N. *Cancer Res* 38(8): 2246-2252; 1978.

One-day-old primary cultures of epidermal cells from newborn BALB/c mice, plated at 10^6 cells/ml were treated for 2 days with various concentrations of N-methyl-N′-nitro-N-nitrosoguanidine (MNNG), 3-methylcholanthrene (3-MC), and 3-methylcholanthrene 11,12-epoxide (3-MC 11, 12-epoxide). Untreated newborn epidermal cells both divided and keratinized in vitro in Medium 199 supplemented with 10% fetal calf serum. After a few weeks the cells enlarged, showed signs of senescence, and then died after about 3 mo in vitro. The epidermal cells treated with MNNG, 3-MC, or 3-MC 11,12-epoxide went through a similar crisis, appearing as though they too were going to die at 3 mo. However, small populations of the remaining cells began to proliferate into colonies that soon grew to confluence. These epithelial cells were characterized by rapid growth, loss of visible keratinization, and subculturability, having been passaged 12 times, in contrast to untreated cells, which were not subculturable. Electron microscope studies did not reveal any true desmosomes, but junctional complexes were present in all cell strains examined. The injection of 10^6 cells from the epidermal cell strains into athymic 'nude' or syngeneic mice resulted in rapidly growing solid tumors that were characterized as highly anaplastic 'undifferentiated' tumors. (12 refs)

78-3168 **Induction and Isolation of Frameshift Mutants in Cultured Chinese Hamster Don Cells.** (Eng) Hamada, K. (Public Health Inst. Hyogo Prefecture, 2-1 Arata-cho, Hyogo-ku, Kobe 652, Japan); Isomura, K.; Teranishi, K.; Watanabe, H. *Mutat Res* 54(1): 61-72; 1978.

The induction, isolation, and characterization of frameshift [8-azaguanine-resistant (8-AG-r)] mutants induced in Chinese hamster Don (CHD) cells are reported. 2-Methoxy-6-chloro -9- [3-(2-chloroethyl)aminopropylamino] acridine dihydrochloride (ICR-191: 10^{-5} M) a potent frameshift mutagen, N-methyl-N′-nitrosoguanidine (MNNG: 5 x 10^{-6} and 10^{-6} M), and ethyl methanesulfonate (EMS: 2 and 3 x 10^{-2} M) increased the frequency of forward mutations from 8-AG sensitivity (8-AG-s) to 8-AG-r over controls significantly (approx 100-fold). Twenty-one 8-AG-r mutants were isolated from a CHD subline (AP-01 cells) after treatment with 10^{-5} and 1.36 x 10^{-5} M ICR-191, and several clonal mutants were tested for their ability to revert to 8-AG-s after treatment with the three mutagens and a carcinogen 2-nitrofluorene (2-NF), also a known frameshift mutagen. Clones ICR-014 and ICR-172 showed a significant increase in reversion frequency over control levels after treatment with ICR-191 and 2-NF, respectively, but not with the other two mutagens. These results suggest that these two mutants have a different frameshift mutation in one of the loci affecting 8-AG-r. Hypoxanthine-guanine phosphoribosyltransferase activities in clones ICR-014 and ICR-172 were 1.9% and 34% of that of the original AP-01 cells. (38 refs)

78-3169 Mutagenesis of Nitrosopiperidines in *Drosophila melanogaster* (Meeting Abstract). (Eng) Nix, C. E. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Brewen, B.; Wilkerson, R.; Epler, J. L. *Mutat Res* 53(1): 66-67; 1978. (1 ref)

78-3170 **Production of Pancreatic Acinar Cell Carcinoma by Combined Administration of 4-Hydroxyaminoquinoline 1-Oxide and Azaserine in Partial Pancreatectomized Rats.** (Eng) Konish, Y. (Dept. Oncological Pathology, Cancer Center, Nara Medical Univ., 840 Shijo-cho, Kashihara, Nara 634, Japan); Denda, A.; Inui, S.; Takahashi, S.; Ueda, N.; Namiki, M. *Cancer Lett* 4(4): 229-234; 1978.

The syncarcinogenic effect of azaserine (AS) and 4-hydroxyaminoquinoline 1-oxide (4-HAQO), as well as the influence of partial pancreatectomy, (PP) on the development of pancreatic carcinomas was studied in five groups of male Wistar rats. Group 1 had sham operation only; Group 2 had PP; Group 3 had 24 weekly injections of 5 mg/kg AS; Group 4 had PP and treatment as in Group 3 starting 3 days after the operation; Group 5 had PP, injection of 7 mg/kg
4-HAQO 3 days later, and then 10 weekly injectic
of 30 mg/kg AS. Groups 1, 2, and 5 were sacrific
at 52 wk. Groups 3 and 4 were sacrificed at 62 wk af
the initial carcinogen administration. No hyperplas
nodules, adenomas, or carcinomas were noted in
Group 1 or 10 Group 2 animals. Of 11 Group 3 anim
10 had hyperplastic nodules, 11 had adenomas, and 1 ha
a carcinoma. Of 14 Group 4 animals, 14 had hyperplast
nodules and adenomas, and 1 had a carcinoma. Of 10 Grou
5 animals, 5 had hyperplastic nodules, 10 had adenomas, an
7 had carcinomas. Liver metastases were noted in two Grou
5 animals. All carcinomas were acinar cell carcinomas th
invaded the surrounding tissue and vascular system. It is co
cluded that PP enhanced 4-HAQO carcinogenesis, which w
further promoted by AS. (9 refs)

78-3171 **The In Vitro Interaction of the Carcinogen
4-Nitroquinoline-N-oxide and Its Metaboli
4-Hydroxylaminoquinoline-N-oxide, with Intact Rat Liv
Mitochondria.** (Eng) Hadler, H. I. (Dept. Chemistry and Bi
chemistry, Southern Illinois Univ., Carbondale, IL, 62901
Cao, T. M. *Chem Biol Interact* 20(2): 219-226; 1978.

The interaction of 4-nitroquinoline N-oxide (NQO) and i
metabolite 4-hydroxylaminoquinoline-N-oxide (HAQ
with the mechanism of oxidative phosphorylation in mit
chondria derived from rat liver was investigated. When r
ducing equivalents were provided by the substrate β-hydro
ybutyrate and they flowed through the three coupling sit
of the respiratory chain, both NQO and HAQO (200 μM
acted as uncoupling agents. When β-hydroxybutyrate w
the substrate, NQO and HAQO caused reducing equivalen
to circumvent the first coupling sequence of the respirato
chain. With succinate as the substrate, the reducing equiv
lents flowed through coupling sites 2 and 3 of the respirato
chain. At 200 μM, NQO caused moderate uncoupling of t
second and third coupling sites of the respiratory chain.
contrast, 200 μM HAQO did not cause any uncoupling
sites 2 and 3 of the respiratory chain. Thus, NQO and HAQ
caused reducing equivalents to circumvent the first couplin
site of the respiratory chain. These results add to the expe
mental similarity between oxidative phosphorylation a
chemical carcinogenesis. (27 refs)

78-3172 **Distribution of 4-Nitroquinoline 1-Oxide R
ductase in the Mucosa of Canine Digesti
Tract.** (Eng) Suzuki, K. (Dept. Biochemistry, Nihon Uni
Sch. Medicine, 30-1, Ohyaguchi Kamimachi, Itabashi-k
Tokyo 173, Japan). *Gann* 69(2): 229-235; 1978.

The distribution of 4-nitroquinoline 1-oxide (4-NQO) redu
tase in the digestive tracts of mongrel dogs was studied. T
activity of the enzyme was highest in the esophagus (14
units) and next highest in the stomach (4.1 units). It w
much lower in the small and large intestines (< 1 unit) a

owest in the rectum (0.2 unit). The enzyme activity in the ipper portion of the esophagus was slightly higher than that n the middle and lower portions, it was significantly higher n the upper and middle portions of the female esophagus han in the same portions of the male esophagus. Only the nucous membrane of the esophagus showed a remarkably iigh enzyme activity, the activities in the tunica propria plus ubmucosa and tunica muscularis being low. 4-NQO reducase was detected in large amounts in the cytosol fraction of he esophageal mucous membrane and in small amounts in he microsome fraction. Uniform enzyme activity ranging rom 3.9 to 4.8 units was detected throughout the stomach: he greater and lesser curvatures, anterior and posterior parietes, and fundus; however, the activity was reduced by hree-fourths in the pyloric antrum. The enzyme activity was slightly higher in females than in males. (32 refs)

78-3173 **Metabolism of N-Nitrosomorpholine by the Rat In Vivo and by Rat Liver Microsomes and Its Oxidation by the Fenton System.** (Eng) Manson, D. (Chester Beatty Res. Inst., Inst. Cancer Res., Royal Cancer Hosp., Fulham Road, London SW3 6JB, England); Cox, P. J.; Jarman, M. *Chem Biol Interact* 20(3): 341-354; 1978.

N-Nitrosomorpholine was converted to N-nitroso-2-hydroxymorpholine by rat liver microsomes and by chemical oxidation in a modified Fenton system. The hydroxy derivative was also synthesized by the oxidation of N-nitrosodiethanolamine with permanganate and characterized as the methoxime and the 2,4-dinitrophenylhydrazone. The Fenton system also yielded products believed to be N-nitroso-2-morpholone and the 2-hydroperoxy- and 2-peroxyderivatives of N-nitrosomorpholine. The only urinary metabolite definitely identified was N-nitrosodiethanolamine. The significance of metabolic 2-hydroxylation in relation to the carcinogenic action of N-nitrosomorpholine is discussed. (30 refs)

78-3174 **Liver and Lung Tumors in Dogs from 4,4′-Methylene-bis(2-methylaniline).** (Eng) Stula, E. F. (Haskell Lab., Elkton Road, Newark, DE, 19711); Barnes, J. R.; Sherman, H.; Reinhardt, C. F.; Zapp, J. A. *J Environ Pathol Toxicol* 1(3): 339-356; 1978.

The occurrence of liver and lung tumors in dogs given 4,4′-methylene-bis(2-methylaniline) (MeMDA) is reported. Six female beagle dogs were given a daily po dose of 100 mg of MeMDA 3x/wk for 6 wk, then 5x/wk for 5 wk, at which time the dose was reduced to 50 mg 5x/wk and given continuously for periods ranging from 2.7 to 7.0 yr. Six female beagle dogs were kept as untreated controls and were sacrificed after 8.3-9 yr. The MeMDA-treated dogs developed renal atrophy with an elevated blood urea nitrogen during an approx 6-mo period prior to death or being sacrificed in extremis. Three of three MeMDA-treated dogs that survived 5.2-7.0 yr developed hepatocellular carcinomas, and two of these three also developed primary lung tumors. No liver or lung tumors were observed in the six control dogs. Four of the six control dogs had mammary tumors, but no such tumors were observed in the six MeMDA-treated dogs. The finding of mammary tumors in control dogs but not in MeMDA-treated dogs is most likely due to the difference in their ages rather than to an effect of MeMDA. It is concluded that MeMDA is carcinogenic for the dog at a probability level of $p < 0.05$ for liver tumors and $p < 0.10$ for lung tumors. (17 refs)

78-3175 **Mutagenicity of Styrene and Styrene Oxide.** (Eng) de Meester, C. (Lab. Analytical Chemistry, Univ. Louvain, Pharmacy Sch., UCL 7369 Brussels B-1200, Belgium); Poncelet, F.; Roberfroid, M.; Rondelet, J.; Mercier, M. *Mutat Res* 56(2): 147-152; 1977.

The mutagenicity of styrene and styrene oxide was examined in various *Salmonella typhimurium* strains with and without the S-9 fraction from Arochlor-induced male Wistar rats. In the absence of S-9, no direct mutagenic effect was observed for styrene at doses of 1 nanomole (nmol) to 100 micromoles (μmol) per plate. In the presence of the fraction, only strain TA1535 demonstrated his+ revertants; the number of revertants increased with doses of 1-11 μmol/plate. However, the higher dose was extremely toxic, with only 5% bacterial survival. At doses of 1 nmol to 100 μmol, styrene oxide was only mutagenic in TA1535 and TA100, both in the presence and absence of the S-9 mix. Toxicity was severe at doses $>$ 60 μmol/plate in the absence of the mix and at doses near 100 μmol/plate in the presence of the mix. These findings suggest that styrene is probably mutagenic, because it is metabolized to styrene oxide. (13 refs)

78-3176 **Deamination of 1-Methylcytosine by the Carcinogen N-Acetoxy-4-acetamidostilbene: Implications for Hydrocarbon Carcinogenesis (Letter to Editor).** (Eng) Scribner, J. D. (Pacific Northwest Res. Foundation, Seattle, WA, 98104); Smith, D. L.; McCloskey, J. A. *J Org Chem* 43(10): 2085-2087; 1978.

Reaction of the carcinogen N-acetoxy-4-acetamidostilbene with 1-methylcytosine in water and acetone resulted in a uracil derivative, apparently 1-(4-acetamidophenyl)-1-[3-(1-methyluracilyl)]-2-hydroxy-2-phenylethane. It is proposed that a similar reaction could take place between cytidine or a nucleic acid and a hydrocarbon epoxide. (11 refs)

78-3177 **Differential Mutagenicity of Reaction Products of Various Pyrazolones with Nitrite.** (Eng) Arisawa, M. (Dept. Microbiology and Chemotherapy, Nippon Roche Res. Center, 200 Kajiwara, Kamakura-247, Japan); Fujiu, M.; Suhara, Y.; Maruyama, H. B. *Mutat Res* 57(3): 287-296; 1978.

Four pyrazolones, antipyrine (AP), aminopyrine (AMP), sulpyrine (SP), and isopropylantipyrine (IPA), were compared with respect to their reactivity with sodium nitrite and the mutagenicity of their reaction products, as determined by the *Salmonella typhimurium* assay. Each pyrazolone (20 mg) was reacted with nitrite (40 mg) in 2 ml of water at pH 1.5-4.5 and incubated at 37 C. In the absence of nitrite, SP was unstable at these pH's and it degraded 50%-65%, but the other compounds were stable. The presence of nitrite, however, led to the complete degradation of AP, AMP, and SP within 1 hr. In contrast, IPA remained essentially intact. Dimethylnitrosamine was produced from AMP in 17%-79% yield, and 4-nitrosoantipyrine was produced from AP in 3%-22% yield. SP yielded at least five unidentified degradation products detectable by high-pressure liquid chromatography. Similar results were obtained at 0.1 mg/ml of the pyrazolones and equal or twofold amounts of nitrite. After lyophilization, the reaction products of AP were found to be mutagenic in *S. typhimurium* strains TA98 and TA100, but the products of AMP and SP were mutagenic only in TA100. In addition to SP, the products of AP and AMP contained several unidentified mutagens. Incubation with an S-9 mix prepared from activated rat liver microsomes did not affect the mutagenicity of the SP reaction products, but it decreased that of the products of AP and AMP. The IPA-nitrite reaction mixture was not mutagenic with either tester strain. Of the four compounds, only IPA appears safe with respect to possible mutagen formation by nitrite under physiological conditions. (6 refs)

78-3178 **Dehydroretronecine-induced Skin Tumors in Mice.** (Eng) Johnson, W. D. (Dept. Pathology, Univ. Wisconsin Medical Sch., Madison, WI, 53706); Robertson, K. A.; Pounds, J. G.; Allen, J. R. *J Natl Cancer Inst* 61(1): 85-89; 1978.

Female Swiss mice that are susceptible to skin tumors were given six sc injections and/or topical applications of dehydroretronecine (DR: 20 mg/kg), a metabolite of the pyrrolizidine alkaloid monocrotaline, and they were observed for tumor development over a 15-mo period. One or more tumors were observed in 63/92 treated mice, and 47/63 had skin tumors. Of the mice with skin tumors, 39/47 had basal cell carcinomas, 5/47 had squamous cell carcinomas, and one mouse each developed a single papilloma, mixed basosquamous carcinoma, and sebaceous adenoma. Three pulmonary adenocarcinomas also occurred. All of the non-skin tumors (except a rhabdomyosarcoma) were in areas distant from the site of DR application or injection. In contrast, most of the basal cell and squamous cell carcinomas developed at the site of injection or application and appeared as well-circumscribed, nonulcerating, sc nodules of varying sizes attached to the overlying epidermis. The results indicate that DR is a proximate carcinogen capable of causing a high inciden of skin tumors in mice at the site of sc injection or topic application. (25 refs)

78-3179 **Synthesis of the Urinary Glucuronic Acid Co jugate of N-Hydroxy-4-aminobiphenyl.** (En Moreno, H. R. (Dept. Pharmacology, Univ. Miami, Sc Medicine, Miami, FL, 33152); Radomski, J. L. *Cancer Le* 4(2): 85-88; 1978.

The glucuronic acid conjugate of N-hydroxy-4-aminobiph nyl was synthesized. The synthetic conjugate was identic by infrared and chromatographic analyses with the conjuga isolated from urine, thus establishing its structure as an N- conjugate, sodium (N,4- biphenyl -N- hydroxy-D-gl curonosylamine). (6 refs)

78-3180 **Activation of [^{14}C]Chlorobiphenyls to Pr tein-binding Metabolites by Rat Liver Mi crosomes.** (Eng) Hesse, S. (Gesellschaft f. Strahlen- und Um weltforschung, Abteilung fur Toxikologie, D-804 Neuherberg/Munich, W. Germany); Mezger, M.; Wolff, T *Chem Biol Interact* 20(3): 355-365; 1978.

The irreversible binding of ^{14}C-2,2′-di- and ^{14}C-2,4, 2′,4′,5′-hexachlorobiphenyl (^{14}C-DCB and ^{14}C-HC to protein was studied in the presence of Wistar r liver microsomes and an NADPH-generating syste Protein-bound radioactivity was found with ^{14}C-DC but not with ^{14}C-HCB. The binding of ^{14}C-metabolit was increased by pretreatment of the rats with phen barbital or polychlorinated biphenyls. Protein bindi was linear for 80 min. In contrast, monohydrox DCB metabolites were formed and degraded within 4 min. Inhibition of secondary oxidation of DCB by scavengin superoxide anions or by glucuronidation of the monopheno markedly decreased the protein binding. Addition of tr chloropropene oxide or styrene oxide, both inhibitors epoxide hydrase, did not significantly stimulate the bindin The results suggest that the majority of reactive DC metabolites arise from secondary metabolism, ie, the subs quent oxidation of the phenolic metabolites. Arene oxide the primary products, appear to play a minor role in th protein binding of DCB. (27 refs)

78-3181 **Epoxide Hydrase: Structure-Activity Relatio ships of In Vitro Stimulators of the Microsom Activity.** (Eng) Ganu, V. S. (Biology Div., Brookhaven Nat Lab., Upton, NY, 11973); Alworth, W. L. *Biochemistr* 17(14): 2876-2881; 1978.

The structure-activity relationships of several in vitr imulators of microsomal epoxide hydrase (EH) were studd using liver microsome preparations from male Sprae-Dawley rats. Benzophenone, 9-fluorenone, diphenylcyopropenone, benzyl phenyl ketone, propyl phenyl ketone, ialcone, and chalcone oxide (ChO) stimulated microsomal H in vitro, as measured by a standard radiometric assay ing styrene oxide as substrate. The most effective of these mpounds, ChO and 9-fluorenone, were more effective than etyrapone, a previously reported stimulator of this enzyme. ther aryl ketones, such as acetophenone, benzyl ethyl kene, dibenzyl ketone, and phenylmethylcyclopropenone, did ot stimulate EH activity. Kinetic analyses indicated that, though chalcone, 9-fluorenone, diphenylcyclopropenone, id ChO increased the Vmax of the EH reaction, diphenylcyopropenone and ChO increased the Vmax and Km by comrable factors. At higher concentrations of styrene oxide, ibstrate inhibition was observed. The kinetics of the mirosomal EH reaction with styrene oxide were consistent ith the formation of an inactive ES_2 (enzyme, $substrate_2$) omplex at high substrate concentrations. One in vitro effect ChO was to largely eliminate the inhibition caused by high yrene oxide concentrations. It is concluded that compounds ntaining an aryl carbonyl substituted with an additional ydrophobic group are effective in vitro stimulators of EH. he data suggest that these compounds bind at a site that is stinct from the catalytic site where styrene oxide binds. (45 fs)

8-3182 **Protein Composition of Nuclear Ribonucleoprotein Particles Isolated from Liver of Rats in ie Early Stages of Feeding of 3′-Methyl-4-dimethylninoazobenzene and from Hepatoma Induced by the ame Carcinogen.** (Eng) Patel, N. T. (Div. Biochemistry, ept. Human Biological Chemistry and Genetics, Univ. Texs Medical Branch, Galveston, TX, 77550); Holoubek, V. *hem Biol Interact* 19(3): 303-316; 1978.

he protein composition of liver nuclear 30S ribonucleorotein particles at three intervals after the start of feeding ′-methyl-4-dimethylaminoazobenzene (3′-MeDAB) or -aminoazobenzene (AB) to male albino Holtzmann rats was ompared with that of the same particles isolated from norial liver and from 3′-MeDAB-induced hepatoma. There ere no qualitative or quantitative differences in the polypepde composition of the core protein moiety of ribonucleorotein particles isolated from the liver of control rats, of nimals fed AB for 3, 6, or 10 wk, or of animals fed ′-MeDAB for 3 wk. The amount of the 42,000-dalton ore polypeptide was decreased after 6 wk of feeding ′-MeDAB and was completely missing after 10 wk. he loss of this polypeptide was not, however, characeristic of malignant transformation, since it was preent in an even higher proportion in nuclear ribonucleorotein particles isolated from 3′-MeDAB-induced epatomas than in particles isolated from control ver cells. 3′-MeDAB bound to the proteins of the liver nuclear ribonucleoprotein particles and interfered with RNA processing. Changes in the composition of the protein moiety of the particles could reflect changes in the liver cell population leading to the final selection of hepatoma cells that are resistant to the toxic effect of 3′-MeDAB on RNA processing. (32 refs)

78-3183 **Comparison of the Carcinogenicity and Immunologic Specificity of Aminoazo Compounds.** (Rus) Koresteleva, T. A. (Inst. Oncology, Leningrad, USSR); Potapenkova, L. S. *Gig Tr Prof Zabol* (5): 24-27; 1978.

The relationship between the carcinogenicity of aniline dyes and scission of their radical groups, known as haptens, was investigated. C3HA mice were fed the carcinogenic dye o-aminoazotoluene (AAT) or the structurally similar noncarcinogenic dye Scarlet R, 2 mg, for 60 to 100 days. Sera from these mice were assayed by Ouchterlony reaction using rabbit antisera containing antibodies to the AAT hapten. The sera of mice treated with AAT, but not those treated with Scarlet R, contained a carcinogenic metabolite that included a structural group of the carcinogen acting the part of the hapten. The data showed that antibodies against the AAT hapten had a high degree of specificity. Although Scarlet R contains the same radical group as AAT, this group was not separated from Scarlet R during the course of its metabolism in the mice. This indicates that aniline dyes that contain carcinogenic groups in their molecules may not be carcinogenic. (8 refs)

78-3184 **Tumor Induction by 7H-Dibenzo[c,g]carbazole in the Respiratory Tract of Syrian Hamsters.** (Eng) Sellakumar, A. (New York Univ. Medical Center, Inst. Environmental Medicine, Tuxedo, NY); Stenback, F.; Rowland, J.; Shubik, P. *J Toxicol Environ Health* 3(5-6): 935-939; 1977.

The carcinogenicity of the tobacco smoke component 7H-dibenzo[c,g]carbazole (DBC: particle size > 30 m) was investigated in Syrian golden hamsters. Group 1 received an intratracheal instillation of 3.0 mg DBC for 10 wk; Group 2 received 0.5 mg for 18 wk. Fifteen of 45 Group 1 animals developed a total of 21 respiratory tract tumors with a mean latency of 41 wk. These tumors were distributed among the larynx (1), trachea (11), bronchi (3), and lungs (5). Squamous cell carcinoma was the predominant tumor type found in the trachea, bronchi, and lungs. Thirty-three of 46 Group 2 animals developed respiratory tract tumors with a mean latency of 22 wk. There were 4 laryngeal, 22 tracheal, 15 bronchial, and 6 pulmonary tumors. Thirteen squamous cell carcinomas were found in the bronchi, 4 in the lungs; 21 tracheal papillomas were also identified. (11 refs)

78-3185 **In Vitro Chemical Carcinogenesis.** (Eng) Heidelberger, C. (Comprehensive Cancer Center, Los Angeles County-Univ. Southern California, Los Angeles, CA); Mondal, S. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 5 pp.; 1978. (no refs)

78-3186 **Analytical and Biological Analyses of Test Materials from the Synthetic Fuel Technologies. I. Mutagenicity of Crude Oils Determined by the *Salmonella typhimurium*/Microsomal Activation System.** (Eng) Epler, J. L. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Young, J. A.; Hardigree, A. A.; Rao, T. K.; Guerin, M. R.; Rubin, I. B.; Ho, C. H.; Clark, B. R. *Mutat Res* 57(3): 265-276; 1978.

The mutagenicity of crude industrial products and effluents was assayed with the Salmonella/microsomal activation system. The test materials (2 crude coal-liquefaction products and 2 natural crude oil samples) were fractionated into primary classes by liquid-liquid extraction and then further fractionated by column chromatography. Prescreening was accomplished over a wide concentration range with the tester strains TA1535, TA100, TA1537, TA1538, and TA98. All testing was carried out in the presence of rat liver microsome preparations from rats induced with either Arochlor 1259 or phenobarbital. Active fractions (mainly the neutral fractions containing polycyclic aromatic hydrocarbons and certain basic fractions) were identified, and dose-response relationships were established. Standard values (revertants/mg test material) were obtained with strain TA98. The total mutagenic activity of the two synthetic fuel samples was consistently higher than that of the natural crude oils. The activities of the subfractions were roughly additive, and they presumably reflected the mutagenic potential of the entire test material. There were several consistencies: (1) all crude and synthetic fuels showed some mutagenic potential, (2) the neutral and basic fractions showed activities regardless of source, and (3) the relative total mutagenic potentials varied over two orders of magnitude. The use of short-term biological test systems coupled with analytical chemical evaluation of complex materials encountered in modern technology may provide basic information for predicting and identifying biohazards for human populations. (18 refs)

78-3187 **Anthracene-induced Melanotic Tumors in *Drosophila*.** (Eng) Corwin, H. O. (Dept. Life Sciences, Univ. Pittsburgh, Pittsburgh, PA, 15260); Gottlieb, F. J. *Environ Res* 15(3): 327-331; 1978.

The possible genetic and teratogenic effects of low level chronic exposure to anthracene, dibenz(a,h)anthracene, benzo(a)pyrene, and pyridine were assayed in *Drosophil melanogaster*. Chronic exposure was accomplished by feedin the Drosophila three different low concentratio (10^{-3}, 10^{-4}, or 10^{-5} M) of individual agents throughout their larval development. Differences in surviv to the adult stage between control and treated fli were taken as signs of immediate toxic effects. Th surviving adults were examined for tumors an developmental abnormalities and mated according to several designs. None of the four agents induced a significan frequency of X-linked mutations detectable by multigeneration tests. The F_1 progeny of anthracene-treated males an females had a significant frequency of melanotic tumor production. No such significant tumor-forming activity was obvious in the F_1 and F_2 progenies from parents treated wit the remaining three agents. Although all three concentration of anthracene induced tumor production, there was n clear-cut indication of a dose response. It is concluded tha the increase in tumor production, that results from anthracene treatment is probably due to the induction of transmissible, dominant mutations having low penetrance. (4 refs)

78-3188 **Determination of Arene Oxides by a Ga Chromatography-Mass Spectrometry System Thermal Reactions of 9,10-Epoxy-9,10-dihydrophenanthrene.** (Eng) Patel, J. R. (Center Bio-Organic Studies, Univ. New Orleans, Lakefront, New Orleans, LA, 70122); Griffin, G. W.; Laseter, J. L. *Anal Lett* B11(3): 239-247; 1978.

An analytical technique in which a gas chromatograph is coupled to a mass spectrometer-computer system was developed to detect arene oxides using 9,10-epoxy-9,10-dihydrophenanthrene as a model substrate. With this technique, both deoxygenation and rearrangement of the model substrate were observed. Reactants and products were identified by comparing the retention times and mass spectral fragmentation patterns of authentic samples. (13 refs)

78-3189 **Inducible DNA Polymerase in Cultured Rat Fibroblasts Treated with Skin Carcinogen Mitomycin C.** (Eng) Bozhkov, V. M. (Inst. Cytology, Acad. Sciences USSR, Leningrad 190121, USSR); Barskaya, T. V.; Fridlyanskyaya, I. I.; Tomilin, N. V. *FEBS Lett* 86(2): 205-208; 1978.

The origin and reaction properties of the DNA polymerase in rat fibroblasts treated with mitomycin C were investigated. MMC treatment resulted in a two-fold increase of DNA polymerase with DNAa or DNAd as template. However, with the template initiator pdA·odT, activity decreased significantly after MMC treatment. Addition of the protein synthesis inhibitor puromycin (PM) completely blocked the rise in DNA-dependent DNA polymerase activity produced by

IMC. Cycloheximide produced similar results, suggesting iat the increase in DNA-dependent activity after MMC eatment is due to the translation of newly formed or preexting mRNAs'. No differences in the sensitivity of constituve and MMC-induced activities to variations in ion concenations (K^+, Na^+, Mg^{2+}, Mn^{2+}) were observed in andard assays with a DNAa template. Spermidine did not ffect MMC-induced DNAa-dependent activity, but it did ihibit the constitutive activity by 30%-35%. This finding as reversed in the assay with DNAd. There was also a diference between the inducible and constitutive DNA polymeases in the rate of incorporation of incorrect precursor leoxycytidine triphosphate) into pdA·odT modified by -bromomethylbenzanthracene. It is concluded that treatent of rat fibroblasts with MMC results in the suppression f constitutive DNAd-polymerase(s) and the synthesis of nother DNAa polymerase with a higher affinity for primer NA and a lower fidelity. (23 refs)

8-3190 **Carcinogenicity and Mutagenicity of Benz(a)anthracene Diols and Diol-Epoxides.** Eng) Slaga, T. J. (Biology Div., Oak Ridge Natl. Lab., Oak idge, TN, 37830); Huberman, E.; Selkirk, J. K.; Harvey, .; Bracken, W. M. *Cancer Res* 38(6): 1699-1704; 1978.

he carcinogenicity of benz(a)anthracene (BA), its five possile trans-dihydrodiols, and five of its diol-epoxides was invesgated following topical application to female CD-1 mice. he mutagenicity of BA and the trans-dihydrodiols was evaated in Chinese hamster V79 cells cocultivated with iradiated golden hamster embryo cells to metabolize the comounds. In the mutagenicity studies, (±)-trans-3,4 ihydroxy -3,4- dihydrobenz(a)anthracene (BA, ,4-dihydrodiol) was approx 10 times more mutagenic than A and about 20 times more mutagenic than the other dihyrodiols. However, it was much less (> 100 times) mutagenic ian benzo(a)pyrene, which was tested for comparison purposes. In the skin tumor-initiating experiments, all BA comounds were applied in a single dose of 2 micromoles, and ach treatment was followed 1 wk later by twice weekly aplications of 10 μg 12-O-tetradecanoylphorbol-13-acetate for 6 wk. BA 3,4-dihydrodiol was approx five times more active ian BA as a tumor initiator; the other BA hydrodiols were ll less active. (±)-trans- 3α,4β-Dihydroxy- 1-γ,2γ-epoxy-,2,3,4-tetrahydro-benzo(a)anthracene was approx 20% iore active as a tumor initiator than BA 3,4-dihydrodiol; the ther diol-epoxides were less active than BA. These findings iggest that the bay-region diol-epoxide of BA may be the ltimate carcinogenic and mutagenic form of BA. (41 refs)

3-3191 **Effect of Fetal Tissue on Experimental (7,12-Dimethylbenz[α]anthracene) Breast ancer in Rats.** (Eng) Reichle, F. A. (Dept. Surgery, emple Univ. Health Sciences Center, Philadelphia, PA, 19140); Noval, J. J.; Obando, M.; Rao, N.; Ryzlak, M. T.; Reichle, R. M. *J Surg Res* 24(6): 563-569; 1978.

The effect of treatment with small quantities of Sprague-Dawley (Holtzman) rat fetal tissue on mammary tumor development was studied in 8-wk-old syngeneic females given a single intragastric instillation of 7,12-dimethylbenz(a)anthracene (DMBA: 10 mg in 1 ml sesame oil). Each rat received two injections of live or dead fetal tissue suspensions (1 g/10 ml) given at the same time: 0.2 ml sc and 0.1 ml ip. The live tissue suspension contained 60%-70% living cells; the dead tissue had been killed by freeze-thawing and homogenization. The dead tissue homogenates were administered weekly for 3 wk prior to and/or after DMBA or for 5 wk starting at the time of DMBA administration. The live tissue suspensions were administered according to the 5-wk schedule. Treatment with dead fetal tissue significantly accelerated the appearance of the DMBA-induced tumors, but treatment with live tissue greatly suppressed tumor development and caused a high rate of tumor regression. Only 1/7 rats given five weekly injections of live tissue had a palpable tumor at 15 wk and, in this group, only eight tumors were found at autopsy at 26 wk. In another experiment, breast tumor development was not altered by live or dead fetal tissue inoculated 2 wk before DMBA treatment. These results are discussed in terms of host immunologic reactions. (23 refs)

78-3192 **The Effect of Levamisole on Experimental Carcinogenesis of Rat Submandibular Gland.** (Eng) Pisanty, S. (Dept. Oral Medicine and Oral Pathology, Harvard Sch. Dental Medicine, Boston, MA, 02115); Eisenberg, E.; Shklar, G. *Arch Oral Biol* 23(2): 131-135; 1978.

The effect of levamisole, a nonspecific enhancer of cellular immunity, on the development of 9,10-dimethyl-1,2-benzanthracene (DMBA)-induced submandibular gland tumors was studied in four groups of male albino rats, 30 or 60 days old. Group 1 animals (10) received a DMBA pellet implanted in the right submandibular gland, and they were inoculated sc with 0.6 mg/day levamisole on three consecutive days every 2 wk. Group 2 animals (10) received DMBA implants but no levamisole. Groups 3 and 4 were levamisole-treated and untreated controls. Although DMBA induced epidermoid carcinomas in both Groups 1 and 2, the tumors developed approx 3 wk later in the Group 1 animals. In addition, the tumors in this group were less anaplastic and less invasive than those in Group 2. Age had no apparent effect on the carcinogenic process. (24 refs)

78-3193 **Species-specific Cellular DNA-binding Proteins Expressed in Mouse Cells Transformed by Chemical Carcinogens.** (Eng) Sen, A. (Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD, 20014); Todaro, G. J. *Proc Natl Acad Sci USA* 75(4): 1647-1651; 1978.

Mouse cells transformed by DNA and RNA tumor viruses and by chemical carcinogens were examined for the presence of specific DNA-binding proteins by DNA-cellulose chromatography. Using mouse DNA-cellulose, single-stranded DNA-binding proteins were obtained from two clones of BALB/3T3 cells transformed by the chemical carcinogens dimethylbenzanthracene (DMBA) and methylcholanthrene. Simian virus 40 transformants also had a DNA-binding protein, ie, the viral tumor (T) antigen, that bound to mouse and human DNA with comparable affinity. Kirsten or Moloney sarcoma virus-transformed cells and two other clones transformed by DMBA or benzo(a)pyrene showed no difference in DNA-binding protein pattern, compared with the untransformed parental cell. The DNA-binding proteins isolated from the chemically transformed cell clones were between 25,000 and 30,000 daltons by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. These cellular T proteins bound to the homologous mouse cellular DNA with a higher affinity than to heterologous human cellular DNA. (26 refs)

78-3194 **Formation of 7,12-Dimethylbenz[a]Anthracene-DNA Adducts in 7,8-Benzoflavone-treated Hamster Embryo Cells.** (Eng) Baird, W. M. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA, 19104); Diamond, L. *Chem Biol Interact* 20(2): 181-190; 1978.

The effects of pretreating secondary cultures of Syrian hamster embryo cells with 7,8-benzoflavone (7,8-BF), a potent inhibitor of the mixed-function oxidase enzyme system, on the metabolism of 7,12-dimethylbenz(a)anthracene (DMBA) and the formation of DMBA-DNA adducts were determined. 7,8-BF pretreatment [0.25, 0.5, or 1.0 nanomole (nmol)/ml] inhibited both the metabolism of DMBA (0.5 nmol/ml) and the formation of adducts. The DMBA-deoxyribonucleoside adducts from 7,8-BF-treated cultures had the same elution profiles on Sephadex LH-20 columns as those from cultures exposed to DMBA alone, but 7,8-BF-treated cultures contained smaller amounts of DMBA-DNA adducts per milligram of DNA. With increasing 7,8-BF concentrations, the decrease in the amount of DMBA-DNA adducts per milligram of DNA was logarithmic with respect to the decrease in the amount of DMBA metabolized. These findings suggest that more than one metabolic step is required for the binding of DMBA to DNA in hamster embryo cells. (31 refs)

78-3195 **Inhibition of Polycyclic Aromatic Hydrocarbon-induced Neoplasia by Naturally Occurring Indoles.** (Eng) Wattenberg, L. W. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN, 55455); Loub, W. D. *Cancer Res* 38(5): 1410-1413; 1978.

The effects of indole-3-carbinol, 3,3′-diindolylmethane, and indole-3-acetonitrile on 7,12-dimethylbenz(a)anthrace (DMBA)-induced mammary tumors in female Sprague-Dav ley rats and on benzo(a)pyrene (BP)-induced forestomach tumors in female ICR/Ha mice were investigated. These i doles occur in edible cruciferous vegetables such as brusse sprouts, cabbage, cauliflower, and broccoli. DMBA was a ministered by po intubation at a dose of 12 mg; BP was a ministered by po intubation in 1-mg doses 2 x/wk for 4 w or in 0.3-mg doses 3 x/wk for 8 wk. When given by po intuba tion 20 hr prior to DMBA administration, indole-3-carbin and 3,3′-diindolylmethane inhibited mammary tumor forma tion; indole-3-acetonitrile was inactive. Indole-3-carbinol ad ded to the diet for 8 days prior to DMBA challenge inhibite mammary tumor formation but indole-3-acetonitrile did no Dietary administration of all three indoles inhibite BP-induced forestomach neoplasia. (26 refs)

78-3196 **Separation of Two Forms of Cytochrome P-45 From Hepatic Microsomes of 1,2,3,4-Dibenzanthracene (DBA)-Pretreated Little Skates (*Raja erinacea*) (Meeting Abstract).** (Eng) Elmamlouk, T. H. (Marine Pharmacology and Biomedicine Section, Lab. Pharmacology NIEHS, Research Triangle Park, NC, 27709); Philpot, R M.; Bend, J. R. *Environ Health Perspect* 20: 229; 1977. (n refs.)

78-3197 **Ultrastructure and Biological Markers of Neoplastic Change in Adult Mouse Epithelial Cells Transformed In Vitro.** (Eng) Knowles, M. A. (Dept. Cellula Pathology, Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2A 3PX, England); Franks, L. M. *Br J Cancer* 37(4): 603-611; 1978.

The ultrastructure and biological markers indicative of neoplastic change in five in vitro-transformed mouse submandibular gland epithelial cell lines were determined. Four of these lines had been transformed by 24 hr treatment with 0.1 μg/ml 7,12-dimethylbenz(a)anthracene; the fifth was isolated from a dimethyl sulfoxide-treated culture. In vitro acinus formation occurred in all lines, and well-differentiated epithelial cells showing epithelial microvilli, desmosomes, and cytoplasmic tonofilaments were present. None of the cells showed the specific ultrastructural features of normal differentiated submandibular gland ducts. All lines formed colonies in semisolid agar and on confluent monolayers of BALB/c 3T3 cells, and all lacked density-dependent inhibition of growth, as demonstrated by a random distribution of thymidine labeling throughout growing cultures. These three growth properties appear to be reliable in vitro markers for epithelial neoplastic transformation in this system; however, colony-forming efficiency in agar was lower than that reported for many transformed mesenchymal cells. (28 refs)

8-3198 **Interindividual Variation in the Responses of Cultured Human Lymphocytes to Exposure rom DNA Damaging Chemical Agents. Interindividual ariation to Carcinogen Exposure.** (Eng) Pero, R. W. (Wal- enberg Lab., Nucleic Acid Biochemistry, Univ. Lund, Fack 031, S-220 07 Lund 7, Sweden); Bryngelsson, C.; Mitelman, .; Kornfalt, R.; Thulin, T.; Norden, A. *Mutat Res* 53(3): 27-341; 1978.

ndividual variations in the in vitro response of human lym- hocytes to N-acetoxy-2-acetylaminofluorene (NA-AAF) nd 7,12-dimethylbenz(a)anthracene (DMBA) were deter- nined by measuring (1) the differential stimulation of un- cheduled DNA synthesis after 1 hr exposure to 10 μM NA-AAF, (2) the level of NA-AAF-induced chromosome berrations remaining after 8 hr of DNA repair synthesis, nd (3) the level of ^{3}H-DMBA bound to DNA after 18 hr ncubation of resting lymphocytes in 5 μM DMBA. Lym- hocytes from 92 men, 90 women, and 20 children were used. ll three parameters indicated that there were individual ariations in the response of lymphocytes to the carcinogens, nd they were correlated with population differences in age, ex, blood pressure, and mortality rates. Males had a greater otential to accumulate DNA damage than females, regard- ess of the sampled population. DNA damage potentials in- reased with increasing age, blood pressure, or mortality ates. The in vitro response of mature granulocytes to a 10- μM NA-AAF dose, as estimated by ^{3}H-thymidine incorpora- ion from unscheduled DNA synthesis, was much lower than he lymphocyte response. Nevertheless, individual variations n granulocyte NA-AAF-induced unscheduled DNA synthe- is paralleled the interindividual fluctuations observed in ymphocyte responses to NA-AAF. (37 refs)

8-3199 **Formation of Free Radicals from Carcinogenic Benz[c]acridines in the Presence of Proteins.** Eng) Okano, T. (Pharmaceutical Inst., Aobayama, Sendai 80, Japan); Horie, T.; Iwaizumi, M.; Motohashi, N. *Gann* 9(1): 109-114; 1978.

he formation of free radicals from carcinogenic and noncar- inogenic benz[c]acridine (BCA) derivatives was studied in he presence of proteins. The noncarcinogenic compounds vere BCA, 11-methyl-BCA, and 9-methyl-BCA; the carcino- enic compounds were 7-methyl-BCA, 7,11-dimethyl-BCA, ,9-dimethyl-BCA, 7,10-dimethyl-BCA, and 7,9,10-trimeth- l-BCA. When mixtures of the BCAs and protein were tirred for long periods and shielded from light, the BCA's vere converted into free radicals. Bovine serum albumin had he greatest effect on accelerating free-radical formation; a maller effect was noted with globulin, histone, and DNase. he g value of the free radicals thus obtained was 2.005. The ntensity of the electron spin resonance (ESR) signals of the ree radicals from carcinogenic derivatives was higher than hat of the free radicals from noncarcinogenic derivatives. here was a correlation between the ESR signal intensity of he free radicals, the charge of the K-region or ring nitrogen of the BCA's, and the carcinogenicity of the BCA's. These findings suggest that free radicals are important in BCA carcinogenesis. (17 refs)

78-3200 **Induction of Antibodies Against Carcinogenic Polycyclic Aromatic Hydrocarbons.** (Eng) Moolten, F. L. (Boston Univ. Sch. Medicine, 80 E. Concord St., Boston, MA, 02118); Capparell, N. J.; Boger, E.; Mahathalang, P. *Nature* 272(5654): 614-616; 1978.

Studies were conducted on the feasibility of inducing antibodies against carcinogenic polycyclic aromatic hydrocarbons (PAH) using an analog of the PAH carcinogen dimethylbenzanthracene (DMBA) substituted with a fluorine moiety to abrogate carcinogenicity. The DMBA analog, 5-fluoro-12-methylbenzanthryl-7-acetic acid (FMBAAA), was nontoxic, did not induce tumors in C57Bl/6J mice, and was nonmutagenic in the Ames bacterial mutagenesis assay. Guinea pigs immunized with GMBAAA yielded antisera capable of binding FMBAAA and also DMBA. One serum was also able to bind to benzo(a)pyrene (BP) and benzo(rst)pentaphene. No binding was observed with the nonaromatic polycyclic compound cholic acid. When globulin fractions from these sera were tested in an in vitro cytotoxicity assay using hamster embryo cells, the antisera completely abrogated the toxicity of 0.01 μg DMBA and greatly reduced the toxicity of 0.05 μg DMBA. The ability of mice immunized with FMBAAA to excrete fed ^{3}H-BP as γ-globulin-bound complexes was significantly higher than that of unimmunized controls. Extrapolation of these data to humans suggests the feasibility of inducing antibody levels capable of binding, and perhaps neutralizing, carcinogens present in the environment in low, but not high, concentrations. (19 refs)

78-3201 **AHH Activity Induced by DMBA and its Binding to DNA of Mammary Cells in Organ Culture (Meeting Abstract).** (Eng) Telang, N. T. (Tumor Biology Lab., Univ. Nebraska, Lincoln, NB, 68588); Kundu, A. B.; Banerjee, M. R. *In Vitro* 14(4): 337; 1978. (no refs)

78-3202 **Steroid Metabolism in the Normal Mammary Gland and in the Dimethylbenzanthracene-induced Mammary Tumor of Rats.** (Eng) Mori, M. (Natl. Inst. Radiological Sciences, 9-1, Anagawa-4-Chome, Chiba-shi 280, Japan); Tominaga, T.; Tamaoki, B. *Endocrinology* 102(5): 1387-1397; 1978.

The in vitro metabolism of progesterone by female Sprague-Dawley rat normal mammary glands and mammary tumors induced by 9,10-dimethyl-1,2-benzanthracene (DMBA; 25 mg/kg, iv) was determined. Following incubation of ^{14}C-progesterone with cell-free homogenates of tumor tissue, 20α-hydroxy-4-pregnen-3-one, 5α-pregnane-3,20-dione, 20α-hydroxy-5α-pregnan-3-one, 3α-hydroxy-5α-pregnan-20-one, and 5α-pregnane-3α,20α-diol were identified as the metabolites. In normal mammary tissue, however, 4-pregnene-3α,20α-diol was isolated in addition to 5α-reduced and 3α- and 20α-hydroxy metabolites. When labeled testosterone was used as a substrate, 5α-dihydrotestosterone and 5α-androstane-3α,17β-diol were the metabolites of the mammary tumor. In the normal mammary gland, only 4-androstene-3α,17β-diol was formed. Although the enzyme activities relevant to the metabolism varied among the tumors examined, the activity of 20α-hydroxysteroid dehydrogenase in the mammary tumor was significantly lower than that in the normal gland. However, the activity of 5α-reductase was higher in some of the mammary tumors than in the normal gland. The 5α-reductase activity in the normal gland was mostly localized in the crude microsomal fraction, but the same enzyme activity in the tumor was detected in all the organelle fractions. The activities of 20α-hydroxysteroid dehydrogenase and NADPH-linked 3α-hydroxysteroid dehydrogenase activities were found mainly in the cytosol fractions of the tumor and normal tissue. The latter was detected only in the cytosol fraction of the normal gland, but was detected in all subcellular fractions of the tumor that were examined. (38 refs)

78-3203 **Cyclic Adenosine 3′,5′-Monophosphate-Binding Protein: Role in Ovariectomy-induced Regression of a Hormone-Dependent Mammary Tumor in Sprague-Dawley Female Rats: Brief Communication.** (Eng) Cho-Chung, Y. S. (Lab. Pathophysiology, NCI, NIH, Public Health Service, U.S. Dept. Health, Education, and Welfare, Bethesda, MD, 20014); Bodwin, J. S.; Clair, T. *J Natl Cancer Inst* 60(5): 1175-1178; 1978.

The relationship between cyclic AMP- and estrogen-binding activities was investigated in 7,12-dimethylbenz(a)anthracene-induced mammary carcinomas in Sprague-Dawley rats during tumor growth and regression following ovariectomy. Within 3 days after ovariectomy, cyclic AMP binding increased markedly and estrogen binding decreased in regressing tumors. These changes were reversed when tumor growth was resumed following the sc injection of estradiol-17β. The data suggest the possible involvement of cyclic AMP binding in the growth control of a hormone-dependent mammary tumor. (26 refs)

78-3204 **Lack of Tumor-promoting Ability of Certain Environmental Chemicals in a Two-Stage Mouse Skin Tumorigenesis Assay.** (Eng) Berry, D. L. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); DiGiovanni, J.; Juchau, M. R.; Bracken, W. M.; Gleason, G. L.; Slaga, T. J. *Res Commun Chem Pathol Pharmacol* 20(1): 101-108; 1978.

Butylated hydroxytoluene (BHT), butylated hydroxyanisole (BHA), Aroclor 1254 (polychlorinated biphenyl, PCB), Firemaster-6 (polybrominated biphenyl, PBB), and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) were tested on CD1 female mice for their tumor-promoting ability following application of 0.2 ml of a 200-nanomole soln of 7,12-dimethylbenz(a)anthracene (DMBA). At a dose of 1 mg twice weekly, neither BHT nor BHA promoted skin tumors; no tumors were induced when they were tested for carcinogenicity without DMBA initiation. PCB and PBB, at a dose of 100 μg twice weekly after DMBA application, were also inactive as promotors. TCDD was tested at a dose of 0.1 μg twice weekly; it did not promote DMBA-induced tumors and did not act as a carcinogen by itself. It did, however, result in skin rashes. None of the compounds significantly increased the intrafollicular epidermis. If the doses of the compounds were increased to levels that would lead to epidermal proliferation, their toxicity would interfere with the carcinogenic response prior to termination of the test. These results indicate that dosage may be an important factor in promotion, since some of these compounds are promotors in pulmonary and hepatic systems. (22 refs)

78-3205 **The Bacterial Mutation Test.** (Eng) Anderson, D. (No Affiliation Given); Styles, J. A. *Br J Cancer* 37(6): 924-930; 1978.

The Ames bacterial-mutation test was evaluated for its ability to screen organic chemicals for potential carcinogenicity. Four strains of *Salmonella typhimurium* (TA1535, TA1538, TA98, and TA100) were exposed to a total of 120 chemicals. The highest mean number of revertants at any dose was used to assess carcinogenic potential. The overall accuracy of the test in predicting carcinogenicity was 91% for carcinogens and 94% for noncarcinogens, indicating that the Salmonella mutation system is useful for predicting the carcinogenicity of organic chemicals. Many compounds (eg, 2-aminoanthracene, 6-aminochrysene, 2-naphthylamine, and 4-aminobiphenyl) were not specific in the type of mutation they caused; ie, they were not specifically base-pair or frameshift mutagens. No attempt was made to rank the chemicals in order of carcinogenic potency on the basis of mutagenic activity. (14 refs)

78-3206 **The Identification of 3-Methylcholanthrene-9,10-dihydrodiol as an Intermediate in the Binding of 3-Methylcholanthrene to DNA in Cells in**

Culture. (Eng) King, H. W. (Chemical Carcinogenesis Div., Inst. Cancer Res., Pollards Wood Res. Station, Nightingales Lane, Chalfont St. Giles, Buckinghamshire HP8 4SP, England); Osborne, M. R.; Brookes, P. *Chem Biol Interact* 20(3): 367-371; 1978.

Two 3-methylcholanthrene (3MC) metabolites, previously shown to be precursors of the DNA-bound form of 3MC observed in cultured embryo cells, were prepared by microsomal metabolism and isolated by high-pressure liquid chromatography (HPLC). From HPLC and mass spectrometric analyses of the metabolites and from comparison with the fluorescence spectra of the five possible dihydrodiols of alkylated benzanthracene, it was inferred that one of the metabolites was 1- or 2-hydroxy-3MC-9,10-dihydrodiol and the other, 3MC-9,10-dihydrodiol. (5 refs)

78-3207 **Genetic Variation in Metabolism of Chemical Carcinogens Associated with Susceptibility to Tumorigenesis.** (Eng) Nebert, D. W. (Developmental Pharmacology Branch, Natl. Inst. Child Health and Human Development, NIH, Bethesda, MD); Levitt, R. C.; Pelkonen, O. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 12 pp.; 1978. (no refs)

78-3208 **Mouse-Strain Difference in Immunoprophylactic and Immunotherapeutic Effects of BCG on Carcinogen-induced Autochthonous Tumors.** (Eng) Tokunaga, T. (Dept. Tuberculosis, Natl. Inst. Health, Shinagawa-ku, Tokyo 141, Japan); Yamamoto, S.; Nakamura, R. M.; Kurosawa, A.; Murohashi, T. *Jpn J Med Sci Biol* 31(2): 143-154; 1978.

The immunoprophylactic and immunotherapeutic effects of BCG on 3-methylcholanthrene (3-MC)-induced tumors were compared in female Swiss-derived SWM/Ms and C3H/He mice. One group of each strain was treated with 5 x 10^7 viable units of BCG id 2 wk before 3-MC (0.5 mg sc), and one group received 3-MC only (controls). In C3H mice, the first tumors appeared 5 wk after 3-MC injection. The number of tumor bearers increased rapidly from 8 to 11 wk, and at 20 wk, tumor incidence was nearly 100% in both groups. In SWM mice, the first tumor appeared at 8 wk, and tumor incidence at 30 wk was approx 80% and 90% in the SWM-BCG and control groups, respectively. The number of tumor bearers increased more slowly in the former. Differences in tumor incidence between the two SWM groups were significant at 9-12, 14, and 18 wk after 3-MC. However, once the tumors were established, they grew progressively and killed the hosts of both strains. Intratumor injection of BCG into nodules > 5 mm in diameter in all tumor bearers resulted in tumor regression or retardation or prolongation of survival time in most SWM-BCG mice, but not in C3H mice. Some SWM controls experienced tumor retardation. It was noted that 4/171 C3H-BCG and 7/35 SWM-BCG mice, but none of the controls, died within 2 wk after BCG injection. A peritoneal macrophage disappearance test and a footpad reaction test showed that C3H and SWM mice have different host responses to BCG: C3H mice are low responders, and SWM mice are high responders. It is not known whether the high tumor incidence in C3H mice is linked with low responsiveness to BCG and/or histocompatibility. The results indicate that the host immune response to BCG is a critical factor in BCG antitumor activity. (25 refs)

78-3209 **Fluorescence Study of DNA-binding Metabolites of Benzo(a)pyrene Formed in Hepatocytes Isolated from 3-Methylcholanthrene-treated Rats.** (Eng) Jernstrom, B. (Dept. Forensic Medicine, Karolinska Institutet, S-104 Stockholm 60, Sweden); Orrenius, S.; Undeman, O.; Graslund, A.; Ehrenberg, A. *Cancer Res* 38(8): 2600-2607; 1978.

Hepatocytes and liver microsomes isolated from 3-methylcholanthrene-treated Sprague-Dawley rats metabolized benzo(a)pyrene (BP) to products that bound to endogenous DNA and exogenous calf thymus DNA, respectively. By a sensitive fluorescence technique, it was possible to characterize the major DNA-binding metabolite in hepatocytes as being produced by further metabolism of 9-hydroxybenzo(a)pyrene (9-OH-BP). In microsomes, two products binding to calf thymus DNA were recovered, a major species formed by activation of 9-OH-BP and a minor fraction formed by further metabolism of 7,8-dihydro-7,8-dihydroxybenzo(a)pyrene (BP 7,8-dihydrodiol). The available evidence indicates that the ultimate products responsible for binding to DNA were identical to 9-OH-BP 4,5-oxide and BP 7,8-dihydrodiol 9,10-oxide, respectively. The data further suggest that metabolic activation of 9-OH-BP results in the quite different DNA:metabolite complexes. The former product(s) seems to be strongly associated with hydrophobic regions in DNA, but the latter metabolite(s) appears to be more exposed to the exterior. (45 refs)

78-3210 **Inducibility of Activation and/or Detoxication Enzymes in Benzo(a)pyrene Metabolism by 3-Methylcholanthrene.** (Eng) Nemoto, N. (Dept. Experimental Pathology, Cancer Inst., 1-37-1, Kami-Ikebukuro, Toshima-ku, Tokyo 170, Japan); Takayama, S. *Toxicol Lett* 1(5/6): 247-252; 1978.

The inducibility of hepatic aryl hydrocarbon hydroxylase (AHH) and two conjugating enzymes, uridine diphosphate-glucuronyltransferase (UDP-G) and sulfotransferase (ST), by 3-methylcholanthrene (MC) was investigated in several strains of mice and rats. There are various types of conjugation in benzo(a)pyrene (BP) metabolism, and the extent of these conjugations is considered to play an important role in the carcinogenicity of BP. The experimental animals were given a single ip injection of 80 mg/kg MC and then killed 24 hr later. UDP-G and ST activities were determined using 3-OH-BP as substrate. Of the five inbred strains, C57BL/6, C3H/He, and BALB/c mice (responsive strains) had increased AHH activity following MC treatment, but DBA/2 and AKR mice (nonresponsive strains) did not. UDP-G and ST activities were not altered in all five strains. Wistar and ACI rats and ICR/JCL mice, which were produced by closed-colony mating, were responsive to AHH and UDP-G induction by MC; their ST activity was not affected. Thus, in some species and strains, induction of AHH and UDP-G may be regulated in parallel. (29 refs)

78-3211 **Comparative Incorporation of Uridine-^{3}H into Nucleolar RNA of Mouse Subcutaneous and Skin Tissues at Early Times after 20-Methylcholanthrene Administration.** (Eng) Gulati, P. K. (Dept. Biophysics, Panjab Univ., Chandigarh-160014, India); Dubey, D. P. *Experientia* 34(3): 396-397; 1978.

^{3}H-Uridine uptake by the nucleolar RNA of the skin and sc tissues was investigated in 55 C57BL/Bcr female mice after they were inoculated with a single carcinogen dose of 20-methylcholanthrene (20-mc: 1 mg in 0.2 ml olive oil sc). Treated mice were given an ip injection of ^{3}H-uridine (6μ Ci/g) and decapitated 2 hr later. ^{3}H-Uridine incorporation into the nucleolar RNA of the skin and sc tissue around the injection site was increased 17 and 3 times, respectively, 24 hr after 20-mc injection. The uptake was normal at 37 hr after carcinogen injection. The functional significance of these changes has not been established, but they may indicate the importance of nucleolar function in the carcinogenic process. (7 refs.)

78-3212 **Carcinogenesis in Immunologically Privileged Sites.** (Eng) Ziegler, M. M. (Dept. Surgery, Univ. Pennsylvania, Philadelphia, PA, 19104); Barker, C. F. *Fed Proc* 37(7): 2057-2058; 1978.

Hamsters were treated with 3-methylcholanthrene (3-MC) either topically, by subepithelial injection, or by subepithelial implantation to determine whether de novo chemical induction of tumors might be easily achieved in sites where immunological surveillance is impaired. Of nine animals receiving topical applications of 10 mg/ml 3-MC to ear skin inlaid in autografted cheek pouches, seven developed papillomas after 11-116 days (mean, 48 days). Similar treatment of seven hamsters with vehicle only resulted in only one tumor. When 3-MC was applied to the autografted cheek pouch itself, 3/5 animals developed tumors. Dye experiments indicated that the cheek pouch and inlaid ear skin in these animals had impaired lymphatic drainage. In sites of normal lymphatic drainage (ear skin autografted to trunk skin), 0/13 animals treated with 3-MC developed tumors. Fibrosarcomas developed in 8/8 hamsters inoculated with 0.8-1.6 mg 3-MC in autografted cheek pouches after 28-169 (mean 121 days). Subepithelia in the trunk area of nine animals produced tumors in only two, with latent periods of 240 and 316 days. When filter paper impregnated with 5% 3-MC was subepithelially implanted in cheek implants of five hamsters, two developed tumors. Of four animals receiving implantations in areas of normal lymphatics, three developed tumors. The mean peripheral WBC count in eight normal hamsters was 4,431/mm^3; this increased to 6,843/mm^3 after a dose of 2 mg 3-MC and 7,733/mm^3 after 3 mg 3-MC. Topical administration of 3-MC for 17 consecutive days did not change the median survival time of MHA hamster skin grafts on LHC hamsters from that of untreated control LHC animals. These findings support the theory that tumor formation is more likely when immunity is impaired. (10 refs)

78-3213 **Carcinogenetic Process in Experimental Squamous Cell Carcinoma in Canine Lungs.** (Jpn) Hayata, Y. (Dept. Surgery, Tokyo Medical Coll., Skinjuku-ku, Tokyo 160, Japan); Kato, H.; Chow, M. C.; Tachibana, M.; Hayashi, N.; Tawara, M.; Kawauchi, T.; Seo, Y.; Amemiya, R. *Nippon Kyobu Shikkan Gakkai Zassh* 15(11): 759-768; 1977.

Carcinogenesis was studied in two adult beagles and four adult mongrel dogs given an injection of 50 mg 20-methylcholanthrene at the bifurcation of the apical and cardiac bronchi every 7 days. Proliferation of the basal cells accompanied by the appearance of atypical nuclei or nuclear division was noted immediately after injection; these cells were replaced by metaplastic cells after 1 wk. All dogs exhibited mildly atypical squamous cell metaplasia after 1-4 wk and moderately atypical squamous cell metaplasia after 2-8 wk. Severely atypical squamous cell metaplasia was noted in four dogs 5-9 wk after the beginning of treatment, and three dogs developed carcinoma in situ between 8 and 14 wk. Invasive carcinoma developed after 18-22 wk in three dogs. These findings suggest that squamous cell metaplasia is an important precursor of squamous cell carcinoma. (20 refs)

78-3214 **Prostaglandin Synthetase Dependent Activation of 7,8-Dihydro-7,8-dihydroxy-benzo(a)pyrene to Mutagenic Derivatives.** (Eng) Marnett, L. J. (Dept. Chemistry, Wayne State Univ., Detroit, MI, 48202); Reed, G. A.; Dennison, D. J. *Biochem Biophys Res Commun* 82(1): 210-216; 1978.

The *Salmonella typhimurium* tester strain TA98 was used to study the prostaglandin synthetase-dependent production of mutagenic derivatives from benzo(a)pyrene (BP) and its dihydrodiol metabolites. There was no increase in the reversion rate of TA98 following incubation with BP in the presence of prostaglandin synthetase and arachidonic acid, and the presence of the bacteria did not affect the oxidation of BP to quinones. Thus, the lack of mutagenicity was not due to an inhibition of prostaglandin synthetase-dependent BP oxidation by the bacterial preparation. The addition of BP-7,8-diol, but not BP-4,5-diol or BP-9,10-diol, to the assay system resulted in a > 10-fold increase in the number of revertants. The activation of BP-7,8-diol was markedly reduced by the omission of arachidonic acid, and inhibited by the prostaglandin synthetase inhibitor indomethacin, and it exhibited a dose-response relationship with respect to the diol. Inclusion of 100 μM of the antioxidant butylated hydroxyanisole in the assay mixture inhibited by 89% the increase in the number of revertants induced by BP-7,8-diol. The data suggest that during prostaglandin biosynthesis, BP-7,8-diol was cooxidized to a BP-diol epoxide, although it is possible that BP-7,8-diol was activated by a unique pathway to strongly mutagenic derivatives that were not diol epoxides. (20 refs)

78-3215 **Metabolism of trans-9,10-Dihydroxy-9,10-dihydrobenzo(a)pyrene Occurs Primarily by Arylhydroxylation Rather than Formation of a Diol Epoxide.** (Eng) Thakker, D. R. (Section Oxidative Mechanisms, Lab. Chemistry, Natl. Inst. Arthritis, Metabolism, and Digestive Diseases, NIH, Bethesda, MD, 20014); Yagi, H.; Lehr, R. E.; Levin, W.; Buening, M.; Lu, A. Y.; Chang, R. L.; Wood, A. W.; Conney, A. H.; Jerina, D. M. *Mol Pharmacol* 14(3): 502-513; 1978.

The oxidative metabolism of trans-9,10-dihydroxy-9,10-dihydrobenzo(a)pyrene was investigated in vitro using microsomal protein preparations from 3-methylcholanthrene-treated Long-Evans rats to determine whether 9,10-diol 7,8-epoxides are formed, and the mutagenic activities of synthetic benzo(a)pyrene (BP) 9,10-diol 7,8-epoxide derivatives were evaluated. Unlike BP 7,8-dihydrodiol, the trans-9,10-dihydrodiol was metabolized primarily to a phenolic derivative rather than a diol epoxide. Chemical and spectral studies established that the phenolic hydroxyl group had been introduced at either position 1 or 3 of the 9,10-dihydrodiol. The weak inherent mutagenic activity of the synthetic 9,10-diol 7,8-epoxides and the very low degree of their metabolic formation explain why metabolic activation of BP 9,10-dihydrodiol in the presence of *Salmonella typhimurium* resulted in a weak mutagenic response compared with studies using the 7,8-dihydrodiol as substrate. Chronic application of 0.15 micromole of BP 9,10-dihydrodiol to the backs of C57BL/6J mice once every 2 wk for 60 wk failed to produce tumors, but the same treatment with BP resulted in a 97% tumor incidence. (37 refs)

78-3216 **Metabolism of Benzo(a)pyrene in Animals with High Aryl Hydrocarbon Hydroxylase Levels and High Rates of Spontaneous Cancer.** (Eng) Busbee, D. L. (Dept. Biology, North Texas State Univ., Denton, TX, 76203); Guyden, J.; Kingston, T.; Rose, F. L.; Cantrell, E. T. *Cancer Lett* 4(2): 61-67; 1978.

Aryl hydrocarbon hydroxylase (AHH) induction and benzo(a)pyrene (BP) metabolism were studied in salamanders (*Ambystoma tigrinum*) removed from a sewage-polluted pond, a population of animals with a high level of spontaneous tumors. Some of these animals were analyzed immediately for AHH content, and others were placed in clean, aerated water. Salamanders that had been maintained in the laboratory were induced with a single ip injection of 3-methylcholanthrane (MC; 10 mg/kg). Animals taken directly from the pond were found to have hepatic microsomal AHH levels about 80% as high as those of animals inoculated with MC. After animals were held in clean water for several months, their AHH levels were as low as those of control untreated laboratory salamanders. Microsomes from the salamanders were reacted with BP, and the metabolites were separated by high-pressure liquid chromatography. BP metabolites produced by salamander liver microsomes were qualitatively similar to those reported for mammals; however, the percentage of the metabolites appeared to differ significantly. Treatment of induced salamander microsomes with cyclohexene oxide or 1,2-epoxy-3,3,3-trichloropropane during incubation significantly changed the metabolic profile. The greatest effect was decreased diol production, indicating a normal epoxide hydrase conversion of intermediate compounds to the diol form. These findings suggest that an inducing agent is present in relatively high concentrations in the polluted water, and that a correlation appears to exist between high AHH induction, the presence of polycyclic hydrocarbon pollutants, and the high spontaneous cancer rate in salamanders. (25 refs)

78-3217 **Molecular and Cellular Events Associated with the Action of Initiating Carcinogens and Tumor Promoters.** (Eng) Weinstein, I. B. (Inst. Cancer Res. and Div. Environmental Science, Columbia Univ., New York, NY). *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 27-28 pp.; 1978. (no refs)

78-3218 **Properties of Aryl Hydrocarbon (Benzo[a]Pyrene) Hydroxylase in Lung Microsomes of Mice.** (Eng) Watanabe, M. (Res. Inst. Tuberculosis and Cancer, Tohoku Univ., Seiryo-machi 4-1, Sendai 980, Japan); Konno, K.; Sato, H. *Gann* 69(1): 1-8; 1978.

The effects of a single po dose of 1 mg/kg benzo(a)pyrene

(BP) or a single ip dose of 1 mg/kg 3-methylcholanthrene (MC) on aryl hydrocarbon hydroxylase activity in C3H/He and DBA/2 mouse lung were determined. Regardless of animal strain, BP increased AHH activity in the lung, but not in the liver, of treated mice. However, AHH activity was increased in the intestine and skin of C3H/He mice but not DBA/2 mice. Comparison of the properties of enzymes induced by MC with the constitutive enzymes in animals treated with corn oil only indicated that the two enzymes had similar pH activity curves and similar apparent Km for NADPH and NADH. However, the constitutive enzyme had a higher Km for BP (25 μM) than the induced enzyme (6 μM). 5,6-Benzoflavone, 7,8-benzoflavone, and 2,2′-bipyridine inhibited the activity of the two enzymes similarly, but N-benzyl-N,α-dimethylphenethylamine hydrochloride and 2-diethylaminoethyl diphenylpropylacetate inhibited the activity of the constitutive enzyme more severely than that of the induced enzyme. Cyclohexene oxide, 1,1,1-trichloropropane oxide, and leupeptin slightly inhibited the activity of the constitutive enzyme, but enhanced the activity of the induced enzyme. The significance of these differences to the carcinogenicity of polycyclic hydrocarbons is discussed briefly. (36 refs)

78-3219 **Formation of DNA-binding Products from Isolated Benzo[α]pyrene Metabolites in Rat Liver Nuclei.** (Eng) Jernstrom, B. (Dept. Forensic Medicine, Karolinska Institutet, S-104 01 Stockholm 60, Sweden); Vadi, H.; Orrenius, S. *Chem Biol Interact* 20(3): 311-321; 1978.

Benzo(a)pyrene (BP) or one of its metabolites (30 μM) was incubated with isolated liver nuclei from 3-methylcholanthrene-treated rats, and the reaction products were analyzed by high-pressure liquid chromatography. In the presence of NADPH, the liver nuclei metabolized 3- and 9-hydroxy-BP (3-OH-BP, 9-OH-BP) and 7,8-dihydro-7,8-dihydroxy-BP (BP 7,8-dihydrodiol) to products that bound to DNA. Max binding was obtained with BP 7,8-dihydrodiol as substrate. This binding was 10, 3, and 60 times higher than that occurring after incubation with BP, 9-OH-BP, and 3-OH-BP, respectively. Both 4,5-dihydro-4,5-dihydroxy-BP and 9,10-dihydro-9,10-dihydroxy-BP were also metabolized extensively by the nuclear fraction, but they did not give rise to DNA-binding products. It is concluded that the DNA-binding species derived from 9-OH-BP is 9-OH-BP 4,5-oxide and the species derived from BP 7,8-dihydrodiol is BP-7,8-dihydrodiol 9,10 oxide. (28 refs)

78-3220 **Studies on the Metabolism and Excretion of Benzo(a)pyrene in Isolated Adult Rat Hepatocytes.** (Eng) Jones, C. A. (Dept. Biochemistry, Univ. Surrey, Guildford, Surrey, GU2 5XH, England); Moore, B. P.; Cohen, G. M.; Fry, J. R.; Bridges, J. W. *Biochem Pharmacol* 27(5): 693-702; 1978.

Hepatocytes from male albino Wistar rats were isolated and used to study the metabolism of benzo(a)pyrene (BP), with particular attention being focused on the fate of the metabolites. BP was metabolized to both ethyl acetate-soluble and water-soluble metabolites. The former cochromatographed with 4,5-dihydro-4,5-dihydroxyBP, 7,8-dihydro-7,8-dihydroxyBP, 9,10-dihydro-9,10-dihydroxyBP, and 3-hydroxyBP and its sulfate ester, benzo(a)pyren-3-yl-hydrogen sulfate. Hydrolysis of the water-soluble metabolites with β-glucuronidase released ethyl acetate-soluble metabolites that cochromatographed with 7,8-dihydro-7,8-dihydroxyBP, 4,5-dihydro-4,5-dihydroxyBP, 9,10-dihydroxyBP, and 3-hydroxyBP. Initially, incubation resulted in only ethyl acetate-soluble metabolites that were partially released into the medium; with continued incubation, water-soluble metabolites were produced and released from the cells. The individual ethyl-acetate soluble metabolites showed significant distributional differences: monohydroxyBPs and (to a lesser extent) the sulfate esters of monohydroxyBPs accumulated intracellularly, 4,5-dihydro-4,5-dihydroxyBP and 7,8-dihydro-7,8-dihydroxyBP were fairly evenly distributed between the cells and the medium, and 9,10-dihydro-9,10-dihydroxyBP was found mainly in the medium. Significant radioactivity from labeled BP was irreversibly bound to cellular macromolecules. (33 refs)

78-3221 **Characteristics of Benzo(a)pyrene Metabolism and Cytochrome P-450 Heterogeneity in Rat Liver Nuclear Envelope and Comparison to Microsomal Membrane.** (Eng) Fahl, W. E. (Dept. Pharmacology, Univ. Wisconsin Medical Sch., Madison, WI, 53706); Jefcoate, C. R.; Kasper, C. B. *J Biol Chem* 253(9): 3106-3113; 1978.

Benzo(a)pyrene (BP) metabolism was compared in Holtzman rat liver nuclear envelopes and microsomes. The formation of BP metabolites was extremely low in the nuclear envelopes and, unlike the microsomes, it was not induced by phenobarbital (PB) pretreatment. However, after 3-methylcholanthrene pretreatment (3-MC), the metabolism increased more in the nuclear envelopes (60 times) than in the microsomes (5.5 times). Thus, the specific activities per nanomole of cytochrome P-450 equalized. Although the product distribution was similar in nuclear envelopes and microsomal membranes, a decrease in dihydrodiols and an increase in phenols were observed in the nuclear envelopes. 3-MC resulted in a 13-fold increase in BP 7,8-dihydrodiol metabolism in the nuclear envelope, and it favored the formation of the anti diol-oxide stereoisomer. Nuclear epoxide hydratase activity was not induced by 3-MC or by PB, which increased the enzyme activity threefold in the microsomes. NADPH-cytochrome c reductase was also induced only in the microsomes. Four separate forms of cytochrome P-450 were identifiable in the nuclear envelope by their type I difference spectra. PB markedly affected the levels of the individual nuclear cytochrome P-450 species without producing a net increase. In contrast, 3-MC doubled the net nuclear envelope cytochrome P-450 content, shifting the absorbance max to 448 nanometers. The absence of a microsomal, camphor-binding cytochrome P-450 species in nuclear envelope preparations indicated insignificant microsomal contamination (< 1%). (44 refs)

78-3222 **Effect of Bile Acids and Neutral Sterols on Benzo[a]pyrene-induced Tumorigenesis in Skin of Mice: Brief Communication.** (Eng) Watanabe, K. (Dept. Surgery, Akita Univ. Sch. Medicine, Akita, Japan); Narisawa, T.; Wong, C. Q.; Weisburger, J. H. *J Natl Cancer Inst* 60(6): 1501-1503; 1978.

A series of experiments was conducted to determine whether neutral sterols and bile acids are cocarcinogenic on female Swiss Ha:ICR mouse skin when applied together with benzo(a)pyrene (BP). A soln of 5 μg BP and 1 mg of the bile acid or sterol (except lithocholic acid, which was applied at a dose of 0.5 mg because of its low solubility) was applied thrice weekly to the skin of the mice for 60 wk. The animals were killed 63 wk after initiation of the experiment. Most of the chemicals had an inhibitory effect on skin tumor formation by BP. Unconjugated bile acids had higher inhibitory action than did conjugated bile acids. The inhibition decreased in the following order of acids: chenodeoxycholic, lithocholic, deoxycholic, and cholic. Within the group of conjugated acids, taurine conjugates were more effective inhibitors than were glycine conjugates. The neutral sterols cholesterol, coprostanol, and coprostanone showed moderately inhibitory effects on BP carcinogenesis. (23 refs)

78-3223 **Mutagenicity to Mammalian Cells in Culture by (+) and (-) trans-7,8-Dihydroxy-7,8-dihydrobenzo(a)pyrenes and the Hydrolysis and Reduction Products of Two Stereoisomeric Benzo(a)pyrene 7,8-Diol-9,10-epoxides.** (Eng) Huberman, E. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Yang, S. K.; McCourt, D. W.; Gelboin, H. V. *Cancer Lett* 4(1): 35-43; 1978.

The mutagenicity of benzo(a)pyrene (BP) and nine of its derivatives was tested by ouabain resistance in Chinese hamster V79 cells, which were derived from a subclone of V79-4 cells. The nine derivatives included the (+) and (-) enantiomers of trans-7,8-diol; (±)trans-7,8-diol; (7/8,9)-triol; (7,9/8)-triol; (7,10/8,9)-tetrol; (7/8,9,10)-tetrol; (7,9/8,10)-tetrol; and (7,9,10/8)-tetrol. Since V79 cells do not metabolize polycyclic hydrocarbons (they do not contain detectable mixed-function oxidase), tests were made in the presence and absence of golden hamster cells, which are capable of metabolizing polycyclic hydrocarbons. None of the compounds were mutagenic for V79 cells alone at concentrations up to about 3 μM. In the presence of golden hamster cells, however, BP and the three trans-7,8-diols were cytotoxic and mutagenic, and the mutagenic response increased as a function of hydrocarbon concentration. At an equal concentration of 0.4 μM, the (-)trans-7,8-diol was about 65% more active than the (±)trans-7,8-diol, about 6 times more active than the (+)trans-7,8-diol, and 10 times more active as a mutagen than BP. All other derivatives were inactive. (39 refs)

78-3224 **DNA and RNA Adducts Formed in Hamster Embryo Cell Cultures Exposed to Benzo(a)pyrene.** (Eng) Ivanovic, V. (Inst. Cancer Res., Coll. Physicians and Surgeons, Columbia Univ., New York, NY, 10032); Geacintov, N. E.; Yamasaki, H.; Weinstein, I. B. *Biochemistry* 17(9): 1597-1603; 1978.

The time course of the covalent binding of ^{14}C-benzo(a)pyrene (BP) to RNA and DNA in hamster embryo cell cultures and the types of nucleoside adducts were investigated. Binding to RNA proceeded rapidly, and it followed a linear time course for at least 48 hr. In contrast, the extent of binding to DNA reached a plateau after approx 18 hr of incubation with ^{14}C-BP. Fluorescence spectroscopy of the modified nucleic acids and high-pressure liquid chromatography of the nucleoside adducts provided evidence that benzo(a)pyrene-7,8-dihydrodiol 9,10-oxide (BPDE) is the major BP metabolite responsible for the covalent modification of hamster embryo cellular nucleic acids. Both BPDE I and II isomers were synthesized and both participated in the modification of the host RNA and DNA. Although guanine was the major BPDE target during in vivo modification of RNA and DNA, there was a low but detectable modification of adenine and cytosine bases. The relative contributions of the various BPDE isomers to in vivo nucleic acid modification varied between DNA and RNA. The types of RNA adducts did not depend on the length of exposure to BP. In contrast, the relative amounts of the multiple types of DNA adducts changed with time, suggesting that they underwent differential rates of excision. The heterogeneity of the DNA adducts in primary hamster embryo cell cultures was greater than that observed in previous studies with human and bovine bronchial explants exposed to BP. Possible differences in DNA-BPDE adducts among species and cell types are discussed. (29 refs)

78-3225 **Postreplication Repair of DNA in Human Fibroblasts after UV Irradiation or Treatment with Metabolites of Benzo(a)pyrene.** (Eng) Waters, R. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Yagi, H.; Jerina, D. M.; Regan, J. D. *Chem Biol Interact* 20(3): 289-297; 1978.

The ability of normal fibroblasts and of excision-deficient xeroderma pigmentosum (XP) and XP variant fibroblasts to perform postreplication DNA repair after increasing doses of either UV radiation or mutagenic benzo(a)pyrene (BP) derivatives was examined. The cells were exposed to UV at doses of 10, 20, and 30 joules/m^2 and to BP 7,8-diol-9,10-epoxide derivatives at the following doses: 5 x 10^{-7} and 10^{-6} M of the trans-diastereomer and 2 x 10^{-6} M of the *cis*-diastereomer. XP cells defective in the excision of both UV-induced pyrimidine dimers and guanine adducts induced by treatment with the BP 7,8-diol-9,10-epoxides were partially defective in their ability to synthesize high-mol wt DNA after the induction of both classes of DNA lesions. The defect was more marked in XP variant cells, despite their ability to remove by excision repair both pyrimidine dimers and the diol epoxide-induced lesions to the same extent as normal cells. Similar amounts of BP 9,10-oxide had no effect in any of the three cell lines. The response of the excision and postreplication DNA repair mechanisms operating in human fibroblasts treated with BP 7,8-diol-9,10-epoxides, therefore, appears to resemble closely that seen after the induction of pyrimidine dimers by UV irradiation. (21 refs)

78-3226 **Repair of DNA Damaged by Mutagenic Metabolites of Benzo(a)pyrene in Human Cells.** (Eng) Regan, J. D. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Francis, A. A.; Dunn, W. C.; Hernandez, O.; Yagi, H.; Jerina, D. M. *Chem Biol Interact* 20(3): 279-287; 1978.

The repair of human skin fibroblast DNA after damage by known and potential benzo(a)pyrene (BP) metabolites was examined in the bromodeoxyuridine photolysis assay. The type of repair was classified as long (UV damage) or short (ionizing radiation damage) using normal cells and cells deficient in UV repair endonuclease from a xeroderma pigmentosum patient. Only (±)-7β,8α-dihydroxy-9β,10β-epoxy-7,8,9,10-tetrahydro-BP (BP diol epoxide 1) and its diastereomer (±) -7β,8α- dihydroxy -9α,10α- epoxy-7,8,9,10-tetrahydro-BP (BP diol epoxide 2) elicited DNA damage recognizable by the UV excision repair system in normal human cells. BP 4,5-, 9,10-, and 11,12-oxides did not elicit damage repairable by this system. Naphthalene 1,2-diol-3,4-epoxides had no measurable activity in the assay. These results indicate that both the BP ring structure and the diol epoxide groups are important in causing the DNA damage that is repairable by the long excision repair system. These results parallel the reported high mutagenic activity of these compounds and thus support the concept that the BP 7,8-diol-9,10-epoxides may be the ultimate, metabolically activated forms of BP. (29 refs)

78-3227 **Human Bronchus-mediated Mutagenesis of Mammalian Cells by Carcinogenic Polynuclear Aromatic Hydrocarbons.** (Eng) Hsu, I. C. (Bethesda, MD, 20014); Stoner, G. D.; Autrup, H.; Trump, B. F.; Selkirk, J. K.; Harris, C. C. *Proc Natl Acad Sci USA* 75(4): 2003-2007; 1978.

Cultured human bronchial explants from 13 subjects activated benzo(a)pyrene (BP) to electrophilic metabolites that bound to DNA in bronchial epithelial cells. Promutagenic and mutagenic BP metabolites were also released into the culture medium. An increase in mutation frequency for ouabain resistance was found in Chinese hamster V-79 cells cocultivated with the bronchial explants in the presence of BP. The proximate carcinogenic form of BP, the 7,8 diol [(±)-r7,t8-dihydroxy-7,8-dihydrobenzo(a)pyrene] was five times more potent as a promutagen than th parent compound. Neither BP nor the 7,8-diol increase the mutation frequency of V-79 cells cultured withou bronchial explants. The mutation frequency was directl related to the binding levels of BP to bronchial DNA and the concentration of BP or the 7,8-diol in th medium. (28 refs)

78-3228 **Cyclopenta(c,d)pyrene: A Highly Mutagenic Polycyclic Aromatic Hydrocarbon.** (Eng) Eisenstadt, E. (Dept. Microbiology, Harvard Sch. Public Health, Boston, MA, 02115); Gold, A. *Proc Natl Acad Sci USA* 75(4): 1667-1669; 1978.

Cyclopenta(c,d)pyrene (CPP), recently identified as a component of carbon black, automobile exhaust, and atmospheric soot, was tested for mutagenicity with histidine auxotrophs of *Salmonella typhimurium*. In the presence of a rat liver microsome activation system, CPP was highly mutagenic for *S. typhimurium* strains TA98, TA100, TA1537, and TA1538. The CPP mutagenicity levels were comparable to those of benzo(a)pyrene and dibenz(a,c)anthracene. The potent mutagenicity of CPP is noteworthy because it is a polycyclic aromatic hydrocarbon without a bay region and it is distributed widely in the environment. On the basis of experimental observations and perturbational molecular orbital calculations, it is proposed that the mutagenic metabolite of CPP is the 3,4-oxide. The carbonium ion derived from the opening of CPP 3,4-oxide is identical to that derived from the opening of benzo(a)pyrene 7,8-diol-9,10-oxide, the metabolite now thought to be an ultimate mutagenic and carcinogenic species. (24 refs)

78-3229 **Prevention of Carcinogenesis by Retinoids.** (Eng) Sporn, M. B. (NCI, Bethesda, MD). *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 31 pp.; 1978. (no refs)

78-3230 **Induction of Epidermal Cell Proliferation by a Tumour Promoter in Vitamin B$_6$-deficient**

Mice. (Eng) Murray, A. W. (Sch. Biological Sciences, Flinders Univ. S. Australia, Bedford Park, S. Australia 5042, Australia). *Experientia* 34(6): 691; 1978.

The effect of decreased induction of ornithine decarboxylase, caused by a vitamin B_6-deficient diet, on the ability of 12-O-tetradecanoylphorbol-13-acetate (TPA) to enhance the proliferative activity of mouse epidermal cells was studied. Female Swiss albino mice were maintained for 14 days on a complete or a vitamin B-deficient diet. Animals were treated with TPA (17 nanomoles in 0.2 ml acetone) and, at varying times after treatment, with 0.1 mg colchicine ip 2 hr before sacrifice. TPA induced a marked proliferative response in the epidermis of mice maintained on both the complete and vitamin B_6-deficient diets. The mitotic indices for both groups did not differ significantly. These data do not support a causal relationship between ornithine decarboxylase and induction of proliferative activity in mouse epidermis. (10 refs)

78-3231 **Metabolic Conversion of 12-O-Tetradecanoylphorbol-13-acetate in Adult and Newborn Mouse Skin and Mouse Liver Microsomes.** (Eng) Berry, D. L. (Biology Div., Oak Ridge Natl. Lab., P.O. Box Y, Oak Ridge, TN, 37830); Bracken, W. M.; Fischer, S. M.; Viaje, A.; Slaga, T. J. *Cancer Res* 38(8): 2301-2306; 1978.

Tritiated 12-O-tetradecanoylphorbol-13-acetate was applied to adult female CD-1 mouse skin. At specified time intervals, the mice were killed, and the labeled phorbol was extracted and subjected to separation and quantitation by high-pressure liquid chromatography. After 24 hr, TPA comprised $> 96\%$ of the recovered label from the skin, and its apparent half-life was 17.8 hr. Pretreatment of adult skin with TPA (10 μg/wk for 4 wk) before treatment with ^{3}H-TPA increased the clearance rate of TPA from the skin. Skin from newborn BALB/c mice was capable of converting TPA into monoesters and phorbol, but the clearance rate in the adult was about 12 times more rapid than that in the newborn. Epidermal homogenates converted TPA 12-O-tetradecanoylphorbol (TP), phorbol-13-acetate, and phorbol. Hepatic homogenates converted TPA to monoesters and phorbol at rates 14 to 15 times faster than those of the epidermal homogenates. Attempts to isolate any previously undescribed TPA metabolites by liver homogenates were unsuccessful, and mixed-function oxidation did not contribute to TPA metabolism. The conversion of TPA to monoesters and phorbol appeared to be related to esterase activity, since α-naphthol acetate inhibited TPA conversion to TP. (36 refs)

78-3232 **Progesterone Receptor Levels in Estrogen-induced Renal Carcinomas after Serial Passage Beneath the Renal Capsule of Syrian Hamsters.** (Eng) Lin, Y. C. (Dept. Biological Chemistry, Harvard Medical Sch., Boston, MA, 02115); Talley, D. J.; Villee, C. A. *Cancer Res* 38(5): 1286-1290; 1978.

Diethylstilbestrol-induced renal tumors in castrated male Syrian hamsters were serially transplanted and examined to determine whether progesterone receptor concentrations change as a function of serial passage. Fragments of primary tumor transplanted subpannicularly in estrogen-treated hamsters increased $< 10\%$ in mass during a 2-mo period; however, transplantation of primary tumor tissue beneath the renal capsule resulted in a 20- to 30-fold increase in tumor mass after 2 mo. There was no statistically significant difference in tumor concentration of progesterone receptor after the first or second serial passage relative to the concentration found in primary tumor tissue. Although the concentration of progesterone receptor was not significantly decreased after the second serial passage, there was a slower growth rate of tumors during this period. In addition to the reduced growth rate, a lower concentration of progesterone receptor was found in untransformed kidney areas of hosts bearing second-serial-passage tumors relative to those with first-passage or primary tumors. The serum concentration of diethylstilbestrol in tumor-bearing hosts was 2 to 4 x 10^{-8} M, and apparently it was not a factor in the change in growth rate of the tumors. It is suggested that the slower growth rate of the tumors after the second serial passage is related to an invasive property or surface change in the tumor cells rather than to significant changes in hormone receptor site concentrations within the cells. (21 refs)

78-3233 **Endogenous Levels of Eight Steroids in the Human Prostate From Birth to Old Age (Meeting Abstract).** (Eng) Hammond, G. L. (Dept. Clinical Chemistry, Univ. Oulu, SF-90220 Oulu 22, Finland). *J Endocrinol* 75(3): 27P-28P; 1977. (1 ref)

78-3234 **Hepatocellular Carcinoma Possibly Induced by Oral Contraceptives.** (Eng) Ham, J. M. (Gastrointestinal Surgical Unit, Prince Wales Hosp., Randwick, North South Wales 2031, Australia); Stevenson, D.; Liddelow, A. G. *Am J Dig Dis* 23(5, Suppl): 38s-40s; 1978.

A 30-yr-old woman developed hepatocellular carcinoma and widespread metastases after 5 yr use of the oral contraceptive (OC) Ovulen (ethynodiol diacetate, 1 mg; mestranol, 0.1 mg). She developed pyrexia of unknown origin upon cessation of OC usage; liver resection was performed 2 yr later. This is the first reported case of widespread metastases occurring with a malignant liver tumor in association with OC use. (11 refs)

78-3235 **Diethylstilbestrol Metabolites and Analogs: In Vivo and In Vitro Estrogenic Activities (Meeting Abstract).** (Eng) Korach, K. S. (Lab. Environmental Toxicology, NIEHS, Research Triangle Park, NC, 27709);

Metzler, M.; McLachlan, J. A. *Environ Health Perspect* 20: 240; 1977. (no refs)

78-3236 **Studies of Estrogen Induced Renal Tumours in Male Syrian Hamsters. I. Cytochemical Studies of Lysosomal Enzymes Acid Phosphatase and β-Glucuronidase.** (Eng) Ismail, A. A. (Tumour Cell Biology Dept., Medical Res. Inst., Alexandria Univ., Alexandria, Egypt); Hamilton, J. M.; Amin, A. M. *Acta Histochem (Jena)* 61(2): 287-295; 1978.

Cytoenzymological changes occurring during diethylstilbestrol (DES)-induced renal tumorigenesis were determined in male Syrian hamsters. The hamsters were inoculated sc with 0.6 mg DES 3x/wk. After 175 days, slightly elevated, white, solid subcapsular nodes were seen. After 225 days, tumor formation was obvious. In normal kidney cells, azo-coupling techniques showed that acid phosphatase (AP) and β-glucuronidase (GU) granules were distributed throughout the cytoplasm, but not in the nuclei. The degree of staining was moderate. During DES treatment, the distribution pattern of the two enzymes did not change. However, there was a gradual increase in AP and GU activity starting at 12 wk (21.6 mg DES) to a max at 30 wk (56 mg DES), when tumors were observed. The staining was intense. The increased enzyme activity could be due to a higher rate of lysosomal enzyme synthesis in the tumor cells or to invasion of tumor tissue by hydrolase-rich macrophages. (15 refs)

78-3237 **Developmental Abnormalities in the Mouse Genital System Associated with Prenatal Exposure to Diethylstilbestrol (Meeting Abstract).** (Eng) McLachlan, J. A. (Natl. Inst. Environmental Health Sciences, Res. Triangle Park, NC, 27709). *In Vitro* 14(4): 333; 1978. (no refs)

78-3238 **Metabolism of Diethylstilbestrol in the C3H Mouse: Chromatographic Systems for the Quantitative Analysis of DES Metabolic Products.** (Eng) Helton, E. D. (Food and Drug Admin., Natl. Center Toxicological Res., Jefferson, AR); Gough, B. J.; King, J. W.; Thenot, J. P.; Horning, E. C. *Steroids* 31(4): 471-484; 1978.

Chromatographic systems were used to study the metabolism of diethylstilbestrol (DES) in female C3H mice. Preliminary excretion studies with po monoethyl-1-^{3}H-DES indicated that excretion via the feces was the principal mode of DES elimination. Metabolic studies with po ^{3}H-DES and/or ^{14}C-DES were performed in mice with a high (+) or a low (-) titer of mouse mammary tumor virus (MMTV). Extraction and partitioning of the fecal radioactivity indicated that 77%-86% was benzene-soluble, the remainder water-soluble. The principal product in the organic phase following purification was DES. Analysis of the aqueous phase following 5β-glucuronidase digestion demonstrated that the principal aglycone was chromatographically identical to DES. The urinary conjugates were resolved into six fractions. The four major fractions were 80% hydrolyzable with β-glucuronidase. Two of these fractions had *trans*-DES as the principal aglycone, but the other two had a major peak similar to, but not chromatographically coincident with *cis*-DES. In additional experiments, MMTV + and MMTV- mice were dosed with ^{3}H-DES, the urine and feces were collected for 24 hr, they were then dosed with ^{14}C-DES, and again urine and feces were collected for 24 hr. Two MMTV+ mice were also previously fed a diet containing 1,000 ppb DES for 120 days. The labeled excretion products were combined and analyzed simultaneously. There were no significant differences in metabolism due to the modes of radioactive labeling, MMTV titer, or prior feeding of DES. (32 refs)

See also:

*(Rev.): 78-3001, 78-3002, 78-3003, 78-3004, 78-3005, 78-3007, 78-3008, 78-3009, 78-3010, 78-3011, 78-3012, 78-3013, 78-3014, 78-3015, 78-3016, 78-3017, 78-3018, 78-3019, 78-3020, 78-3021, 78-3022, 78-3023, 78-3024, 78-3026, 78-3027, 78-3028, 78-3031, 78-3034, 78-3049, 78-3050, 78-3054.

*(Phys.): 78-3242.

*(Immune.): 78-3370, 78-3371, 78-3394, 78-3405, 78-3435, 78-3437, 78-3445, 78-3449, 78-3452.

*(Path.): 78-3468, 78-3470, 78-3471, 78-3472, 78-3478, 78-3493, 78-3520, 78-3521, 78-3523, 78-3524, 78-3543, 78-3545, 78-3547, 78-3549, 78-3552, 78-3555.

*(Epid.-Biom.): 78-3580, 78-3581.

78-3239 **Stochastic Late Effects after Partial Body Irradiation in Diagnostic Radiology: Evaluation of Approximate Data.** (Eng) Pauly, H. (Institut fur Radiologie der Universitat Erlangen-Nurnberg, Krankenhausstrasse 12, D-8520 Erlangen, W. Germany). *Radiat Environ Biophys* 15(1): 21-33; 1978.

A method is described for estimating the risk of a patient developing cancer after partial-body, diagnostic x-ray exposure. The cumulative possibility, *p*, of a radiation-induced neoplasm is obtained from the equation $p = Gt\ Es$, where *Es* is the energy imparted to the soft tissues of the trunk and head. *Gt* is the mean integral incidence function for the trunk and head, reflecting the cancer inducibility of organs and tissues at these sites. The value of *Gt* [0.3 kilojoule^{-1} (kJ^{-1})] was obtained from mortality risk factors adopted by the International Commission on Radiological Protection. The *Es* imparted to the body in typical radiographic procedures ranges from 1 to 30 mJ, but it can reach 1 J in an extensive fluoroscopic examination of the gastrointestinal tract. The corresponding values for *p* are 10^{-6} to 10^{-5} in typical examinations, 10^{-4} in extensive examinations. (14 refs)

78-3240 **Preliminary Report on Radiation and Heart Disease.** (Eng) Bross, I. D. (Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263); Ball, M.; Rzepka, T.; Laws, R. E. *J Med* 9(1): 3-15; 1978.

The hypothesis that low levels of diagnostic radiation can produce both leukemia and heart disease in men was tested using data from the Tri-State Survey (New York, Maryland, and Minnesota) and a mathematical model. The radiation range considered was within a factor of 10 of 5 rads (0.5-50 rads). The results indicate that even at the lowest levels, an estimated 5% of exposed men will suffer DNA damage that will result in heart disease or leukemia. For the minority of individuals affected by the radiation, there is a tenfold increase in the risk of leukemia and a threefold increase in the risk of heart disease. (11 refs)

78-3241 **Hashimoto's Thyroiditis: The Commonest Radiation-induced Thyroid Disease (Meeting Abstract).** (Eng) Okerlund, M. D. (Nuclear Medicine Section, Univ. California Medical Center, San Francisco, CA); Beckmann, A.; Caro, M.; Sommers, J.; Kaufman, L.; Galante, M.; Hunt, T. *Clin Res* 26(2): 107A; 1978. (no refs)

78-3242 **Radiation-induced Thyroid Tumors in Infant Rats.** (Eng) Christov, K. (Inst. Oncology, Acad. Medicine, Sofia-56, Darvenitza, Plovdivsko pole 6, Bulgaria). *Radiat Res* 73(2): 330-339; 1978.

The susceptibility of the thyroid gland in infant rats to the carcinogenic effect of x-rays was examined with doses similar to those used to treat the hyperplastic thymus in human infants. Irradiation of 10-day-old Wistar rats with 300 rads of x-rays led to the development of thyroid tumors in 40% of the animals 15 mo later, whereas the same dose caused tumor growth in 15% of adult animals (60 days old). The latent period was also shorter in animals irradiated as infants. A subsequent treatment of irradiated animals with methylthiouracil significantly increased the number of thyroid adenomas and carcinomas and shortened the latent period in both infant and adult rats. The thyroid tumors examined were predominantly of a follicular type. Papillary and medullary tumor variants were observed in a few animals only. The results indicate that the thyroid gland of suckling animals is more susceptible to the tumorigenic stimulus of x-radiation than that of adult animals. (24 refs)

78-3243 **Carcinogenic Effect of Isolated and Combined Chronic External (Gamma) and Internal (^{131}I) Radiation on Rats.** (Rus) Burykina, L. N. (Inst. Occupational Hygiene and Vocational Diseases, Moscow, USSR); Vasil'eva, L. A.; Likhachev, Iu. P.; Ponomareva, V. L.; Veselovskaia, K. A. *Med Radiol (Mosk)* (4): 35-40; 1978.

The long-term effects of chronic exposure to ^{131}I combined with prolonged whole body radiation were studied in random-bred albino rats. The animals were divided into five groups: Groups 1 and 2 received ^{131}I (1-2 x 10^{-4} and 0.25 x 10^{-4} μCi/g/day, respectively, daily, for 6 mo; total absorbed dose, 1,900-3,000 and 700 rads, respectively); Group 3 received whole-body ^{137}Cs γ radiation (0.4-0.8 R/day, 5x/wk, total dose, 50-100 rads); Group 4 received ^{131}I + γ radiation (total absorbed dose 1,600-2,600 rads); Group 5 served as the control group. The av duration of survival in the groups was 606, 623, 617, 611, and 645 days, respectively. Tumors were observed in 67.2% of rats in Group 1 (44.4% had thyroid tumors), 57.5% in Group 2 (14.8%), 54.2% in Group 3 (6.8%), 66.0% in Group 4 (40.9%), and 33.8% in Group 5 (7.9%). The structural changes in the thyroid tissue were more pronounced in Group 4 rats (compared with rats in Groups 1 and 2). (8 refs)

78-3244 **Long-Term Results of Administration of Uranium to Rats.** (Rus) Filippova, L. G. (No affiliation given); Nifatov, A. P.; Liubchinskii, E. R. *Radiobiologiia* 18(3): 400-405; 1978.

The long-term results of intratracheal administration of various doses of uranium (U) were studied in Wistar rats. Animals received a single dose of the nitrate salt of U(IV) or U(VI). Exposure to 19.5 mg/kg U(VI) resulted in 100% mortality within 30 min; exposure to 9.76 mg/kg and 5.82 mg/kg U(VI) resulted in 100% and 42.6% mortality, respectively, within 2 wk. Administration of 18.7 mg/kg U(IV) resulted in 31% mortality within the first wk of exposure (the life-span of the survivors was decreased by approx 100 days). From 72.7% to 79.3% of the animals exposed to U(IV) and U(VI) at 5.27-5.82 mg/kg died of nephrosclerosis. Osteogenic sarcomas were detected in 2.9% and 5.5% of the rats exposed to 1.57 and 5.82 mg/kg U(VI) and in 2.0% and 4.5% of the rats exposed to 2.85 and 5.27 mg/kg U(VI), respectively. (9 refs)

78-3245 **The Effect of Chronic Exposure to ^{238}Pu(IV) Citrate on the Embryonic Development of Carp and Fathead Minnow Eggs.** (Eng) Till, J. E. (Environmental Sciences Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830). *Health Phys* 34(4): 333-343; 1978.

The effects of plutonium (Pu) on developing fish eggs were studied. A quantitative analysis of ^{238}Pu(IV) citrate uptake by carp eggs indicated that Pu accumulated in the eggs and reached a concentration factor of approx 4 at hatching (72 hr after exposure began). Autoradiographs revealed that although some Pu was concentrated on the egg chorion, the Pu that penetrated the chorion was uniformly distributed throughout the perivitelline fluid, embryo, and yolk sac. Using the uptake data for carp eggs, dose conversion factors were calculated for both carp eggs and fathead minnow eggs exposed to ^{238}Pu during embryological development. These values were 2,100 and 7,500 rads/μCi/ml for carp and fathead minnows, respectively. Experiments were conducted in which both carp and fathead minnow eggs were exposed to Pu in soln during embryogenesis; the percentage of eggs hatching, the number of abnormal larvae produced, and the survival of larvae were the indicators of radiation toxicity. Concentrations >1 μCi/ml were required to prevent both species of eggs from hatching. It is concluded that fish eggs developing in natural aquatic ecosystems contaminated with Pu probably do not receive a significant dose from the Pu alpha radioactivity. Future analysis of fecundity and the production of abnormalities in the offspring of larvae exposed during embryological development would provide an additional degree of sensitivity to the toxicity test system used here. (19 refs)

78-3246 **Distribution and Kinetics of Metabolism of ^{238}Pu after Intratracheal Administration to Rats.** (Rus) Nisimov, P. G. (No affiliation given). *Radiobiologiia* 18(3): 406-411; 1978.

The distribution and kinetics of metabolism of intratracheally administered ^{238}Pu were studied in random-bred rats. The animals received a soln of ^{238}Pu nitrate (2.5 μCi/kg), and the animals were sacrificed 0.5 hr to 226 days later. Tissue residues of the radionuclide were determined histoautoradiographically. Almost 24% of the radioactivity was eliminated from the lungs within 30 min of administration, 43.7% over 42 hr, 30.4% over 10 days, and 1.84% over 226 days. The max concentration of ^{238}Pu in the liver (8.26%) was detected 24 hr after administration (48.45% of liver radioactivity was eliminated within 7 days, and 51.55% was eliminated within 30 days). The accumulation of radioactivity in the bone tissue increased gradually from 4.11% on day 1 after administration to 30.31% on day 3, 49.31% on day 16, and 53.39% on day 32. (5 refs)

78-3247 **Distribution of Tritium in the Different Organs of Calves and Pigs After Ingestion of Various Tritiated Feeds.** (Eng) Kirchmann, R. (Dept. Radiobiology, C.E.N./S.C.K., B-2400 Mol, Belgium); Charles, P.; van Bruwaene, R.; Remy, J.; Koch, G.; van den Hoek, J. *Curr Top Radiat Res* 12(1): 291-312; 1977.

The distribution of tritium (^{3}H) administered as tritiated water (HTO) or incorporated in feed was studied in male calves and pigs. Three calves received fresh milk supplemented with 50 μCi/ml HTO (total ^{3}H activity ingested over a 28-day period: 25.6-40.4 μCi/animal). Two other calves received 125 g ^{3}H-powdered milk (total ingested activity: 482 μCi/calf). Pigs in groups of two or three animals were treated as follows: (1) a single ip injection of 39.3 μCi HTO; (2) 28.4 μCi/day HTO (total ingested activity: 568 and 766.8 μCi/animal); (3) ^{3}H-potatoes (total ingested activity: 21-48.1 μCi/animal); and (4) ^{3}H-powdered milk (total ingested activity: 60.6-154 μCi/animal). After the animals were slaughtered, ^{3}H concentrations were determined in tissue water and organic matter. Ingestion of ^{3}H powdered milk, compared with HTO, increased the total incorporation in organic matter by a factor of 5.6 in the pigs and a factor of 15 in the calves. In pigs fed the ^{3}H potatoes, there was a 15.6-fold increase in incorporation. For both species, the specific activity in the tissue water f the organs was a little lower than that of ingested HTO. 'he activity was very low after the ingestion of ^{3}H-milk, and here were no species differences. Radioactivity was found in ll the nucleic acids, lipids, and proteins isolated from pig ver and spleen. However, there was no evidence that ^{3}H was ctually incorporated into the DNA molecules of the liver an organ consisting of nondividing cells) or the spleen (an rgan containing actively dividing cells). (10 refs)

8-3248 **Retention and Distribution of ^{241}AmIII in Neonatal Beagles.** (Eng) Stevens, W. (Radiobiology Div., Dept. Anatomy, Univ. Utah, Salt Lake City, UT, 4132); Atherton, D. R.; Bates, D.; Lloyd, R. D.; Buster, D. .; Bruenger, F. W. *Health Phys* 33(6): 553-559; 1977.

The distribution of americium in beagles was studied during heir first week of life. Seven litter mates (4 males, 3 female 250-280 g) were inoculated iv with 3.0 μCi ^{241}AmIII/kg when 1 day old and killed 1, 3, or 5 days later. Cumulative excretion during the time observed was 6.7% of the dose, 5.5% of which occurred during the first day. Mean recovery of the injected Am was 96%. The deposition of Am in the skeleton and soft tissues was compared to data obtained from three beagles inoculated with an equivalent dose of Am when 18 mo old and sacrificed 1, 7, and 8 days later. Skeletal retention was 76% of the injected dose at 1 day and 84% at 5 days in the neonates. The skeletons of the 18-mo-old beagles contained 29% of the dose at 1 wk. The Am content of the bones in the neonatal skull was three times that seen in dogs inoculated as young adults. Initial liver deposition of Am in neonates was 7%, compared to 50% in young adult dogs. The subcellular distribution of Am in liver also differed in the two groups: in neonates, a greater fraction of the nuclide was associated with soluble cytosol proteins, and the Am content of mitochondria and lysosomes increased at a much slower rate than that observed in mature animals. It is speculated that the relatively low retention in the soft tissue organs of the neonate should make them less susceptible to radiation damage. However, the mineralized tissue, which retains 80% of the injected dose, is in a state of rapid growth and reorganization, and an increased incidence of early bone tumors may occur. (13 refs)

78-3249 **Postprandial Changes in the Exhalation Rate of Radon Produced In Vivo.** (Eng) Rundo, J. (Center for Human Radiobiology, Argonne Natl. Lab., Argonne, IL, 60439); Markun, F.; Sha, J. Y. *Science* 199(4334): 1211-1212; 1978.

Changes in the rate of exhalation of radon following a meal were investigated in several radium dial painters first exposed to radium between 1916 and 1925 and in one man involved in a radium spill in 1952. There was a radon exhalation rate at about 0.5 hr after a meal, followed by a decrease to normal values by 1.5-2 hr. There was also a strong correlation between the exhalation rate of radon and the pulse rate. This suggests rate of range in radon exhalation observed postprandially is related to the change in blood flow associated with the digestive processes. This could represent the flushing of a radon reservoir in the viscera. (7 refs)

78-3250 **Natural and Manmade Radiation Exposure.** (Ger) Mehl, J. (Bundesministerium des Innern, Postfach, D-5300 Bonn 7, W. Germany). *Kertechnik* 20(5): 221-228; 1978.

Annual dose-equivalent values for various organs and tissues and annual effective whole-body dose-equivalent values resulting from exposure to natural and manmade radiation were assessed. The prospective incidence rates of radiation-induced diseases, including cancer and leukemia, in West Germany were calculated by assuming a linear dose-effect relationship and by using risk factors published by the International Commission on Radiological Protection. The incidence rates of radiation-induced cancers and leukemias are compared with the spontaneous incidence rates for 1974. The data should be useful in the development of a program to reduce the deleterious effects of ionizing radiation. (21 refs)

78-3251 **Sister-Chromatid Exchange Induced by X-Ray of Human Lymphocytes and the Effect of L-Cysteine.** (Eng) Abramovsky, I. (Div. Medical Genetics, Dept. Pediatrics, Mount Sinai Sch. Medicine, City Univ. New York, Fifth Ave. and 100th St., New York, NY, 10029); Vorsanger, G.; Hirschhorn, K. *Mutat Res* 50(1): 93-100; 1978.

The characteristics of sister-chromatid exchanges (SCE's) detectable in normal human lymphocytes exposed to different dosages of x-radiation were studied. All irradiations (25 to 200 rads) were performed on cells that had completed at least one round of replication in 5-bromo deoxyuridine (BUdR). A significant dose-dependent increase in SCE's per cell occurred when the lymphocytes were irradiated in the G_1 phase of their second mitotic division. A slight increase was noted after irradiation of lymphocytes in the G_2 phase. Cellular viability was reduced markedly in the G_1 lymphocytes at 200 rads and in the G_2 lymphocytes at 50-100 rads. The increase in SCE frequency was accompanied by an increase in chromosome aberrations: terminal deletions, interstitial deletions, and chromosome exchange figures in G_1 cells; chromatid gaps, breaks, and exchanges in G_2 cells. The antimutagen L-cysteine decreased the frequency of SCE's in irradiated G_1 cells, although the frequency was still significantly higher than in nonirradiated cells. L-cysteine increased cell viability, especially after radiation doses of 100 rads or more. However, the addition of L-cysteine to lymphocytes that had been exposed to x-radiation did not reduce SCE frequency or cell death significantly. An analysis of SCE's in human cells may provide a sensitive and reliable method for screening potential chromosome mutagens. (23 refs)

78-3252 **Spontaneous and Radiation-induced Oncogenesis in Hamsters.** (Eng) Trentin, J. J. (Div. Experimental Biology, Baylor Coll. Medicine, Houston, TX, 77030). *Fed Proc* 37(7): 2084-2086; 1978.

An attempt was made to increase the incidence of lymphomas in weanling Syrian hamsters by fractionated radiation. The animals received 240 R of whole-body x-radiation once a week for 4 wk. Of 22 hamsters surviving >60 days, only 1 developed a lymphoma weekly. Doses of 20, 40, 80 or 150 R were then given for 10, 20, or 30 wk. Of 185 animals that survived the treatment, 13 had tumors, mostly of the liver and spleen, at autopsy. These tumors were diagnosed as reticulum cell sarcomas, small cell sarcomas, or undifferentiated tumors of the liver and spleen. The highest tumor incidence was only 12%-13% in the four highest total exposure groups (1,500, 1,600, 2,400, and 3,000 R). These findings indicate that, compared with the mouse, the hamster is remarkably more resistant to irradiation lymphomagenesis. The nature of this resistance is not known. (16 refs)

78-3253 **Comparison of Survival and Unscheduled DNA Synthesis Between an Insect and a Mammalian Cell Line Following X-Ray Treatments.** (Eng) Koval, T. M. (Cancer Res. Unit, Clinical Radiation Therapy Res. Center, Allegheny General Hosp., Pittsburgh, PA, 15212); Myser, W. C.; Hart, R. W.; Hink, W. F. *Mutat Res* 49(3): 431-435; 1978.

Survival and unscheduled DNA synthesis following x-irradiation were compared in insect cells from the *Trichoplusia ni* (TN-368) cabbage looper cell line and V-79 Chinese hamster cells. The TN-368 cells were x-irradiated with 0.5 to 10 kR, and the V-79 cells with 0.05-1 kR, and incubated for 7 days, and plating efficiencies were counted. The TN-368 cells were >30 times more resistant to x-rays than the V-79 cells. In the unscheduled DNA synthesis study, the TN-368 cells incorporated >10 times more ^{3}H thymidine via unscheduled DNA synthesis than the V-79 cells after each of the three x-ray exposures (0.5, 1.0, and 1.5 kR) investigated. Both the rate and extent of unscheduled DNA synthesis were much greater in the TN-368 cells. The extent of unscheduled DNA synthesis is correlated with the excision repair of DNA base damage, since single-strand break repair was essentially complete by 30 min and unscheduled DNA synthesis continued for at least 4 hr. The high yield of unscheduled DNA synthesis in TN-368 cells after x-irradiation is due to their havi a larger av size for x-ray-repaired regions or a greater numb of repaired regions per unit length of DNA. (10 refs)

78-3254 **Mammary and Other Tumors as a Response t Radiation and Multiple Stresses.** (Eng) Warre S. (194 Pilgrim Road, Boston, MA, 02215); Brown, C. *Arch Pathol Lab Med* 102(5): 224-226; 1978.

Tumor incidence was investigated in female NEDH rats tha were parabiosed at about 35 days of age. Torsion between th partners was prevented by approximating the adjacent scapulae of the partners using a suture of either braided stainles steel or tantalum. Subsequently, one partner received 1,00 R whole-body 250-kilovolt peak x-radiation while the othe partner was shielded by lead. Benign mammary tumors developed in 8.6% of the control pairs, 12.7% of the shielde partners, and 19.7% of the irradiated rats. Mammary cancer developed in 5% of the control pairs, 14.2% of the shielde partners, and 11.9% of the irradiated rats. When a hormona imbalance was imposed by unilateral nephrectomy i each rat (which increased prolactin levels), th mammary cancer rate was 50% in the shielded partn and 10% in the irradiated rats. Mesenchymal sarcom developed at the anastomosis in parabiont rats. Th incidence rate in single controls was 3.1%, in par biont control pairs 12.5%, in pairs with one partn irradiated 16.6%, and in pairs with both irradiate 23.1%. About 33% of these sarcomas occurred at the junction of the scapulae, where the metallic suture was presen as a foreign body. Tumor incidence elsewhere in irradiate partners was increased threefold over controls. It is suggeste that radiation is the dominant factor in both mammary an anastomotic tumors. (8 refs)

78-3255 **Effects of Radiation Type and Dose and th Role of Glucocorticoids, Gonadectomy, an Thyroidectomy in Mammary Tumor Induction in Mammotropin-secreting Pituitary Tumor-grafted Rats.** (Eng) Clifton, K. H. (Radiobiology Labs., 420 N. Charter St., Madison WI, 53706); Crowley, J. J. *Cancer Res* 38(6): 1507-1513 1978.

The effects of 11-100 rads' neutron or 50-500 rads' γ-ra whole-body radiation, glucocorticoids, gonadectomy, an thyroidectomy on mammary tumor induction in mammotropin-secreting pituitary tumor (MtT)-grafted Fischer an W/Fu rats were determined. These studies yielded the following results: (1) neutrons were significantly more efficient tha γ-rays in the induction of mammary carcinomas; (2 adrenalectomy with mineralocorticoid replacement potentiated mammary carcinoma induction in irradiated MtT-grafted rats; (3) potentiation of carcinoma induction b adrenalectomy was reversed by glucocorticoid treatment; an

4) gonadectomy and thyroidectomy of irradiated MtT-graft-d Fischer rats did not potentiate mammary carcinoma inluction as did glucocorticoid deficiency and, in combination vith the latter, they may reduce the adrenalectomy effect. tatistical analysis of the data by procedures that take into ccount time at risk as well as tumor frequency indicates that nultiple mammary tumors do not occur independently (ie, first mammary tumor significantly increases the probability f development of another tumor). The statistical procedure sed in this analysis is presented. (25 refs)

8-3256 **Leukemia Risk from Neutrons.** (Eng) Rossi, H. H. (Radiobiological Res. Lab., Dept. Radiology, Coll. Physicians and Surgeons, Columbia Univ., 630 W. 168th St., New York, NY, 10032); Mays, C. W. *Health Phys* 34(4): 353-360; 1978.

The leukemia incidence among atom bomb survivors was reevaluated in terms of absorbed dose to the marrow, and the results were used to estimate the risk from present permissible neutron doses. The predicted lifetime incidence of leukemia is 800 cases/1,000,000 persons exposed to a 1-rad dose to the marrow. For a 50-yr occupational exposure at the max permissible dose of 0.5 rad/yr to marrow and total body, the leukemia incidence will be 2%, a value that is five times higher than the naturally occurring lifetime incidence of 0.4% in the US. Because these risks are unacceptably high, current permissible neutron doses should be reduced by at least one order of magnitude. (15 refs)

78-3257 **The Effect of 8-Methoxypsoralen Plus UV-Light on the Frequency of Viral Transformation by Polyoma Virus In Vitro (Meeting Abstract).** (Eng) Morhenn, V. B. (Dept. Dermatology, Stanford Univ. Sch. Medicine, Stanford, CA); Kaye, J. A. *Clin Res* 26(3): 573A; 1978. (no refs)

78-3258 **Ultraviolet-Light-induced Unscheduled DNA Synthesis by Resting and Growing Mouse Oocytes.** (Eng) Pedersen, R. A. (Lab. Radiobiology, Univ. California, San Francisco, CA, 94143); Mangia, F. *Mutat Res* 49(3): 425-429; 1978.

Resting (from 2- to 3-day old mice) and growing (from 12- to 13-day old mice) mouse oocytes were exposed to UV light (0, 15, 30, or 60 joules/m^2), cultured in medium containing ^{3}H-thymidine, and examined for unscheduled DNA synthesis by autoradiography as a measure of DNA excision repair. Both resting and growing oocytes incorporated ^{3}H-thymidine into their nuclei in significantly greater amounts than control (unirradiated) oocytes. Growing oocytes, however, incorporated about six times more ^{3}H-thymidine than resting oocytes. There are several possible explanations for this difference: the rates of thymidine uptake and its endogenous pool size may differ between stages; the actual amount of damage to the DNA of resting and growing oocytes may be different; and growing oocytes may actually have higher activities of excision-repair enzymes. It is concluded that both resting and growing oocytes are capable of unscheduled DNA synthesis after UV irradiation. (19 refs)

78-3259 **Neoplastic Transformation Induced by a Direct Perturbation of DNA.** (Eng) Barrett, J. C. (Natl. Inst. Environmental Health Sciences, NIH, P.O. Box 12233, Research Triangle Park, NC, 27709); Tsutsui, T.; Ts'o, P. O. *Nature* 274(5668): 229-232; 1978.

The effects of 5-bromodeoxyuridine (BUdR) and/or near-UV radiation on Syrian hamster embryo (SHE) cells were investigated. BUdR alone (10^{-6} M for 24 hr) was cytotoxic to early-passage SHE cells, and exogenous deoxycytidine reduced this effect. Irradiation with near-UV light (6 fluorescent bulbs at a distance of 7.5 cm; approx 330 ergs/mm^2/sec) for 1-5 min after BUdR treatment significantly enhanced the lethality of the latter and resulted in single-strand breaks in cellular DNA. BUdR followed by near UV irradiation, but neither agent alone, caused somatic mutations in the hypoxanthine phosphoribosyltransferase and (Na^+, K^+) ATPase loci. BUdR (2.5 x 10^{-6} M for 24 hr) plus irradiation for 1 or 3 min also caused morphological transformation and neoplastic transformation in SHE cells. Cells from transformed cultures exhibited colony formation in soft agar and formed tumors after injection into newborn hamsters. Cytotoxicity, single-strand breakage, mutation frequency, and morphological transformation increased with increasing dosage of irradiation, and cytotoxicity and morphological transformation increased with BUdR dose. The data suggest that a direct perturbation of cellular DNA is sufficient to initiate neoplastic transformation. (22 refs)

78-3260 **Effects of Ultraviolet Light on Nude Mice. Cutaneous Carcinogenesis and Possible Leukemogenesis.** (Eng) Eaton, G. J. (Inst. Cancer Res., Fox Chase Cancer Center, 7701 Burholme Ave., Philadelphia, PA, 19111); Custer, R. P.; Crane, A. R. *Cancer* 42(1): 182-188; 1978.

The effects of UV irradiation (14.8 millijoules/hr for 2 hr, 5 days/wk) on nude mice (nu/nu) of two different genetic backgrounds (BALB/c and C57BL/6), their congenic partners (+/+), and hairless (hr/hr) mice were studied. All nu/nu mice died within 117 days without developing cancers, although three did develop papillomas. Multiple squamous cell carcinomas were induced after an av of 176 (BALB/c) or 194 (C57BL/6) exposures in all seven nu/nu mice that had been reconstituted with syngeneic spleen cells prior to treatment. In two C57BL/6 mice, there were metastases to the lungs and lymph nodes. No tumors appeared in the congenic BALB/c mice after 317 exposures, and only one tumor arose in the congenic C57BL/6 mice after 244 exposures. In contrast, multiple tumors developed in all 24 hr/hr mice after 61-136 exposures, and one tumor metastasized to the lungs. All tumors were epidermal in origin, and they varied in degree of differentiation from simple hyperkeratosing papillomas to highly anaplastic types with abundant mitotic activity. Striking granulocytic proliferation was provoked in one hr/hr and in five reconstituted nu/nu mice; two of the latter appeared to have true granulocytic leukemia. The results demonstrate the susceptibility of athymic nude mice to the cutaneous carcinogenic activity of UV light, provided their life-spans are extended to permit prolongation of the irradiation. (9 refs)

78-3261 **Ultraviolet Exposure and Skin Cancer Response.** (Eng) Green, A. E. (ICAAS, Space Sciences Res. Bldg., Univ. Florida, Gainesville, FL, 32611). *Am J Epidemiol* 107(4): 277-280; 1978.

The response of human skin to exposure to UV light is considered in terms of the applicability of reciprocity to the problem. Reciprocity holds if the cumulative risk S to a homogeneous population group of age A is a function of the cumulative exposure E = DA (D is the UV dose/yr). If reciprocity were known to work even approximately, dose dependence could be inferred from age dependence. The reciprocity implications for UV exposure leading to skin cancer were calculated mathematically. The results suggest that reciprocity fails for nonmelanoma skin cancer but that it may hold for melanoma skin cancer. (16 refs)

78-3262 **Fluorescent Light-induced Chromosome Damage and Its Prevention in Mouse Cells in Culture.** (Eng) Parshad, R. (Dept. Pathology, Howard Univ. Coll. Medicine, Washington, DC, 20059); Sanford, K. K.; Jones, G. M.; Tarone, R. E. *Proc Natl Acad Sci USA* 75(4): 1830-1833; 1978.

A 20-hr exposure to fluorescent light produced chromatid breaks in adult mouse C57BL/6N lung cells grown in Dulbecco-Vogt medium supplemented with fetal bovine serum. The light-induced damage was enhanced slightly by increasing the oxygen concentration in the gas phase of the culture. The effective wavelength(s) of light was in the visible range between 400 and 450 nanometers (nm), and it was probably the mercury emission peak at 405 or 436 nm. Addition of catalase or glutathione with ascorbic acid to the culture medium reduced the number of chromatid breaks to a level not significantly different from that in the shielded cultures Thus, the production of H_2O_2 in the culture medium or in the cell was apparently responsible for the chromatid breaks Most chromosome abnormalities observed in long-term cultures of mouse cells may result from exposure of cells o medium to fluorescent room light in the presence of atmospheric oxygen. These genetic abnormalities can be minimized by shielding cells and medium from light, lowering the oxygen partial pressure of the medium, and including reducing agents such as glutathione and ascorbic acid in the medium (21 refs)

78-3263 **Photoaugmentation: A Photobiological Phenomenon.** (Ger) Spiegel, H. (Dermatologische Klinik und Poliklinik, Universitat Munchen, Frauenlobstrasse 9-11, D-8000 Munich 2, W. Germany); Plewig, G. Hofmann, C.; Braun-Falco, O. *Arch Dermatol Res* 261(2) 189-200; 1978.

The phenomenon of photoaugmentation was investigated in 37 subjects (aged 15-78 yr) of both sexes. UVA, UVB, UVC were applied with a high-intensity grating monochromator Photoaugmentation could be induced following UVA plus UVB or up to 3 hr later, but not after 24 hr UVA [351-389 nanometers (nm)] at 15 joules (J)/cm^2 and UVB (290-310 nm) at 13 mJ/cm^2 resulted in one-half the minima erythema dose. The reversed application of UVB plu UVA also led to photoaugmentation. With UVA plu UVC, photoaugmentation could not be elicited i every patient. Photoaugmentation was also demonstrated in albino rabbits using a new light apparatu with a liquid-filled light-conducting core. Photoaugmentation seems to be an important biological phenomenon in association with sunburn, pigmentation, phototoxicity, phototherapy, photochemotherapy, and UV light-induced cutaneous premalignancies and malignancies. (22 refs)

78-3264 **Solar Radiation: A Possible Etiological Factor in Malignant Melanoma in Israel. A Retrospective Study (1960-1972).** (Eng) Anaise, D. (Surgical Service Office, Veterans Admin. Hosp., Northport, NY, 11568); Steinitz, R.; Ben Hur, N. *Cancer* 42(1): 299-304; 1978.

The role of excessive sunlight exposure on the incidence of malignant melanoma was studied based on 966 malignant melanoma cases reported to the Israeli cancer registry between 1960 and 1972. The annual incidence rose from 20/million in 1960 to 40/million in 1970. The age-adjusted incidence rate was 34/million among Jews from Europe and America, 2.7/million among Jews from Africa and 4.1/million among Jews from Asia. The incidence among European-born Jews who had been expose o the Isreali climate for 20-30 yr prior to diagnosis as 58/million, compared with only 17/million among uropean-born Jews who had immigrated to Israel nly 2-5 yr before diagnosis. The disease tended to anifest itself on the lower extremities in women (55% n those aged 50-79 yr and 41% in those aged 0-19 yr) and n the trunk in men (30.7%). The preponderance of the disease on the lower extremities of women was higher in those vho had been in Israel for 20-30 yr (61%) than in those who ad been in the country only 2-5 yr (49%). The age-adjusted ate was 20/million for Jerusalem compared with 35/million nd 32/million for Haifa and Tel Aviv, respectively, and it vas 54/million among Kibbutz dwellers compared with 17/million among city dwellers. It is suggested that the dark kin of the non-Caucasian races protects them against the evelopment of malignant melanoma. (13 refs)

78-3265 **Effect of Ultrasonic Irradiation on Mammalian Cells and Chromosomes in Vitro.** (Eng) Rosebo-ro, J. A. (Jet Propulsion Lab., California Inst. Technology, Pasadena, CA); Buchanan, P.; Norman, A.; Stern, R. *Phys Med Biol* 23(2): 324-331; 1978.

The effect of ultrasound on human peripheral blood lymphocytes, RBC's, and HeLa cells was examined in vitro at an ultrasonic frequency of 65 kilohertz. The whole blood and HeLa cell suspensions were exposed to continuous and pulsed ultrasonic power levels of 0.12-2.24 watts (W) for 1 min. Irradiation was carried out with the whole blood or HeLa cell suspensions coupled directly to a cylindrical transducer; heating of the cell suspensions above 41 C was avoided. Irradiated and unirradiated peripheral blood lymphocyte chromosome cultures were prepared and scored for selected numerical and morphological aberrations. There was no significant difference in the frequency of chromosomal aberrations between irradiated and unirradiated cells. Cell survival decreased with time after ultrasonic irradiation. The surviving fraction of lymphocytes was an exponential function of the ultrasonic energy to which the cells were exposed independently of the transducer power output over the full power range. RBC survival was independent of power only at levels > 0.7 W. Below this threshold, no destruction of RBC's was observed. The finding of an energy threshold for cell destruction has implications for the biological hazards of ultrasound, showing that peak power as well as av power is an important consideration. (8 refs.)

See also:
*(Rev.): 78-3007, 78-3023, 78-3050, 78-3051, 78-3052, 78-3053, 78-3055.
*(Chem.): 78-3075, 78-3159, 78-3225.
*(Immun.): 78-3428, 78-3450, 78-3452.
*(Path.): 78-3474, 78-3498, 78-3501, 78-3522, 78-3549.
*(Epid.-Biom.): 78-3571, 78-3582, 78-3583.

78-3266 **Virus-Specific RNA Species Present in the Cytoplasm of Rous Sarcoma Virus-infected Chicken Cells.** (Eng) Brugge, J. S. (Dept. Pathology, Univ. Colorado Medical Center, Denver, CO, 80262); Purchio, A. F.; Erikson, R. L. *Virology* 83(1): 16-26; 1977.

The species of virus-specific cytoplasmic RNA found in wild-type (wt) and transformation-defective (td) Rous sarcoma virus (RSV)-infected chicken cells were studied. The RNA from each infected cell type was separated into polyadenylated and nonpolyadenylated RNA fractions, sedimented through sucrose gradients, and hybridized with a complementary DNA probe to identify the virus-specific RNA species. The ability of each class of RNA to serve as messenger RNA (mRNA) was ascertained by analysis of the RNA released from purified polyribosomes by puromycin. Two major species of virus-specific RNA were detected in the poly(A) fraction of wt and td RSV-infected chicken cells: one sedimenting at 35S and one sedimenting at 21S. Both species were released from polyribosomes with puromycin, the proportion of 35S RNA relative to 21S RNA being decreased in the puromycin-released RNA. A small amount of virus-specific RNA sedimenting slower than 14S was also detected in puromycin-treated and untreated RNA samples. The presence of two distinct virus-specific mRNA molecules suggests that the integrated provirus has internal sites for the initiation of transcription or that individual mRNA molecules are cleaved from 35S RNA. (32 refs)

78-3267 **Virus-specific RNA in Selected Cell Lines Derived from RSV-induced Tumour.** (Eng) Kisselyov, F. L. (Cancer Res. Center, Acad. Medical Sciences USSR, Moscow, USSR); Tatosian, A. G.; Svoboda, J. *Folia Biol (Praha)* 23(4): 225-234; 1977.

The presence of virus-specific RNA was studied in three Rous sarcoma virus (RSV)-transformed mammalian tumor cell lines. Line XC, derived from Wistar rats, was transformed by the Prague strain of RSV (PR-RSV) and is characterized by virogenic cells that contain a complete RSV genome. The other two tumor cell lines, RVP_3 (induced in mice by PR-RSV) and RVA_4 (induced in mice by the Schmidt-Ruppin strain of RSV), were nonvirogenic and did not contain any viral genome. Cellular RNA was hybridized to a PR-RSV ^3H-DNA probe synthesized by the endogenous RNA-directed DNA polymerase reaction. The RNA from RVA_4 cells did not hybridize with the ^3H-DNA probe of PR-RSV. The max level of hybridization was 28%-30% for RVP_3 and 10%-12% for XC cells. It was calculated that the virus-specific RNA amounts to 0.01% a 0.2% of the total high-mol-wt RNA in XC and RV cells, respectively. Sucrose gradient centrifugati indicated that the virus-specific RNA sedimented 20S-25S. RNA molecules that would have correspond to the complete virus genome (30S-40S) were nev seen. (27 refs.)

78-3268 **Integrated and Nonintegrated Viral DNA Chick Cells Infected with the Rous Sarcom Virus (Meeting Abstract).** (Fre) Stedman, N. (Laboratoire Biologie Cellulaire et Moleculaire, Institut de Cancerolog et d'Immunogenetique, 14, avenue Paul-Vaillant-Couturie 94800 Villejuif, France); Mariage, R.; Goubin, G.; Rousse M.; Hillova, J.; Hill, M. *Bull Cancer (Paris)* 65(1): 40; 197 (no refs)

78-3269 **Identification of a Transformation-specific Pr tein Induced by a Rous Sarcoma Virus.** (Eng Jay, G. (Pediatric Oncology Branch, Lab. Molecular Biolog NCI, Bethesda, MD, 20014); Shiu, R. P.; Jay, F. T.; Levin A. S.; Pastan, I. *Cell* 13(3): 527-534; 1978.

A protein with an apparent mol wt of 56,000 daltons (56K and an isoelectric point of 6.3 was identified in extracts chick embryo fibroblasts transformed by a wild-type nonde fective Rous sarcoma virus (Schmidt-Ruppin strai SR-RSV) using antisera from rats bearing SR-RSV-induce tumors. This protein was not detected in cells infected b transformation-defective mutants with either a partial complete deletion of the *src* gene or in cells infected by nontransforming avian leukosis virus. The 56K protein wa synthesized at both the permissive and nonpermissive tem peratures in cells infected by either of two conditional-leth mutants (Ts68 and T5) that are temperature-sensitive in ce transformation. The 56K protein also accumulated to th same extent at both temperatures. However, the amount this protein that accumulated in cells infected by Ts68 or T relative to the structural polypeptides, differed significantl from that seen with the nondefective virus. The protein extremely unstable, with a half-life of 20 min, and it is n a precursor to any of the detectable virion polypeptides. Incu bation of the rat antiserum with purified, disrupted virus di not affect its immunoreactivity to this particular protein. is concluded that the 56K protein is a nonstructural protei specific to cells transformed by RSV. (37 refs)

-3270 **The Detection of Glycoproteins Immunological-**
ly Related to RSV gp85 in Uninfected Avian
ells and in Sera from Uninfected Birds. (Eng) Bosch, V.
mperial Cancer Res. Fund Labs., Lincoln's Inn Fields, Lon-
)n WC2A 3PX, England); Kurth, R.; Smart, J. E. *Virology*
)(1): 226-240; 1978.

ifferent antisera were used to examine the presence and
tent of relatedness of components that cross-react with the
ral glycoprotein (gp85) in avian cell extracts and in sera
om uninfected birds. Cell extracts from chick helper fac-
r-negative (chf-) fibroblasts contained components detected
/ anti-avian myeloblastosis virus (anti-AMV) gp85 and
ti-Prague B Rous sarcoma virus (RSV) sera, but not an-
-Prague C gp85 serum. Chf(+) chicken cell extracts con-
ined in addition an antigenic determinant, presumably viral
)85, recognized by anti-Prague C gp85 serum. The sera
om uninfected chickens may contain two components im-
unologically related to gp85. One component recognized
a radioimmunoprecipitation assay (RIA) employing an-
-AMV gp85 serum was present in high amounts in the sera
f chf(+) and chf(-) birds, but a second component detected
an RIA employing anti-Prague B RSV serum was present
nly in the sera of chf(+) birds. It is suggested that the com-
onents that immunologically cross-react with, but are not
lentical to, endogenous viral glycoprotein are products of
enes that are related to the *env* gene of Rous-associated virus
/pe 0 but are nonfunctional as viral glycoprotein. (38 refs)

8-3271 **The Generation of the Two Envelope Glyco-**
proteins of Rous Sarcoma Virus from a Com-
on Precursor Polypeptide. (Eng) Klemenz, R. (Swiss Inst.
xperimental Cancer Res., 1066 Epalinges, Switzerland);
iggelmann, H. *Virology* 85(1): 63-74; 1978.

he relationship of two envelope glycoproteins of Rous sar-
oma virus (gp85 and gp35) to each other and to a 92,000-dal-
on polypeptide from infected chicken embryo fibroblasts was
udied. The two envelope glycoproteins were purified from
H-arginine- and ^{35}S- and ^{3}H-methionine-labeled virus by aga-
ose gel filtration, and gp35 was further purified by sodium
odecyl sulfate gel electrophoresis. After removal of the ter-
inal sialic acid residues, the two glycoproteins were oxi-
ized and trypsinized, and the resulting peptides were sepa-
ated to a cation-exchange column. The results suggested that
p85 and gp35 do not share their major methionine- or argi-
ine-labeled peptides. Using anti-gp85 serum, an intracellular
rotein with an apparent mol wt of 92,000 daltons was im-
unoprecipitated from pulse-labeled infected chicken em-
ryo fibroblasts. The tryptic peptide elution pattern of this
rotein showed striking similarities to those obtained from
p85 and gp35: the four arginine-containing oligopeptides of
e 92,000-dalton protein were present in gp85 plus gp35.
hus, the 92,000-dalton protein appears to be the precursor
f the two viral glycoproteins. Using preferential labeling of
e 92,000-dalton polypeptide, it was determined that gp85
is located in the N-terminal position of the precursor molecule and gp35 is located in the C-terminal position. (31 refs)

78-3272 **Biochemical Properties of Avian Type-C Virus *gag* Gene-coded Proteins: Comparison with Structural Proteins Coded for at Analogous Positions Within the Mammalian Type-C Viral Genome.** (Eng) Reynolds, F. H. (Viral Oncology Program, NCI/Frederick Cancer Res. Center, P.O. Box B, Frederick, MD, 21501); Hanson, C. A.; Stephenson, J. R. *Virology* 86(1): 177-185; 1978.

The biochemical properties of the *gag* gene-coded structural proteins of two avian C-type virus isolates, avian myeloblastosis virus and the Prague strain of Rous sarcoma virus, were examined and compared with those of the previously characterized Rauscher murine leukemia virus *gag* gene proteins. The avian virus structural proteins had mol wts of 27,000, 19,000, 15,000, and 12,000 daltons. The results revealed striking similarities between proteins mapping at corresponding positions within the respective genomes of the avian and mammalian C-type viruses. These two groups of viruses may be evolutionarily related. (32 refs)

78-3273 **The Function of a Virus-coded Molecule Responsible for Malignancy.** (Eng) Bader, J. P. (Lab. Tumor Virus Genetics, NCI, NIH, Bethesda, MD, 20014). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Disease, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 443-445; 1978.

The function of a Rous sarcoma virus (RSV) protein responsible for malignancy was investigated in cells infected with the RSV mutant RSV-BH-Ta, which induces morphological transformation when infected cells are maintained at 37 C but not at 41 C. Shift of RSV-BH-Ta-infected cells from 41 to 37 C resulted in morphological changes (intense cytoplasmic vacuolization, increase in cell size, increase in cell water) that were detectable within 30 min. These changes were independent of glucose uptake, hyaluronate synthesis, alkaline phosphatase, adenosine deaminase, or the large external transformation-sensitive protein. Of the components in the culture medium, only NaCl was necessary for vacuolization; Cl^- could be replaced by any of several anions without affecting vacuolization, but substitution of other cations for Na^+ resulted in distinguishable morphologic changes. Change in temperature to 41 C when Na^+ was the cation resulted in vacuole loss, but with other cations, the vacuoles failed to revert. These findings, combined with the results of staining tests using the cationic dye Neutral Red, suggested that the vacuoles are cation-specific organelles with channels extending to the cell surface. Vacuolated transformed cells had an increased Na^+ uptake but no difference in K^+ uptake compared with normal cells. This suggested that the vacuoles are responsible for increased Na^+ uptake. The transformation protein required neither RNA nor protein synthesis to function, but it did require glycosylation as part of its metabolic activation. (5 refs)

78-3274 **Absence of Adenosine Deaminase Activity in a Mammalian Cell Line Transformed by Rous Sarcoma Virus.** (Eng) Shipman, C. (Dental Res. Inst., Univ. Michigan, Ann Arbor, MI, 48109); Drach, J. C. *Science* 200(4346): 1163-1165; 1978.

An established line of mammalian cells (B-mix K-44/6) that lacks detectable adenosine deaminase (AD) activity is described. The B-mix K-44/6 cells were subcloned from a population of embryonic rat cells transformed in vitro by the Prague strain of Rous sarcoma virus. AD activity was measured in B-mix K-44/6, baby hamster kidney (B-mix K-44/6), BHK-21/4, and human epidermoid carcinoma (KB) cells. The respective AD activities were <0.004, 8.21, and 22.7 nanomoles substrate degraded/min/mg protein. Rates of deamination of arabinosyladenine (ara-A) and adenosine were compared for the three cell types grown in monolayer cultures in a medium essentially devoid of AD activity. Ara-A (100 μM) was deaminated to arabinosylhypoxanthine with a half-life of 2 hr in KB cells and 3 hr in BHK-21/4 cells; KB cell deamination could be detected after a 53-hr incubation period with B-mix K-44/6 cells when ara-A or adenosine were used. Similar findings were obtained with 105,000g cytosol preparations from B-mix K-44/6 cells. Since this line of cells can be readily adapted to growth in medium containing horse serum, a deaminase-free system not requiring the use of an AD inhibitor is available. (31 refs)

78-3275 **Genetic Control of Susceptibility of Mice to Rous Sarcoma Virus Tumorigenesis. II. Segregation Analysis of Strain A.SW-associated Resistance to Primary Tumor Induction.** (Eng) Whitmore, A. C. (Dept. Bacteriology and Immunology, Sch. Medicine, Univ. North Carolina at Chapel Hill, Chapel Hill, NC, 27514); Babcock, G. F.; Haughton, G. *J Immunol* 121(1): 213-220; 1978.

The inheritance of susceptibility of mice to primary sarcomas induced by neonatal injection of Rous sarcoma virus (RSV)-induced chicken tumor material was studied in segregating populations derived from F1 hybrids between the resistant strain A.SW (*H-2-8*) and the susceptible strains A/WySn, B10.A (both *H-2-a*), CBA/J (*H-2-8*), and C57BL/10 (*H-2b*). In F1 hybrids between A.SW and each of the four susceptible strains, res tance is apparently dominant. In the (A/WySn x A.SV F2, (A.SW x B10.A)F2, and (A.SW x CBA/J)F2 gene tions, tumor incidence was significantly associat with *H-2* type. *H-2-aa* and *H-2-kk* homozygot were as susceptible as A/WySn, B10.A, and CBA/J parent strain mice; and *H-2-88* homozygotes were as resistant A.SW parental mice. *H-2-as* and *H-2-ks* heterozygotes we of intermediate susceptibility. No evidence was se for association of susceptibility with three coat col genes or with sex. There was no maternal influence inheritance of susceptibility. In a (B10.LP x A.SW) C57BL/10 backcross generation, *H-2-sb* segregants we significantly more resistant to Rous tumorigenesis th were *H-2-bb* segregants. No influence of genes link to *agouti* or of X-linked genes was seen in this bac cross generation. When mice of high and low prima tumor incidence strains were immunized with t same allogeneic Rous sarcomas, resistant strains develope stronger cellular anti-Rous tumor immunity (as measured b the Winn lymphocyte-tumor cell transfer assay) than did sus ceptible strains. (42 refs)

78-3276 **Replication of the Chick Embryo Neuroretina Cells Transformed by the Rous Sarcoma Virus Genetic Analysis (Meeting Abstract).** (Fre) Poirier, F. (Institut du Radium, 91405 Orsay, France); Calothy, G. *Bul Cancer (Paris)* 65(1): 33; 1978. (no refs)

78-3277 **Murine Rous-Sarcoma-specific Immunity Detected by Leukocyte Adherence Inhibition: Inhibitory Effect of Normal Serum.** (Eng) Wikstrand, C. J (Dept. Pathology, Duke Univ. Medical Center, Duke Univ. Durham, NC, 27710); Proctor, J. O.; Hartsell, S. C.; Haughton, G. *Int J Cancer* 21(5): 639-647; 1978.

The standard one-stage lymphocyte adherence inhibitio (LAI) assay was used to investigate cellular and humoral immune responses in the murine Rous sarcoma system. Significant cellular reactivity to Rous sarcoma antigen extracts wa detected when the responder peritoneal exudate cells (PEC were obtained from C57BL/10 ScSn mice bearing primar or transplanted Rous sarcomas or from immunized mice. N cellular reactivity to methylcholanthrene (MC)-induced tumor antigen extracts was observed. Similarly, specifi cell-mediated recognition of MC tumor antigen extracts wa demonstrated. Initial experiments to assess the role of serum components in the LAI assay demonstrated that fresh o frozen serum from normal C57BL/10 ScSn, B10.D2, or A/WySn mice (aged <6 wk or >7 mo) nonspecifically abrogated the specific loss of PEC adherence. (33 refs)

78-3278 **Structural Studies of Polypeptide p12 From Rous Sarcoma Virus (Meeting Abstract).** (Eng)

/iesemann, M. (Duke Univ. Medical Center, Durham, NC, 7710); Sharief, F. S.; Herman, A. C.; Vanaman, T. C. *Fed roc* 37(6): 1773; 1978. (no refs)

8-3279 **Comparative Studies of the Phospholipids of Rous Sarcoma Virus Transformed and Uninɛcted Chicken Embryo Fibroblasts and Their Plasma Memranes (Meeting Abstract).** (Eng) Maldonado, R. L. (Scheie ye Inst., Univ. Pennsylvania, Philadelphia, PA, 19104); lough, H. A. 37(6): 1597; 1978. (no refs)

8-3280 **Synthesis of Phospholipids During Infection of Chick Embryo Cells by the Rous Virus (Meetıg Abstract).** (Fre) Audubert, F. (Groupe de Recherche no. , Institut Gustave-Roussy, 94800 Villejuif, France); Semıel, M. *Bull Cancer (Paris)* 65(1): 32; 1978. (no refs)

8-3281 **Role of B Cells in the Expression of Genetic Resistance to Growth of Rous Sarcoma in the Chicken.** (Eng) McBride, R. A. (Dept. Pathology, New York Medical Coll., Valhalla, NY, 10595); Watanabe, D. H.; chierman, L. W. *Eur J Immunol* 8(2): 147-149; 1978.

'o determine the possible role of antibody-forming cells (B ells) in the development and growth of Rous sarcoma, the ffects of neonatal bursectomy (Bx) on Rous sarcoma virus RSV)-inoculated genetically resistant and susceptible chickns were studied. The chickens were from two inbred lines hat differed with respect to the major histocompatibility omplex (MHC) antigens: G-B1 (susceptible to Rous saroma) and G-B2 (resistant). Newly hatched birds were Bx nd given 4 mg cyclophosphamide immediately and 2 days ater. At 5 wk of age, sera were tested for immunoglobulin; t 6 wk, tumors were induced by wing web injection of the chmidt-Ruppin strain of RSV. The Bx birds showed absent r only trace quantities of IgG and normal amounts of IgM. The rate of Rous sarcoma growth was greater among the Bx irds throughout the study period. Tumors induced in Bx and ıon-Bx G-B1 birds grew at a similar rate up to day 21, at vhich time tumor regression began to occur in some non-Bx irds. Rapidly growing tumors developed in all G-B1 birds nd Bx G-B2 birds, but in only 25% of non-Bx G-B2 birds. 3x G-B1 and Bx G-B2 birds showed a higher mortality rate han the non-Bx controls. The data indicate that Bx alters he expression of the resistance trait controlled by the R-Rs-1 ene in line G-B2 birds. It is likely that the delayed and weak esponse of some non-Bx G-B1 birds to Rous sarcoma, which ppears to be B cell-dependent, has a different basis than the nore vigorous early response displayed by non-Bx G-B2 irds. (22 refs)

78-3282 **Effects of Dexamethasone on the Morphogenesis of Two Mutants of Rous Sarcoma Virus.** (Eng) Ogura, H. (Dept. Biochemistry, Cancer Inst., Okayama Univ. Medical Sch., Okayama, Japan); Harms, E.; Rohrschneider, J. M.; Bauer, H. *Intervirology* 10(1): 1-10; 1978.

The effect of 10^{-6} or 10^{-5} M dexamethasone (DXM) on the Bryan high-titer strain of Rous sarcoma virus [BH RSV(-)Q, clone 3] and on *LA* 334, two replication-defective mutants of avian sarcoma virus, was investigated. Treatment of Japanese quail cells transformed by BH RSV(-)Q, clone 3, with DXM (either dose) resulted in the appearance of intracytoplasmic A-type particles; these particles were not present in the absence of DXM. The particles had an overall diameter of 65-75 nanometers, and they consisted of two concentric shells surrounding an electron-lucent central core 40-50 nanometers in diameter. Treatment with $> 10^{-5}$ M DXM caused cytotoxicity and no A-type particles were noted. Chick embryo cells (CEC) infected with the temperature-sensitive mutant *LA* 334, which is defective for both transformation and viral replication, showed an abnormal accumulation of viral core substances and aberrant budding at the nonpermissive temperature (41 C). CEC infected with *LA* 334 and treated with DXM exhibited a more extensive accumulation of abnormal budding without an increased release of viral particles. However, DXM did not lead to an abnormal morphogenesis of virus under permissive conditions. It is suggested that the effect of DXM is amplified in the viral replication-defective systems. (37 refs)

78-3283 **Proteins of Rous-associated Virus 61, an Avian Retrovirus: Common Precursor for Glycoproteins gp85 and gp35 and Use of Pactamycin to Map Translational Order of Proteins in the *gag*, *pol*, and *env* Genes.** (Eng) Shealy, D. J. (Biophysics Lab., Graduate Sch., Univ. Wisconsin-Madison, Madison, WI, 53706); Rueckert, R. R. *J Virol* 26(2): 380-388; 1978.

Chick embryo cells infected with Rous-associated virus 61 (RAV-61), a subgroup F virus, contained a precursorlike protein, pr90, that was specifically precipitated by antiserum against the envelope glycoproteins gp85 and gp35 of Rous sarcoma virus, strain Prague B. Tryptic peptide mapping showed that pr90 contained the tryptic sequences of both gp85 and gp35. Pactamycin mapping experiments indicated that the two glycoproteins are translated from the *env* gene messenger RNA (mRNA) in the order (5′) gp85-gp35. These experiments also indicated a translational order of p19-(p27,p12)-p15 for the *gag* proteins, which was the same as that obtained in pactamycin mapping studies of the precursor pr76 of avian myeloblastosis virus. Therefore, the stoichiometry of the RAV-61 core proteins was apparently unchanged when the virions were assembled in the presence of pactamycin. The reverse transcriptase proteins, unlike those of the *env* and *gag* genes, fell on the right side of the pactamycin map. This result is in agreement with the concept that most, if not all, of the reverse transcriptase protein is translated by a readthrough of the *gag*(*pol*) message, rather than by translation of a hypothetical *pol*-mRNA devoted solely to the synthesis of that protein. (34 refs)

78-3284 **Detection of Nucleotide Sequences Related to the Transforming Gene of the Avian Retrovirus in Normal Vertebral DNA (Meeting Abstract).** (Fre) Roussel, M. J. (Unite de Virologie, no. 102, INSERM, place de Verdun, 59045 Lille, France); Stehelin, D. *Bull Cancer (Paris)* 65(1): 39-40; 1978. (no refs)

78-3285 **Size Distribution of Globin Pre-mRNA in AEV Transformed Erythroblasts (Meeting Abstract).** (Eng) Therwath, A. (Institut de Recherche en Biologie Moleculaire, Tour 43, 2 Place Jussieu, F. 75021, Paris, France); Mengod, G.; Scherrer, K. *Bull Cancer (Paris)* 65(1): 32-33; 1978. (no refs)

78-3286 **Avian Myelocytomatosis and Erythroblastosis Viruses Lack the Transforming Gene *src* of Avian Sarcoma Viruses.** (Eng) Stehelin, D. (Virology Unit 102, Place de Verdun, 59045 Lille, France); Graf, T. *Cell* 13(4): 745-750; 1978.

Molecular hybridization experiments with complementary DNA specific for the detection of the *src* transforming gene of avian sarcoma viruses (ASV) showed that avian myelocytomatosis virus strain MC29 and avian erythroblastosis virus strain ES4 lacked nucleotide sequences related to the *src* gene. Furthermore, virus-transformed chicken fibroblasts and hematopoietic cells showed no enhanced level of transcription of the cellular nucleotide sequences related to the *src* gene. Thus, these two replication-defective viruses may contain their own transforming gene(s) or induce cellular genes unrelated to the *src*-like cellular sequences. (33 refs)

78-3287 **Avian Myeloblastosis Virus DNA Polymerase Directed Full-Length Globin and Procollagen Transcripts: Characterization of Template Specific Reaction Conditions (Meeting Abstract).** (Eng) Krueger, L. J. (NHLBI, NIH, Bethesda, MD, 20014); Anderson, W. F. *Fed Proc* 37(6): 1604; 1978. (no refs)

78-3288 **The Influence of Carbohydrate on the Antigenicity of the Envelope Glycoprotein of Avian Myeloblastosis Virus and B77 Avian Sarcoma Virus.** (Eng) Van Eldik, L. J. (Dept. Microbiology and Immunology, Duke Univ. Medical Center, Durham, NC, 27710); Paulson, J. C.; Green, R. W.; Smith, R. E. *Virology* 86(1): 193-204; 1978.

The immunological reactivity of the surface glycoproteins (gp85) of avian myeloblastosis virus (AMV) and B77 avian sarcoma virus was examined by competition radioimmunoassay (C-RIA). The influence of the protein and carbohydrate portions of the glycoprotein on the antigenic reactivity was investigated by systematic digestions of AMV gp85 with proteases and glycosidases. Digestion of AMV gp85 with trypsin, trypsin and chymotrypsin, or Pronase had no effect on the antigenicity detected in C-RIA's using rabbit antibody against AMV gp85. However, digestion of AMV gp85 with a mixture of neuraminidase, β-galactosidase, β-N-acetylglucosaminidase, and endo-β-N-acetylglucosaminidase eliminated the reactivity detected in C-RIA with the same antisera. Sequential digestion of AMV gp85 with various combinations of glycosidases indicated that removal of sialic residues had no effect on antigenicity and that remova of β-galactose and β-N-acetylglucosamine residue abolished antigenic reactivity. Furthermore, glycosidase digestion of ^{125}I-labeled AMV gp85 decreased the reactivity in direct RIA. Thus, the immunological reactivity of AMV gp85 depends upon an intact carbohydrate side chain. Digestion of AMV gp85 with proteases and glycosidases, followed by C-RIA using antibody against B77 gp85, showed that protease treatment resulted in a partial loss of antigenic reactivity. Treatment of B77 gp85 with proteases and glycosidases, followed by C-RIA using antibody prepared against B77 gp85, showed that protease treatment resulted in a partial loss of antigenic reactivity, glycosidase treatment in no loss of antigenic reactivity. (33 refs)

78-3289 **Structure of Avian Tumor Virus DNA Intermediates.** (Eng) Guntaka, R. V. (Dept. Microbiology, Coll. Physicians and Surgeons, Columbia Univ., New York, NY). *Biochem Biophys Res Commun* 82(1): 335-341; 1978.

The mechanism of conversion of virion RNA to double-stranded DNA in avian sarcoma virus (ASV)-infected duck embryo cells was studied. ASV-infected fibroblasts were fractionated, and the nucleic acids were extracted and centrifuged to equilibrium in an isopycnic Cs_2SO_4 gradient; in some cases, heat denaturation or treatment with alkali was performed before banding. The results indicated the presence of RNA-DNA duplexes consisting of DNA minus strands and virion RNA covalently linked to plus-strand DNA. Nucleic acids of the various pools were analyzed in neutral rate zonal sucrose gradients after heat denaturation and alkali igestion. The sedimentation profiles of all pools showed that ie minus-strand DNA consisted of molecules of complete ngth (28S) as well as one-fourth to one-third genome length 2S to 15S). The plus strands in pool I contained 8S DNA, ome of which were covalently linked to parental RNA. The lus strands of pool III consisted predominantly of 8S pieces nd some duplex molecules with single strand tails or gaps; ere were no free single-strand structures of either polarity. ool IV contained duplex viral DNA of genome size as well s duplex DNA of one-third to one-half length minus strands ase-paired with short plus strands. The data indicate that here is a covalent linkage between virion RNA and newly ynthesized plus-strand DNA and that plus-strand DNA ynthesis initiates at several discrete positions. (11 refs)

8-3290 **Synthesis and Processing of Avian Sarcoma Virus Glycoproteins.** (Eng) Hayman, M. (Can-er Res. Fund, P.O. Box 123, Lincoln's Inn Fields, London VC2A 3PX, England). *Virology* 85(2): 475-486; 1978.

vian sarcoma virus (ASV) glycoproteins gp85 and gp37 are ormed by proteolytic cleavage of a glycosylated precursor olypeptide, pr95, of mol wt 95,000. The intracellular site of ynthesis and cleavage of this precursor polypeptide was tudied in ASV B77-infected chick embryo fibroblasts. Folowing a 30-min pulse label, pr95 was found exclusively in he membrane fraction of infected cells. Of the other two recursor polypeptides, the *gag* precursor was located in both he membrane and cytoplasmic fractions, and the *gag-pol* preursor was located exclusively in the cytoplasmic fraction. ubfractionation of the membrane fraction revealed that pr95 vas associated with the rough endoplasmic reticulum, inlicating that it was synthesized on membrane-bound polyomes. Pulse-chase experiments demonstrated that pr95 was leaved within the membrane fraction, and further fractionaion indicated that this cleavage took place within the endolasmic reticulum of the cell. The cleavage products gp85 and p37 were then transported to the plasma membrane of the ell for incorporation into budding virus. These observations re discussed in relation to virus assembly. (18 refs)

8-3291 **Effect of Dibutyryl Cyclic AMP on Intracellular Levels of Avian Sarcoma Virus Specific NA.** (Eng) Guntaka, R. V. (Dept. Microbiology, Coll. hysicians and Surgeons, Columbia Univ., New York, NY, 0032); Weiner, A. J. *Nature* 274(5668): 274-276; 1978.

he effect of dibutyryl cyclic AMP (dbcAMP) on the inracellular levels of avian sarcoma virus (ASV)-specific RNA vas studied. Primary cultures of chick embryo fibroblasts CEF) were infected with ASV containing the transforming *src*) gene, and virus-specific RNA was monitored by nucleic cid hybridization. Max depression (75%-80%) in the levels f virus-specific RNA was observed after treatment of the cells for 8 hr with 250 μg/ml cAMP. Removal of dbcAMP from cultures after 8 hr completely reversed suppression in > 8-16 hr. There was a corresponding decrease in the levels of virus released into the medium after cAMP treatment. Similar results were obtained with different strains of ASV. When the experiments were carried out using transformation-defective (*src*-negative) ASV-infected cells, cAMP did not affect the levels of viral RNA. When CEF were infected with a temperature-sensitive ASV mutant, virus-specific RNA was specifically reduced in cells treated with cAMP only at the permissive temperature. In another experiment, *src* RNA was suppressed 9- to 10-fold by cRNA, whereas virus-specific RNA was suppressed only 4-fold. The data suggest that dbcAMP, in the presence of the *src* gene product, suppresses virus-specific RNA transcription by an unknown mechanism. (19 refs)

78-3292 **In Vitro Transcription of Reconstituted 35S RNA-tRNAtrp Template Primer Complexes by the Avian Oncornavirus DNA Polymerase: Effect of Temperature on the Size of the DNA Transcripts.** (Eng) Leis, J. P. (Dept. Surgery, Duke Univ. Medical Center, Durham, NC, 27710); Smith, R. E.; Dierks, P.; Parsons, J. T.; Collett, M. S.; Faras, A. J. *Virology* 85(1): 28-42; 1978.

The temperature dependence of the synthesis of long and short DNA transcripts from avian sarcoma virus 35S RNA-transferRNA-trp (RNA-tRNA-trp) template-primer complexes in vitro was studied. After 4 hr incubation at 15 C, about 70% of the DNA product synthesized was in the 100-nucleotide size class (DNA_{100}), whereas DNA molecules as large as 1,000 nucleotides in length were synthesized after 60 min incubation at 30 C. A precursor-product relationship between DNA_{100} and the larger DNA transcripts was established by pulse-chase experiments. Hybridization analysis indicated that the DNA_{100} synthesized at 15 C contains nucleotide sequences complementary to the 5′ end of the viral RNA genome. The longer tRNA-trp-initiated DNA transcripts synthesized at 30 C were found to contain at least some nucleotide sequences complementary to the 3′ end of the viral genome. It is suggested that when the tRNA-trp-primed DNA reaches the 5′-RNA terminus, it becomes single-stranded and then anneals to the redundant sequence at the 3′ end of the RNA, where it is elongated. The temperature-dependent step may be required to facilitate the alignment of the 5′ and 3′ termini of the viral genome to allow continued elongation of the 5′-initiated DNA_{100} transcripts at the 3′ end of the viral genome. (40 refs)

78-3293 **Viral Polyproteins in Chick Embryo Fibroblasts Infected with Avian Sarcoma Leukosis Viruses.** (Eng) Hayman, M. J. (Imperial Cancer Res. Fund, Lincoln's Inn Fields, P.O. Box 123, London WC2A 3 PX, England). *Virology* 85(1): 241-252; 1978.

The synthesis of virus-specific proteins was studied in chicken embryo fibroblasts infected with various isolates of avian sarcoma virus. Detergent extracts of pulse-labeled virus-infected cells were analyzed by immune precipitation. Three virus-specific precursor polyproteins with mol wts of 180,000 (180K), 95K, and 76K were detected, and additional immune precipitation experiments were performed to characterize the first two. The 76K polyprotein is the major precursor to the nonglycosylated *gag* proteins of the virion, and it has been extensively described previously. The 95K protein was precipitated by antiserum against a mixture of the two virion glycoproteins gp85 and gp37, and the 180K protein contained protein sequences in common with the virion polymerase and *gag* proteins. Additional pulse-chase experiments indicated that the 180K protein did not represent a precursor to the major 76K *gag* precursor polyprotein, although it could represent the precursor to the virion polymerase. It is probably synthesized as an occasional read-through product from the *gag* messenger RNA. The data indicate that the three precursor proteins are solely precursors to virion proteins and that they exist independently of the morphological phenotype of the cell. (43 refs)

78-3294 **Contrasting Characteristics of Marek's Disease Herpesvirus Isolated from Chickens with and Without Avian Leukosis Virus Infection.** (Eng) Campbell, W. F. (Dept. Microbiology, Mayo Clinic, Rochester, MN, 55901); Kufe, D. W.; Peters, W. P.; Spiegelman, S.; Frankel, J. W. *Intervirology* 10(1): 11-23; 1978.

The effect of avian leukosis virus (ALV) infection on Marek's disease herpesvirus (MDHV)-induced tumor development was studied. MDHV isolated from chickens free of naturally occurring ALV infection produced characteristic foci in both chicken embryo fibroblasts (CEF) and chicken kidney cells (CKC). MDHV-A, extracted from the feather follicle epithelium of chickens naturally infected with ALV, did not induce cytologic changes in CEF cultures, but did cause focus formation in CKC cultures. ALV was detected in MDHV-A, but not in MDHV preparations. MDHV and MDHV-A were then injected in ALV-free LSI-SPF chickens. MDHV-A elicited a high incidence of early mortality that was not accompanied by the gross tumor spectrum characteristic of Marek's disease, although extensive histologic lesions were present. The differences between MDHV and MDHV-A were not as striking in another line of ALV-free chickens (SPAFAS). In contrast, among conventional chickens with naturally occurring ALV infection, neither MDHV-A nor MDHV caused appreciable early mortality, although both resulted in gross tumor development. These findings indicate that the presence of ALV can influence the in vitro and in vivo characteristics of MDHV. However, they do not imply that MDHV required the presence of ALV to produce disease. (37 refs)

78-3295 **Characterization of an Apparently Nononcogenic Marek's Disease Virus.** (Eng) Schat, K. A. (Dept. Avian and Aquatic Animal Medicine, New York Stat Coll. Veterinary Medicine, Cornell Univ., Ithaca, NY 14853); Calnek, B. W. *J Natl Cancer Inst* 60(5): 1075-108 1978.

An apparently nononcogenic Marek's disease virus (MDV that can protect against MDV and against the non-virus producing MD tumor transplant, JMV, was isolated. Thi virus, SB, and a clone, SB-1, differed from pathogenic isolate in in vitro growth characteristics: failure to induce lymphomas or neural lesions, good growth in chick embryo fibroblasts (CEF) and chick kidney cultures, production o larger plaques in CEF and smaller plaques in CKC than th pathogenic viruses, and production of herpesvirus of turkey (HVT). Serologically, SB could be distinguished from MD and the avirulent HVT. SB failed to induce lesions characteristic of MD during a 6- to 11-wk experimental period. I addition, SB was nononcogenic in immunosuppressed chickens or in chickens inoculated with the virus in ovo. However SB did cause cytolytic infection. The term 'nononcogenic' rather than 'apathogenic' is proposed to class fy this and similar isolates. A total of 500 focus-forming units of SB protected S-strain and P-line chicken against intraarterial challenge with MDV at highl significant levels ($p < 0.005$ and $p < 0.01$, respectively) The lesion spectrum in the MD-positive, but vaccinate chickens was different from that in nonvaccinate birds. This suggests that SB vaccination provided a immune response at the level of transformation. SB-1 protected against JMV challenge at highly significant levels ($p <$ 0.0005) 7 or 14 days postvaccination. Protection was absen in SB-1 vaccinated birds challenged with a transmissible lymphoid tumor transplant, which suggests that the protectio against JMV was specific. (46 refs)

78-3296 **In Vitro Establishment of Marek's Disease Herpesvirus-transformed Productive and Nonproductive Lymphoblastoid Cell Lines.** (Eng) Munch, D. (Dept. Veterinary and Animal Sciences, Univ. Massachusetts, Amherst, MA, 01003); Hohlstein, L.; Sevoian, M. *Infect Immun* 20(1): 315-318; 1978.

Two Marek's disease herpesvirus (MDV)-transformed lymphoblastoid cell lines (JMV-1 and JM-1) were established in vitro. Cell line JMV-1 was established from peripheral JMV lymphoblasts from moribund chickens; it has been maintained in culture for 40 wk. Injection of JMV-1 cells into 1-day-old S-line chickens produced a disease that was indistinguishable from the fatal lymphoblastic leukemia produced by injection of whole blood from moribund JMV-infected chicks. Cell line JM-1 was produced from a MD lymphoma induced by the JM strain of MDV. It has been maintained as a suspension culture for 20 wk. Injection of JM-1 into 1-day-old susceptible P-line chickens resulted in the neural and visceral lymphomas characteristic of MD. Cocultivation with chicken embryo fibroblasts, attempts to rescue MDV from kidney cell cultures established from chickens inoculated with JMV-1 or JM-1, and immunofluorescent staining indicate that JMV-1 is a nonproductive line and JM-1 is a productive line. (6 refs)

78-3297 **Are Spleen Focus-forming Virus Sequences Related To Xenotropic Viruses and Expressed Specifically in Normal Erythroid Cells?** (Eng) Pragnell, I. B. (Beatson Inst. Cancer Res., Garscube Estate, Glasgow, England); McNab, A.; Harrison, P. R.; Osterag, W. *Nature* 272(5652): 456-458; 1978.

To study the role of the spleen focus-forming virus (SFFV) component of the Friend virus (FV) complex during erythroblastosis and subsequent erythroid cell transformation, an SFFV-specific complementary DNA (cDNA) probe was synthesized using FV released from transformed F4-6 cells. The SFFV-specific probe annealed > 80% to the 60S RNA of the FV complex and < 10% to the helper lymphatic leukemia virus (LLV) 70S RNA. Although total FV cDNA contained 25% xenotropic- and 33% AKR ecotropic-related sequences, these sequences were homologous to the helper LLV-related sequences and not to the SFFV cDNA. To test the hypothesis that SFFV specificity may lie in the viral transcription of sequences homologous to those involved in normal differentiation, nuclear RNA from normal erythroid cells was hybridized with the purified SSFV cDNA. These experiments detected sequences homologous to FV cDNA and SFFV cDNA in fetal liver RNA and total fetal nuclear RNA. The significance of these low levels of transcription of virus-specific information remains to be elucidated. (23 refs)

78-3298 **The Sizes of RNA Subunits Isolated from High and Low Leukaemogenic Friend Virus.** (Eng) Evenson, D. P. (Sloan-Kettering Inst. Cancer Res., New York, NY, 10021); Scotto, A.; Pla, D.; de Harven, E. *J Gen Virol* 39(3): 475-486; 1978.

The sizes of noncovalently linked RNA subunits isolated from highly leukemogenic Friend virus derived from the plasma of leukemic mice were compared with the sizes of RNA subunits isolated from low leukemogenic Friend virus grown in tissue culture by two RNA extraction procedures. Histograms derived from electron microscope measurements showed that the lengths of the RNA subunits from tissue culture virus (TCV) appeared to belong to different size classes that were similar regardless of whether the TCV RNA was isolated from 3-, 9-, 36-, or 72-hr cultures. The av sizes of each of these classes were approx 0.4, 0.7, and 1.1 μm, corresponding to mol wts of 0.5, 0.9, and 1.4 x 10^6, respectively. The sizes of noncovalently linked RNA subunits isolated from plasma virus (PV) were significantly larger than those of TCV RNA subunits, with the majority of the PV RNA subunits ranging from 1.4 to 2.5 μm in length, corresponding to mol wts of 1.8-3.0 x 10^6. The max PV RNA subunit length was about 3.5 μm. The histograms suggested that PV RNA may also have size classes, but they are not as evident as those for TCV RNA. Data obtained from sucrose gradient sedimentation of heat-denatured Friend leukemia virus RNA agreed with the electron microscope data. Since it can be expected that an intact, large RNA subunit is needed for max biological expression, the difference in leukemogenic potential observed between TCV and PV could be related to RNA subunit size. (29 refs)

78-3299 **Effect of Toyocamycin on Production of Friend Virus in Culture (Meeting Abstract).** (Fre) Mauchauffe, M. (Laboratoire d'Hematologie Experimentale, Hopital Saint-Louis, 75010 Paris, France); Tavitian, A.; Michel, M. L.; Larsen, C. J. *Bull Cancer (Paris)* 65(1): 36-37; 1978. (no refs)

78-3300 **Alteration of Cellular Ribonucleases Associated with Murine Oncogenic Virus Infection.** (Eng) Drake, W. P. (Section Immunology and Cell Biology, Cancer Res. Center, NIH, NCI, 3100 Wyman Park Drive, Baltimore, MD, 21211); Frazier, L. M.; Sidle, D.; Mardiney, M. R. *Biomedicine* 28(1): 24-28; 1978.

Changes in lymphocyte RNase activity were measured in male BALB/c mice infected with Friend leukemia virus (FLV) or Moloney sarcoma virus (MSV). Lymphocytes from mice infected with FLV possessed elevated levels of RNase activity within 8 days after infection. Mice infected with MSV demonstrated an analogous elevation of RNase activity within 7-9 days postinfection. Cellular RNase activity diminished in the FL model concomitant with the occurrence of significant numbers of erythroblasts in the peripheral blood, reaching a low around day 24 postinfection. RNase activity in lymphocytes from mice infected with MSV returned to normal 1-2 wk subsequent to host rejection of the tumor. It is concluded that elevation of RNase activity within the lymphocyte represents an early event in oncogenic viral infection in these tumor models. Elevation of RNase activity in a target not predestined to undergo immediate neoplastic transformation may represent a mechanism by which the cell can restrict neoplastic transformation. (15 refs)

78-3301 **Enhancing and Inhibiting Effects of Spleen Cells from Tumour-bearing Mice on Growth of Virus-induced Primary Sarcoma.** (Eng) Kimura, H. (Dept. Surgery, Inst. Pulmonary Cancer Res., Sch. Medicine, Chiba Univ., Inohana 1-8-1, Chiba 280, Japan); Aoki, T. *Br J Cancer* 37(4): 553-565; 1978.

The effects of adoptive transfer of spleen cells from tumor-bearing mice on the growth of Moloney murine sarcoma virus (M-MuSV)-induced primary tumors in BALB/c mice were investigated. The effects were bidirectional, depending on the time of lymphocyte injection. When spleen cells taken from tumor-bearing mice 12 days after M-MuSV inoculation were injected into recipient mice before M-MuSV inoculation, the appearance of tumors was significantly delayed and their incidence was reduced. However, when these lymphocytes were injected after the development of tumors in recipient mice, tumor growth was significantly enhanced. The data indicate that these inhibiting and enhancing effects are mediated mainly by T cells. (32 refs)

78-3302 **Syngeneic Tumor Enhancement of Moloney Sarcoma Accompanied by Decreased Immunity in the Regressors.** (Eng) Pasqualini, C. D. (Seccion Leucemia Experimental, Instituto de Investigaciones Hematologicas, Academia Nacional de Medicina, Las Heras 3092, 1425. Buenos Aires, Argentina); Mayer, A. M.; Piazzon, I. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 98-99; 1978.

Sc implantation of a glass cylinder containing Moloney sarcoma tissue in BALB/c mice increased in both tumor growth and mortality. This effect could be transferred by progressor serum. The corresponding regressor serum did not specifically inhibit tumor growth, compared with serum obtained after an sc transplant. Thus, the glass cylinder not only conditions for tumor enhancment, it also decreases tumor immunity. (6 refs)

78-3303 **Hydrophobic Binding Site(s) on Moloney-Murine Leukemia Virus P30.** (Eng) Swanson, S. K. (Dept. Medical Viral Oncology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263); Sulkowski, E.; Manly, K. F. *Virology* 85(1): 211-221; 1978.

The specificity of a hydrophobic binding site(s) on the Moloney murine leukemia virus (Mo-MuLV) p30 protein for binding various amino acid and hydrocarbon ligands was studied. Disrupted Mo-MuLV was applied to a decyl-agarose column and eluted with phosphate-buffered saline, NaCl, and 50% ethylene glycol. The p30 protein thus obtained showed no major change in its group-specific antigenic determinant. There was a positive correlation between the affinity of this protein for alkyl-substituted agarose and the chain length of the alkyl groups. This chain-length dependence of binding and the fact that p30 was eluted with 50% ethylene glycol but not with 1.0 M NaCl indicate that hydrophobic binding was involved. Significant purification of p30 was achieved by application of the disrupted virus to a hexyl-agarose column connected in tandem with an octyl-agarose column, followed by a separate elution of the octyl-agarose column with 50% ethylene glycol. p30 bound to the hydrophobic ligand β-hydroxy-γ-phenoxypropylagarose and to L-tryptophyl-L-tryptophan-agarose, but not to phenylalanine-agarose. This affinity can be explained by a general hydrophobic interaction. (33 refs)

78-3304 **Steroid Hormones Increase the Growth of MuLV in Rat Tumor XC Cells.** (Eng) Gautsch, J. W. (Dept. Cellular and Developmental Immunology, Scripps Clinic and Res. Foundation, 10666 N. Torrey Pines Road, La Jolla, CA, 92037); Bedigian, H. G.; Meier, H. *Virology* 83(2): 462-466; 1977.

The effects of various steroid hormones on the growth of Moloney murine leukemia virus (MuLV) in rat tumor XC cells were investigated. Infection of XC cells with 0.1 plaque-forming unit MuLV/cell resulted in the formation of syncytial plaques by 3-5 days; no syncytia formed in cultures that were not able to divide more than twice before confluency. Addition of 10^{-6} M dexamethasone (DXM) increased both the size and number of plaques. Various steroid hormones were then tested for enhancement of plaque formation at doses of 1 and 10 µg/ml. Corticosterone, hydrocortisone, prednisolone, and progesterone were as effective as DXM in inducing plaques; cortexolone, epiandrosterone, and aldosterone were slightly less effective; and allopregnane-3β,20β-diol, androstenedione, androsterone, cortisone, dehydroepiandrosterone, and estriol had minimal activity. Adrenosterone were slightly less effective; and allopregnane-3β,20β-diol, androstenedione, androsterone, cortisone, dehydroepiandrosterone, and estriol had minimal activity. Adrenosterone, deoxycorticosterone, estradiol, estrone, etiocolanolone, pregnanediol, and testosterone had no activity. This hormone-induced increase in virus production was specific for XC cells. It is suggested that the ability of steroid hormones to eliminate the restriction on the growth of MuLV in XC cells reflects a property of the hormone to alter only certain tissues or those in a certain metabolic state and that the ability of DXM to stimulate MuLV production results from action at the level of viral RNA transcription. (19 refs)

78-3305 **Leukemogenicity of Clonal Isolates of Murine Leukemia Viruses.** (Eng) Jolicoeur, P. (Institut de Recherches Cliniques de Montreal, 110 Ouest Avenue des Pins, Montreal, Quebec, Canada); Rosenberg, N.; Cotellessa, A.; Baltimore, D. *J Natl Cancer Inst* 60(6): 1473-1476; 1978.

The leukemogenicity of several clones of in vitro passaged N- and B-tropic BALB/c viruses and two in vitro-passaged clones of Moloney murine leukemia virus (M-MuLV) was examined. The five clones of B-tropic viruses tested induced lymphosarcoma when injected into neonatal BALB/c mice. The frequency of disease was similar to that observed when cloned M-MuLV stocks were used, but the latent period was longer. No pathologic changes were associated with injection of the N-tropic clones tested. B-tropic virus could be recovered from the spleens of leukemic mice and from inoculated, nonleukemic mice. No virus was recovered from mice inoculated with N-tropic clones, indicating that the N-tropic virus clones did not establish a persistent infection in the mice. This study supports previous findings that the naturally occurring B-tropic BALB/c mouse virus is oncogenic in its strain of origin. (23 refs)

78-3306 **MSV Tumor Resistance and Endogenous MuLV Expression in SJL Mice.** (Eng) Colombatti, A. (Netherlands Cancer Inst., Sarphatistraat 108, Amsterdam, Netherlands); D'Andrea, E.; De Rossi, A.; Collavo, D.; Biasi, G.; Chieco-Bianchi, L. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S. eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 94-97; 1978.

Although adult SJL mice do not develop tumors following injection of Moloney murine sarcoma virus (M-MSV), the embryonic cells of this strain are susceptible to transformation, and young SJL mice develop tumors when inoculated with full-strength virus. Genetic experiments suggest that a single dominant gene is responsible for resistance. Resistance was correlated with the expression of high titer endogenous ecotropic murine leukemia virus (MuLV). MuLV may block M-MSV viral receptors, or a cross-reacting natural immunity could account for the resistance of SJL adults. (8 refs)

78-3307 **Structure of the Genome of Moloney Murine Leukemia Virus: A Terminally Redundant Sequence.** (Eng) Coffin, J. M. (Cancer Res. Center, Tufts Univ. Sch. Medicine, Boston, MA, 02111); Hageman, T. C.; Maxam, A. M.; Haseltine, W. A. *Cell* 13(4): 761-773; 1978.

The genome of Moloney murine leukemia virus (Mo-MuLV) was analyzed by digestion with ribonuclease T1 and separation of the digestion products by two-dimensional gel electrophoresis. Thirty large oligonucleotides were isolated and characterized. Of these oligonucleotides, number 21 was present in twice the molar yield of the rest. The 30 oligonucleotides were mapped on the genome by determining their yields in various size classes of 3′ terminal fragments of Mo-MuLV RNA. The physical map thus obtained suggested that oligonucleotide 21 was present at both the 3′ and 5′ ends of the genome. The genome structure was confirmed by analyzing Mo-MuLV RNA oligonucleotides complementary to strong stop DNA, which was a copy of the 5′ terminal 134 nucleotides of the Mo-MuLV genome. Some of the RNA oligonucleotides protected from RNase digestion by hybridization to this DNA, including oligonucleotide 21, were present near both the 3′ and 5′ ends. Comparison of these results with the nucleotide sequence of strong stop DNA showed that there was a terminal redundancy of 49-60 nucleotides in the Mo-MuLV genome RNA. The role of the redundant sequence in the synthesis of proviral DNA is discussed. (24 refs)

78-3308 **Genetic Control of Moloney Virus Replication (Meeting Abstract).** (Fre) Gisselbrecht, S. (Laboratoire d'Immunologie et de Virologie des Tumeurs, INSERM U-152, Hopital Cochin, 75014 Paris, France). *Bull Cancer (Paris)* 65(1): 37; 1978. (no refs)

78-3309 **Murine Leukemia Viruses Containing an Approx 30 S RNA Subunit of Unknown Biological Activity, in Addition to the 38 S Subunit of the Viral Genome.** (Eng) Duesberg, P. H. (Dept. Molecular Biology and Virus Lab., Univ. California, Berkeley, CA, 94720); Scolnick, E. M. *Virology* 83(1): 211-216; 1977.

The detection of a 30S RNA species in (1) Kirsten murine leukemia virus (Ki-MLV) propagated in NIH 3T3 clone 1 cells and (2) Moloney MLV (Mo-MLV) propagated in chronically infected mouse thymus-bone marrow cells (line TB) is reported. The morphology of the cell lines propagating these viruses was indistinguishable from that of uninfected controls. The 30S species existed in addition to the 38S virion RNA. Both the 30S and 38S RNA had the ability to form 50S-70S RNA complexes and to be packaged by virus particles. The size of the Ki-MLV 30S RNA was judged to be 2 x 10^6 daltons based on its electrophoretic mobility; the size of the 30S RNA in Mo-MLV was slightly larger. Since both viruses can exist even if they contain only 38S RNA, it appears that the small RNA species is not an essential component of the viral genome. It is suggested that the 30S RNA is derived from a defective virus unrelated to MLV. (21 refs)

78-3310 **Expression of a Surface Antigen Associated with the Gross Virus by Cells Infected with the Radiation Leukemia Murine Virus (RadLV-Rs) (Meeting Abstract).** (Fre) Gerlier, D. (Centre Leon Berard, 69373 Lyon Cedex 2, France); Guillemain, B.; Dore, J. F.; Duplan, J. F. *Bull Cancer (Paris)* 65(1): 32; 1978. (no refs)

78-3311 **Endogenous Virus Antigens of Murine Lymphoblastoid Cell Lines.** (Eng) Wise, K. (Dept. Microbiology, Univ. Alabama in Birmingham, Birmingham, AL, 35294); Acton, R. *Protides Biol Fluid Proc Collo* 25: 707-714; 1977.

Several lymphoblastoid cell lines were examined for the presence of the endogenous murine leukemia virus (MuLV)-related cell surface antigens gp70 and the Gross cell-surface antigen (GCSA). The AKR thymus-derived BW5147 line expressed relatively large amounts of gp70 (as recognized by anti-gp70 antiserum), as did EL4G+ cells; normal B6 splenocytes showed only low levels of reactivity. The EL4 line expressed reduced amounts of surface gp70, and the incomplete inhibition seen in the absorption curve indicates that gp 70 present on these cells may contain only a portion of the determinants recognized by this system. The antiserum reacted with a cell membrane component with a mol wt of 70,000 and additional material in the range of 40-50,000 daltons. Limited proteolysis suggested that the latter material represented gp70 degradation products. The effect of infection with vesicular stomatitis virus on the expression of cell surface components was investigated in BW5147 and EL4G+ cells. Although no effect on Thy-1, H-2Kk, and gp70 surface components was observed, a marked selective increase in GCSA expression was noted in both lines. (18 refs)

78-3312 **Cell-Free Synthesis of a Precursor Polyprotein Containing Both gag and pol Gene Products by Rauscher Murine Leukemia Virus 35S RNA.** (Eng) Murphy, E. C. (Dept. Biology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); Kopchick, J. J.; Watson, K. F.; Arlinghaus, R. B. *Cell* 13(2): 359-369; 1978.

Evidence is presented that Rauscher murine leukemia virus (RLV) reverse transcriptase (RT) shares tryptic peptides with a 200,000-dalton polypeptide synthesized in vitro from RLV 35S RNA. The latter is identical in its tryptic maps to the *gag*- and *pol*-related polyprotein precursor (Pr200gag-pol) observed in RLV-infected cells. Nine of 11 methionine-containing tryptic peptides found in an anti-RT-precipitable 80,000-dalton virion polypeptide (p80pol) were contained in Pr200gag-pol. In addition, virion p80pol comigrated in sodium dodecyl sulfate-polyacrylamide gels with the major polypeptide found in partially purified preparations of active RT. The in vitro synthesized 200,000-dalton polypeptide was identical to intracellular Pr200gag-pol as determined by comparing ion-exchange profiles of methionine-labeled tryptic peptides. The frequency of the in vitro synthesis of Pr200gag-pol was 1/25 to 1/20 that of the combined synthesis of the RLV 65,000- and 75,000-dalton *gag* precursors. A similar ratio of Pr200gag-pol to Pr65gag and Pr80gag has been observed in virus-infected cells. Thus, 35S viral genomic RNA is a messenger RNA for both *gag* and *pol* gene products. In experiments in which the arginine in the cell-free reaction mixture was replaced by canavanine, protein synthesis was inhibited approx 50% and the major product comigrated with Pr80gag. These findings indicated that the usual termination signal occurs after the translation of Pr80gag and that Pr65gag is derived from Pr80gag by in vitro processing. The Pr200gag-pol may result from an occasional read-through (5% frequency) of a single termination codon at the end of the *gag* gene. (34 refs)

78-3313 **Phagocytosis of Red Blood Cells from Rauscher Leukemia Virus-infected Mice by Macrophages from Normal Syngeneic Mice.** (Eng) Knyszynski, A. (Section Biological Ultrastructure, Weizmann Inst. Science, Rehovot, Israel); Leibovich, S. J.; Danon, D. *J Reticuloendothel Soc* 23(4): 243-251; 1978.

The mechanism by which mouse macrophages interact with RBC from normal and Rauscher leukemia virus (RLV)-infected syngeneic SWR mice was studied in vitro. The "young" but not the "old" RBC from normal mice were phagocytized, whereas both "old" and abnormal "young" cells from RLV-infected mice were usually phagocytized. Incubation of the RBC from RLV-infected mice at 37 C in the presence of neuraminidase did not affect phagocytosis of these cells, but trypsin treatment generally decreased phagocytosis. Adsorption of fetal calf serum with unfractionated or young RLV RBC from RLV-infected mice markedly reduced the phagocytosis of the young RBC. The level of phagocytosis of RBC by mineral oil- or thioglycollate-stimulated peritoneal macrophages was considerably lower than that of unstimulated macrophages. Trypsin treatment of the macrophages almost completely eliminated their capacity to recognize and phagocytize RBC from infected mice. The data indicate that macrophages from normal animals recognize and phagocytize RBC from RLV-infected mice in vitro and that the recognition process depends on the presence of trypsin-sensitive receptors on the surfaces of the macrophages. It would appear that the defense mechanism that prevents the accumulation of deteriorated RBC in the circulation is in charge of the first-line defense mechanism against the development of Rauscher virus leukemia. (22 refs)

78-3314 **Physical and Chemical Properties of an Attenuated Mutant of the Rauscher Virus (Meeting Abstract).** (Fre) Barbieri-Weill, D. (Lab. de Virologie et de Culture de Tissus, Institut Gustave-Roussy, 94800 Villejuif, France); Leibovitch, S.; Athan, E. *Bull Cancer (Paris)* 65(1): 36; 1978. (no refs)

78-3315 **Serological and Biochemical Characterization of the Mouse Mammary Tumor Virus with Localization of p10.** (Eng) Cardiff, R. D. (Dept. Pathology, Sch. Medicine, Univ. California, Davis, CA, 95616); Puentes, M.

J.; Young, L. J.; Smith, G. H.; Teramoto, Y. A.; Altrock, B. W.; Pratt, T. S. *Virology* 85(1): 157-167; 1978.

Antisera against the five major mouse mammary tumor virus (MMTV) polypeptides (gp52, gp36, p28, p14, and p10) were characterized, and the immunological relationship between the virion and subvirion particles was studied. Antisera against p10, gp52, gp36, and p28 precipitated primarily their own antigens, and antiserum against whole virion (anti-MMTV) precipitated gp52, gp36, p28, and p10. Anti-p14 precipitated primarily p28 and p10 rather than p14. p14 was not precipitated by any of the antisera. In a radioimmunoassay, anti-p10 did not precipitate intact iodinated MMTV at any dilution, anti-p28 and anti-p14 produced some precipitation at low dilutions, and anti-MMTV, anti-gp52, and anti-gp36 precipitated the intact virions. These data were confirmed by immunodiffusion assay. Virions, cores, bald particles, and A particles contained p28 and p10 reactivity. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis revealed that p28 and p10 were the major polypeptides in virions and bald particles, but that p10 was reduced in the cores. No mature MMTV polypeptides were identified in the A particles. A salt-detergent extraction for hydrophobic proteins indicated that p10 was hydrophobic and lipophilic. The data suggest that gp52 and gp36 are on the envelope surface, that p28 and p14 are core polypeptides, and that p10 is probably membrane-associated. A model of MMTV maturation is suggested in which p10 links the nucleocapsid to the envelope. (28 refs)

78-3316 **Induction of Mammary Tumors in C57BL/Haag Female Mice by a Low Oncogenic Mammary Tumor Virus.** (Eng) Vlahakis, G. (Gene Regulation Section, Lab. Molecular Biology, NCI, NIH, Bethesda, MD, 20014); Chopra, H. C.; Sawdon, M. R. *Int J Cancer* 21(5): 648-651; 1978.

Newborn C57BL/Haag mice, a substrain that is negative for murine mammary tumor virus (MuMTV), were foster-nursed by a DD strain mother to ensure the introduction of MuMTV into the substrain. (DD strain mice have a high spontaneous mammary tumor incidence). The foster-nursed litter, designated C57BL/HaagfDDF$_1$, was then inbred until the F_4 generation to give a total of 87 females. The overall mammary tumor incidence in the C57BL/HaagfDD line was 62.1%, and the av age at tumor appearance was 20.1 mo. Tumors developed in 5/6 F_1, 19/37 F_2, 27/41 F_3, and 3/3 F_4 mice. Electron microscopy revealed B particles in the tumors, providing additional evidence for the transmission of DD MuMTV. The MuMTV's of DD and RIII mice may be related, since both albino strains originated in Europe, develop pregnancy-dependent mammary tumors, and carry black and agouti genes. (18 refs)

78-3317 **Expression of MuMTV-Related Antigens in Normal and Malignant Human Mammary Epithelial Cells (Meeting Abstract).** (Eng) Yang, N. S. (Michigan Cancer Foundation, Detroit, MI, 48201); Furmanski, P. *Fed Proc* 37(6): 1747; 1978. (no refs)

78-3318 **Effect of a Mouse Mammary Tumor Virus-derived Protein Vaccine on Primary Tumor Development in Mice.** (Eng) Creemers, P. (Tumor Virology Branch, REP Insts., Organization Health Res. TNO, 151 Lange Kleiweg, Rijswijk, ZH, Netherlands); Ouwehand, J.; Bentvelzen, P. *J Natl Cancer Inst* 60(6): 1461-1466; 1978.

The effect of murine mammary tumor virus (MuMTV)-derived protein vaccines on spontaneous tumor development was determined in GR, BALB/cfC3H, BALB/c, and C3Hf mice. Approx 60% of the protein fractions in the vaccines consisted of the major viral membrane glycoprotein gp52. Sc injection of 10 μg MuMTV-S-derived vaccine significantly delayed the appearance of primary mammary tumors in GR and BALB/cfC3H mice (strains with high incidences of mammary cancer). In BALB/c and C3Hf mice (which have moderate tumor incidences at an advanced age), this treatment resulted in a slight and substantial acceleration, respectively, of primary tumor development. The induced cellular immune reactivity for vaccination was stronger in the GR strain than in the BALB/c strain. Antibody titers to tumor cells were also higher in this strain. The influence of different vaccination schemes with an MuMTV-O-derived protein vaccine on primary tumor development was studied in BALB/cfC3H mice. The vaccine was precipitated on alum before it was injected sc. A dose of 10 μg vaccine resulted in a 61% decrease in tumor incidence. Two or five additional booster injections with 1 μg protein vaccine had no beneficial effect, although the amount of antibody was increased after the booster injections. (29 refs)

78-3319 **Structural Components of Mouse Mammary Tumor Virus. II. Isolation and Purification of Virion Polypeptides.** (Eng) Yagi, M. J. (Dept. Neoplastic Diseases, Mount Sinai Sch. Medicine, New York, NY, 10029); Stutzman, R. E.; Robertson, B. H.; Compans, R. W. *J Virol* 26(2): 448-456; 1978.

Mouse mammary tumor virus (MMTV) glycoproteins and nonglycosylated polypeptides, which were obtained from a BALB/cfC3H/Crgl mouse mammary tumor cell line, were purified by preparative sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Primary amino groups were labeled with fluorescamine to enable visualization of MMTV polypeptides in the gels. Protein bands were sliced from the gels and eluted with 90%-95% recovery. Eight MMTV polypeptides, including three of the major viral components and five minor proteins, were obtained routinely. Double-diffusion assays and immunoelectrophoresis confirmed the retention of antigenicity identical to that of untreated MMTV virions.

Antisera from MMTV-free BALB/c mice immunized with these purified proteins reacted with the polypeptide immunogen as well as with detergent-disrupted MMTV virions from mouse milk or cell culture. Double-diffusion assays using the specific mouse antisera failed to detect any cross-reactivity among the isolated polypeptides. A hemagglutination-inhibition assay demonstrated that the ability of MMTV virions to inhibit the hemagglutinating properties of influenza virus resided in the glycosylated polypeptides gp52, gp37.7, and gp33. (21 refs)

78-3320 **Radioimmunoassays That Demonstrate Type-specific and Group-specific Antigenic Reactivities for the Major Internal Structural Protein of Murine Mammary Tumor Viruses.** (Eng) Teramoto, Y. A. (Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD, 20014); Schlom, J. *Cancer Res* 38(7): 1990-1995; 1978.

The 28,000-dalton (p28) major structural polypeptide of mouse mammary tumor virus (MMTV) was isolated and used to develop a highly sensitive and specific radioimmunoassay. Under conditions of limiting antibody in competitive-binding assays, as little as 50 picograms of purified p28, as well as disrupted MMTV virions and mammary tumor extracts, competed specifically with ^{125}I-labeled MMTV p28. The p28 polypeptide was further shown to contain both group-specific and type-specific antigenic determinants, thus allowing for further differentiation of various MMTV strains. By using this radioimmunoassay for MMTV p28 together with previously established radioimmunoassays for MMTV 52,000-dalton glycoprotein, levels of MMTV gene expression can now be analyzed in terms of both viral glycoproteins and nonglycoproteins. This type of comparison is important in light of recent evidence of the noncoordinate polypeptide chain initiation of glycosylated vs nonglycosylated MMTV proteins in infected cells. These combined radioimmunoassays should further clarify MMTV replication and gene expression in murine mammary tumors. (38 refs)

78-3321 **Isolation of Separate Precursor Polypeptides for the Mouse Mammary Tumor Virus Glycoproteins and Nonglycoproteins.** (Eng) Schochetman, G. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD, 21501); Long, C. W.; Oroszlan, S.; Arthur, L.; Fine, D. L. *Virology* 85(1): 168-174; 1978.

Monospecific antisera to the major glycoproteins (gp52 and gp36) and the major nonglycoprotein (p27) of murine mammary tumor virus (MMTV) were used in conjunction with tryptic peptide fingerprinting to isolate an intracellular MMTV precursor polypeptide to p27. All three antisera precipitated polypeptides of about 75,000 daltons; proteins with mol wt of 160,000 (H-1) and 48,000 (H-2) were also precipitated. Polypeptide gPr 75, which was precipitated by anti-gp-52 and anti-gp-36 sera, has been shown to be glycosylated and to be the precursor to gp52 and gp36. Polypeptide Pr75, which was precipitated by anti-p27 serum, had not been isolated previously, and it was not precipitated by normal rabbit serum. Pr75 bound to single-stranded DNA (ssDNA) and was easily separated from gPr75 by ssDNA-Sepharose column chromatography. Analysis of the tryptic peptide fingerprints of the virion proteins and precursor polypeptides showed that gPr75 contained the tryptic peptides of only gp52 and gp36, either of which bound to ssDNA. Fingerprints of the Pr75 precursor revealed the tryptic peptides of p14 and p27. Thus, at least p27 and p14 are derived from Pr75, which is distinct from gPr75. (15 refs)

78-3322 **Synthesis of Mouse Mammary Tumor Virus Ribonucleic Acid in Isolated Nuclei from Cultured Mammary Tumor Cells.** (Eng) Stallcup, M. R. (Dept. Biochemistry and Biophysics, Univ. California, San Francisco, CA, 94143); Ring, J.; Yamamoto, K. R. *Biochemistry (Washington)* 17(8): 1515-1521; 1978.

Procedures were designed to characterize the cell-free synthesis of mammary tumor virus (MTV) RNA in nuclei isolated from murine mammary carcinoma (GR) cells. The nuclei were incubated under conditions in which all three endogenous RNA polymerases were active. RNA synthesized in vitro was distinguished from preexisting nuclear RNA by labeling the in vitro products with ^{3}H-cytosine triphosphate; the level of MTV RNA synthesis was measured by molecular hybridization with unlabeled viral DNA. Synthesis required the addition of nucleoside triphosphates and was inhibited 90% by 10 μg/ml actinomycin D. Pretreatment of GR cells with dexamethasone had no significant effect on the amount of total RNA synthesis in isolated nuclei. In contrast, synthesis of MTV RNA was stimulated 10- to 20-fold in nuclei from dexamethasone-treated cells relative to untreated control nuclei. In vitro viral RNA synthesis was inhibited by 97% following exposure of the nuclei to 0.3 μg/μl α-amanitin, suggesting that this synthesis is carried out exclusively by RNA polymerase II. The fraction of total RNA synthesis that was viral specific (about 0.2%-0.4% in nuclei from dexamethasone-treated cells and 0.01%-0.03% in controls) was similar to that detected in pulse labeled RNA from whole GR cells in culture. This procedure appears to avoid artifacts noted in other in vitro transcription systems. (54 refs)

78-3323 **The Specificity of Neutralizing Antibodies to Feline Leukaemia Viruses.** (Eng) Russell, P. H. (ICRF Tumour Immunology Unit, Dept. Zoology, Univ. Coll. London, London W.C.1., England); Jarrett, O. *Int J Cancer* 21(6): 768-778; 1978.

The specificity of virus-neutralizing antibodies to feline leukemia virus (FeLV) of subgroups A, B, and C was investigated. The FeLV-A isolates were monotypic, but there was antigenic variation among the isolates of subgroups B and C. There was also considerable cross-reactivity between subgroups, especially between subgroups A and C. It is concluded that subgroup classification based on interference did not correspond with neutralizing behavior for subgroups B and C. (24 refs)

78-3324 **Feline Oncornavirus-associated Cell Membrane Antigen: Evidence for an Immunologically Crossreactive Feline Sarcoma Virus-coded Protein.** (Eng) Stephenson, J. R. (Lab. RNA Tumor Viruses, NCI, Bethesda, MD 20014); Khan, A. S.; Sliski, A. H.; Essex, M. *Proc Natl Acad Sci USA* 74(12): 5608-5612; 1977.

The nature of the feline oncornavirus-associated cell membrane antigen (FOCMA) was investigated. Mink and rat cells nonproductively transformed by feline sarcoma virus (FeSV) expressed FOCMA as well as the 5′-terminal feline leukemia virus (FeLV) *gag* gene proteins p15 and p12. Both nonproducer lines were negative for FeLV gp70, p30, and p10. Feline lymphoma cells, including those from several tumors that lacked detectable levels of FeLV structural protein expression, were all FOCMA-positive. In the two nonproducer cell lines, FeLV p15 and p12 expression was associated with 25,000 and 80,000-100,000 mol wt proteins, as determined by agarose gel filtration under denaturing conditions. The 80,000-100,000 mol wt protein also exhibited antigenic determinants in common with FOCMA. Immunoprecipitation and polyacrylamide gel electrophoresis of the 80,000-100,000 mol wt protein indicated the presence of a 65,000 mol wt protein that exhibited only FOCMA determinants. These findings indicate that the 80,000-100,000 mol wt protein represents a precursor containing p15, p12, and FOCMA and that it is subject to posttranslational cleavage, giving rise to a 25,000 mol wt protein containing p15 and p12 and a second protein containing FOCMA antigenic determinants. The demonstration of FOCMA expression in naturally occurring feline tumors, even in the absence of FeLV structural proteins, and the results of previous studies suggest that FOCMA represents a transformation-specific protein. (19 refs.)

78-3325 **Influence of Antisera to Oncornavirus Glycoprotein (gp71) on Infections of Cats with Feline Leukemia Virus.** (Eng) de Noronha, F. (Dept. Pathology, Cornell Univ., Ithaca, NY, 14850); Schafer, W.; Essex, M.; Bolognesi, D. P. *Virology* 85(2): 617-621; 1978.

To determine if there is an interspecies cross-reactivity in in vivo serum therapy studies, 6-mo-old kittens that had been inoculated 6 days previously with feline leukemia virus (FeLV) were treated with either normal goat serum, goat antiserum to FeLV, or goat antiserum to purified Friend leukemia virus (FLV) gp71. All the kittens (9/9) treated with normal goat serum became persistently viremic, but 6/8 given anti-FeLV and 6/9 given anti-FLV gp71 resisted the persistent viremia. Only 1/9 animals that remained viremic by 101 days postinfection had detectable virus neutralizing antibody titers, but 11/12 animals that did not become viremic developed significant levels of this antibody. At 114 days postinfection, two virus-free animals initially treated with anti-FeLV and two virus-free animals initially treated with anti-FLV gp71 were challenged with FeLV. None of the four developed persistent viremia. These findings suggest that viral antigens, in particular their interspecies determinants, can be used as targets for immunotherapeutic regimens for the prevention and control of diseases induced by oncornaviruses. (23 refs)

78-3326 **Distribution of Bovine Leukemia Virus Proviral DNA Sequences in Tissues of Animals with Enzootic Bovine Leukosis (Meeting Abstract).** (Eng) Kettmann, R. (Departement de Biologie Moleculaire, Universite Libre de Bruxelles, Brussels, Belgium); Burny, A.; Cleuter, Y.; Ghysdael, J.; Mammerickx, M. *Bull Cancer (Paris)* 65(1): 40-41; 1978. (no refs)

78-3327 **Radioimmunoassay of Antibodies Specific for the Bovine Leukemia Virus in French Cattle (Meeting Abstract).** (Fre) Levy, D. (Laboratoire d'Immunologie et Virologie des Tumeurs, INSERM U. 152, Hopital Cochin, 27, rue du Faubourg Saint-Jacques, Paris, 14, France); Deshayes, L.; Parodi, A. L. *Bull Cancer (Paris)* 65(1): 38-39; 1978. (no refs)

78-3328 **Biosynthesis of Bovine Leukemia Virus (BLV) Major Internal Protein (p24) in Infected Cells and *X. laevis* Oocytes Microinjected with BLV 60-70S RNA.** (Eng) Ghysdael, J. (Departement de Biologie Moleculaire, Universite de Bruxelles, Brussels, Belgium); Hubert, E.; Cleuter, Y. *Arch Int Physiol Biochim* 85(5): 978-979; 1977.

Xenopus laevis oocytes were inoculated with 60S-70S bovine leukemia virus (BLV) RNA and used to study the biosynthesis of p24, the major internal protein of p24. The oocytes were labeled with ^{3}H-leucine and either frozen immediately or incubated in culture medium containing an excess of unlabeled leucine for up to 2 wk. Lysates were prepared from the different batches, and p24-related sequences were precipitated with anti-p24 serum. Electrophoresis and fluorography indicated that p24 was generated by proteolytic cleavage of a 68,000 to 70,000-dalton precursor polypeptide . The same high-mol-wt precursor was observed when 60S-70S BLV RNA was translated in a reticulocyte lysate treated with micrococca nuclease; however, p24 was not generated in this system. The cleavage pattern in *X. laevis* oocytes was the same as that in fetal lamb kidney cells infected with and producing BLV; cleavage in the oocytes was much slower than that in infected cells. (5 refs)

78-3329 **Further Characterization of a Herpesvirus-positive Orangutan Cell Line and Comparative Aspects of In Vitro Transformation with Lymphotropic Old World Primate Herpesviruses.** (Eng) Rabin, H. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD, 21501); Neubauer, R. H.; Hopkins, R. F.; Nonoyama, M. *Int J Cancer* 21(6): 762-767; 1978.

The orangutan suspension cell line, CP-81, and its associated B-cell-tropic virus, *Herpesvirus pongo*, were characterized. CP-81 lacked myeloid markers of lysozyme activity and phagocytosis, but it was positive for lymphocytic N-alkaline phosphatase activity and it released *H. pongo*. *H. pongo* had 30%-40% DNA homology with Epstein-Barr virus (EBV) and it was present at two to three genome copies per CP-81 cell. Gibbon cell lines transformed by *H. pongo* (733/*H. pongo*), EBV (733/EBV), and *H. papio* (733/H. papio) were established. Cells from all three lines stained positively for virus-associated intracellular antigens. 733/*H. papio* and 733/EBV cells were fibrinolytically active, but only 733/EBV cells produced interferon and lymphotoxin. 733/EBV and 733/*H. papio* cells had the normal diploid chromosome number of gibbon cells, whereas 733/*H. pongo* cells were hypodiploid due to an apparent loss of the Y chromosome. Gibbon lymphocytes transformed by *H. pongo* and EBV and transplanted intracranially (ic) into nude mice showed success rates of 75% and 45%, respectively. Transplants of similar cells transformed by *H. papio* were only 10% successful. None of these lines were able to be transplanted sc, and none manifested a high degree of colony formation in 0.33% agarose. CP-81 cells could be transplanted both ic (100% success rate) and sc (70%), and they showed a relatively high degree of colony formation in agarose. EBV-transformed marmoset cells, line B95-8, could be transplanted ic (66%) but not sc, and they had a low but significant ability to grow in agarose. *H. papio*-transformed baboon cells, line 594S, could be transplanted ic (25%) but not sc, and they grew at very low levels in agarose. (25 refs)

78-3330 **Relatedness Between Genomes of Gibbon Type C Oncornaviruses and the Neoplastic Diseases with Which They are Associated (Meeting Abstract).** (Eng) Sun, L. (Univ. California, Davis, CA, 95616); Kawakami, T. G. *Fed Proc* 37(6): 1745; 1978. (no refs)

78-3331 **Genetic Locus, *Bevi*, on Chromosome Six Controls the Replication of Baboon Type C Virus in Human Cells (Meeting Abstract).** (Eng) Lemons, R. S. (NCI, NIH, Bethesda, MD, 20014); O'Brien, S. J.; Sherr, C. J. *Bull Cancer (Paris)* 65(1): 41; 1978. (no refs)

78-3332 **Isolation of Foamy Virus Type II from Hematopoietic Cells of Baboons with Hemoblastoses and Those of Healthy Animals.** (Eng) Agrba, V. Z. (Inst. Experimental Pathology and Therapy, USSR Acad. Medical Sciences, Gora Trapezija, Sukhumi, USSR); Kokosa, L. V.; Cuvirov, G. N.; Bierwolf, D.; Widmaier, R.; Graffi, A.; Lapin, B. A.; Sangulia, I. A.; Rudolph, M. *Arch Geschwulstforsch* 48(2): 97-111; 1978.

Sixteen monolayer and 5 suspension cultures of hematopoietic (bone marrow, spleen, and lymph node) cells from 2 baboon embryos and 14 baboons aged 1 day to 18.6 yr were studied for foamy virus type II. Four animals were healthy, and 12 had hemoblastoses. Giant cells and foamy virus type II were found in 13 monolayer and 3 suspension cultures of 12 animals. The positive animals included both healthy ones and those with hemoblastoses. Foamy virus was also found in one 1-hr-old baboon, which is indicative of the intrauterine transmission of foamy virus. Some animals also appeared to be contaminated by foamy virus type I. The findings are in agreement with those of other researchers. The role played by foamy virus is not known. (20 refs)

78-3333 **Pathologic and Immunologic Response to the Baboon Endogenous Virus and Its Pseudotype.** (Eng) Kalter, S. S. (Southwest Foundation Res. and Education, San Antonio, TX); Heberling, R. L.; Eichberg, J. W.; Boenig, D. M.; Cummins, L. B. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 100-102; 1978.

The pathologic and immunologic response of various animals to baboon xenotropic endogenous virus (BaEV) and its murine sarcoma pseudotype [MSV(BaEV)] was determined. BaEV did not produce tumors in a wide variety of nonhuman primates, beagle dogs, hamsters, or nude mice. BaEV was isolated from organ explants of beagle dogs inoculated in utero. MSV(BaEV) produced tumors in newborn dogs, chimpanzees, white-lipped and cottontop marmosets, and baboons (following intracerebral inoculation). Cell-mediated immunity to both viruses was demonstrated mainly by a blastogenic response. (15 refs)

78-3334 **Regulation of Viral Functions in SV40 Transformed Mouse Cells (Meeting Abstract).** (Eng)

Zouzias, D. C. (New York Univ. Medical Sch., New York, NY, 10016); Basilico, C. *Fed Proc* 37(6): 1746; 1978. (no refs)

78-3335 **Transformation of Skin Fibroblast Cells of a Cystinotic Patient by Simian Virus 40: Evidence for an Establishment of a Permanent Cell Clone Which Retains the Original Metabolic Defect.** (Eng) Darai, G. (Institut fur Medizinische Virologie, Universitat Heidelberg, Im Neuenheimer Feld 324, D-6900 Heidelberg 1, W. Germany); Harms, E.; Flugel, R. M.; Braun, R.; Munk, K. *Cytobiologie* 17(1): 42-50; 1978.

Simian virus 40 (SV40) was used to transform human cystinotic cells (line HCF-132) in order to obtain a human cystinotic cell line capable of indefinite growth in vitro. The cell clones thus established (SV-HCF-132) showed an epithelial-like morphology, unlike the parental cells, which showed a fibroblastoid morphology. More than 80% of the SV-HCF-132 cells contained SV40 tumor (T) antigen within their nuclei. The parental HCF-132 cells did not show any immunofluorescence typical of T antigen. The transformed cell clones grew in soft agar, but the parental HCF-132 cells did not. The doubling time during exponential growth was 24 hr for SV-HCF-132 and 72 hr for HCF-132 cells; the saturation density was higher for the former cells. The modal chromosome number in transformed clone No. 6 was 68. The cystine content of clone No. 6 was as high as that of the parental cells. The high cystine concentration did not change after 40 passages of the SV-HCF-132 cells. In addition, SV40-transformed skin fibroblasts from a patient with Fanconi's anemia and SV40-transformed cells from a healthy child had normal cystine levels. Thus, the high cystine content of SV-HCF-132 cells was not due to the effects of long-term culturing or to viral transformation per se. The growth characteristics of the SV-HCF-132 clones make them suitable for the large-scale preparation of cellular constituents, eg, lysosomes, which seem to be affected in cystinotic patients. (29 refs)

78-3336 **Characteristics of Protein Phosphorylation in Isolated Plasma Membrane and Reticulum Fractions of SV 40-Transformed Mouse Fibroblasts (Meeting Abstract).** (Ger) Joos, T. F. (Friedrich-Miescher-Laboratorium, Max-Planck-Gesellschaft, Spemannstrasse 37-39, D-7400 Tubingen, W. Germany); Anderer, F. A. *Hoppe Seylers Z Physiol Chem* 359(3): 282; 1978. (no refs)

78-3337 **The 5′-Terminal Leader Sequence of Late 16 S mRNA from Cells Infected with Simian Virus 40.** (Eng) Ghosh, P. K. (Dept. Medicine, Yale Univ. Sch. Medicine, New Haven, CT, 06510); Reddy, V. B.; Swinscoe, J.; Choudary, P. V.; Lebowitz, P.; Weissman, S. M. *J Biol Chem* 253(10): 3643-3647; 1978.

The relationship of the late 16S messenger RNA (mRNA) leader of simian virus 40 (SV40) to the major structural protein (VP1) coding segment of this mRNA was investigated. This 16S mRNA directs the synthesis of VP1, and it is composed of a leader sequence of 203 nucleotides joined to a transcript that contains all the codons for VP1. The leader is transcribed from DNA between 0.723 and 0.762 map unit, but the VP1 coding transcript is copied from DNA from 0.94 to 0.17 map unit. The leader sequence joins the transcript of the coding region at a position 42 nucleotides upstream from the initiator codon for VP1 by a 3′:5′-phosphodiester linkage. Longer leader sequences also occur in a portion of SV40 late mRNA. (31 refs)

78-3338 **The Structure of SV40 Chromatin.** (Eng) Zentgraf, H. (Inst. Virus Res., German Cancer Res. Center, Heidelberg, W. Germany); Keller, W.; Muller, U. *Philos Trans R Soc Lond [Biol]* 283(997): 299-303; 1978.

Simian virus 40 (SV40) nucleoprotein complexes were studied by electron microscopy. Depending on the isolation procedure, SV40 chromatin has two different conformations: complexes isolated in the presence of 0.15 M NaCl had compact globular structures, but those isolated in the presence of 0.6 M NaCl had the typical beads-on-a-string appearance of the primary nucleofilament. Concomitant with this structural change was a variation in the histone pattern and sedimentation behavior of the complexes: with 0.15 M NaCl, the isolated complexes contained both the nucleosomal histones and histone H1, and they sedimented in sucrose gradients at 70S. Increasing the ionic strength to 0.6 M NaCl resulted in the removal of histone H1 from the complexes and in a decrease of the sedimentation coefficient to 40S. DNA-relaxing enzyme was associated with the SV40 nucleoprotein complexes. The number of superhelical turns in DNA was identical in both types of complex. Therefore, the transition from the condensed to the open structure of viral chromatin does not require a change in the topological winding number of its DNA. (23 refs)

78-3339 **Nucleotide Sequence of the Simian Virus 40 *Hind*-K Restriction Fragment.** (Eng) Rogiers, R. (Laboratorium voor Moleculaire Biologie, Rijksuniversiteit Gent, Ledeganckstraat, 35, B-9000 Ghent, Belgium); Van de Voorde, A.; Soeda, E.; Fiers, W. *Eur J Biochem* 85(1): 205-224; 1978.

The nucleotide sequence of the restriction fragment *Hind*-K, which represents 4.2% of the simian virus 40 (SV40) genome and is located near the middle of the late region, is reported. The sequence was established by analysis of transcription products, synthesized by using *Escherichia coli* RNA polymerase and nucleoside triphosphates, one of which was (α-^{32}P)-labeled. Strand assignment was possible by hybridization of asymmetric, labeled transcripts of total SV40 DNA to filter-bound *Hind*-K fragments. The sequence was confirmed by using direct DNA-sequencing methods. The *Hind*-K sequence is 219 base pairs long. The message strand is particularly rich in adenosine (39%) and purines. The nucleotide sequence could unambiguously be translated into an amino acid sequence, and the N-terminal codon of the viral protein VP1 could be identified. The amino-terminal part of VP1 is rich in proline and lysine. The nucleotide sequence of *Hind*-K also codes for the carboxyl-terminal part of the viral protein VP2 and VP3 genes, which partly overlap the VP1 gene. (52 refs)

78-3340 **Nucleotide Sequence of Part of the Simian Virus 40 *Hind*-D Restriction Fragment: The Presumed Initiation Region of the VP2 Gene.** (Eng) Ysebaert, M. (Laboratorium voor Moleculaire Biologie, Fakulteit der Wetenschappen, Rijksuniversiteit te Gent, K. Lod Ledeganckstraat 35, B-9000 Ghent, Belgium); Van Heuverswyn, H.; Van de Voorde, A.; Fiers, W. *Eur J Biochem* 85(1): 195-204; 1978.

The nucleotide sequence at the beginning of the restriction fragment *Hind*-D, from simian virus 40 (SV40) DNA was derived by partial chemical degradation of (5′-^{32}P)-labeled restriction fragments followed by analysis on polyacrylamide gel. The *Hind/Hae* fragment, a product of double digestion, corresponds to 2.65% of the SV40 genome and is adjacent to the previously published *Hind*-L and *Hind*-M segments. The sequence is 140 nucleotides long, and it contains an ATG codon that probably represents the initiation codon of the VP2 protein. This codon is preceded by an untranslated region that is characterized by an alternation of (dA+dT)-rich and (dG+dC)-rich blocks. It seems likely that these remarkable alternations have an important, but as yet unknown, biological meaning. (37 refs)

78-3341 **The Structure of a Cloned Substituted SV40 Genome.** (Eng) Oren, M. (Dept. Virology, Weizmann Inst. Science, Rehovot, Israel); Lavi, S.; Winocour, E. *Virology* 85(2): 404-421; 1978.

The structure of the DNA of a cloned host-substituted simian virus 40 (SV40) variant was investigated by restriction endonuclease cleavage mapping and hybridization of the cleavage products to monkey and wild-type SV40 DNA's. The circular DNA of this variant comprised four tandemly repeated segments, each of which consisted of covalently linked viral and monkey DNA sequences. Only two of the repeated subunits were of equal size. A detailed cleavage map of the variant genome is presented. The viral sequences in each of the four subunits were derived from the portion of the wild-type SV40 genome (0.59-0.73 map unit) that contains the origin of replication. Monkey DNA sequences of the reiterated type were linked to monkey DNA sequences of the less-reiterated or nonreiterated type in three subunits. The monkey DNA sequences in the fourth subunit were predominantly of the reiterated type. Hybridization experiments to clarify the relationship between the host DNA sequences in the cloned variant and those in other independently derived substituted SV40 populations indicated that some populations share both reiterated and nonreiterated host DNA sequences, whereas other populations share only the reiterated sequences. (35 refs)

78-3342 **Use of Defective SV40 Replicons for the Propagation of Prokaryotic DNA in Mammalian Cells.** (Eng) Fareed, G. C. (Dept. Microbiology and Immunology, Molecular Biology Inst., Univ. California at Los Angeles, Los Angeles, CA, 90024); Upcroft, P. *Miami Winter Symp* 14: 49-71; 1977.

The use of simian virus 40 (SV40) as vector for the propagation of specific prokaryotic DNA segments in mammalian cells is discussed. In one experiment, segments of 520 and 2,400 base pairs from the immunity region of bacteriophage λ were linked in vitro to SV40 vectors and propagated in monkey cells in the presence of added helper, wild-type SV40 DNA. In both recombinant genomes, the structure of λ DNA segment was largely unchanged after serial passage in monkey cells. In another experiment, an *Escherichia coli* DNA segment was inserted after *Eco*RI and *Hpa*II cleavages, into a deletion in the late gene region of SV40. It was propagated with the aid of a helper early gene SV40 mutant. This purified chimeric DNA, which contained the SV40 genetic information needed for cellular transformation and for viral DNA replication, was used to transform rat embryo cells and to transfect monkey kidney cells. Preliminary findings on the structure of the viral and bacterial DNA sequences in these cell lines are summarized. (16 refs)

78-3343 **Interaction of SV40 T Antigen with Selected Regions of SV40 DNA.** (Eng) Livingston, D. M. (Sidney Farber Cancer Inst., Boston, MA); Tenen, D. G.; Jessel, D.; Haines, L. L.; Woodard, V.; Modest, A. P.; Maxam, A.; Hudson, J. *Miami Winter Symp* 14: 103-119; 1977.

Simian virus 40 (SV40) tumor (T) antigen was isolated from the SV40-transformed human cell line SV80 and examined for binding to SV40 DNA. The antigen was extracted from purified nuclei of mass cultures and purified by fractionation, chromatography, and glycerol gradient sedimentation. Electrophoresis demonstrated a species migrating with a mol wt of approx 90,000 daltons. Binding studies indicated that the antigen bound to virtually all molecules in a fixed aliquot of labeled SV40 DNA I. The question of whether T antigen binds selectively to one or more specific regions of the SV40 genome was investigated using restriction endonuclease fragments of the genome. Preferred T-antigen binding sites were noted in regions identified by *Hin* d(II + III) fragments A, B, and C. Repetition of these experiments using T antigen isolated from SV40-transformed Chinese hamster lung cells gave similar results. Thus, the binding phenomena are not specific to T antigen from a given SV40-transformed cell line. The interaction of T antigen with one or more sets of sequences within the SV40 early region might reflect a role for this protein in transcription. (38 refs)

78-3344 **Isolation and Characterization of SV40 T Antigen.** (Rus) Drise, O. B. (Cancer Res. Center, Acad. Medical Sciences USSR, Moscow, USSR); Voskoboinik, A. D.; Polotskaya, A. V.; Shlyankevich, M. A.; Shapot, V. S. *Biokhimiia* 42(8): 1471-1477; 1977.

The purification and isolation of functionally active simian virus 40 (SV40) T antigen are presented. SV40-induced hamster tumors were homogenized and subjected to several purification procedures. Precipitation with ammonium sulfate resulted in 5-fold purification, diethylaminoethylcellulose chromatography in 60-fold purification, electrophoresis in polyacrylamide gel in 460-fold purification, and immunoadsorption of foreign proteins with immunoadsorption of foreign proteins with immunochemical filters in 1,300- to 1,600-fold purification. Upon electrophoresis, T antigen was separated into three fractions with mol wts of 100,000, 200,000, and 400,000, of which only the 200,000 mol wt fraction showed endonuclease activity. (25 refs.)

78-3345 **Antigenic Similarity Between Extracts of Fetal Cells and Simian Virus 40 Tumor Specific Surface Antigens (Meeting Abstract).** (Eng) Weppner, W. A. (Univ. South Alabama, Mobile, AL, 36688); Coggin, J. H. *Fed Proc* 37(6): 1561; 1978. (1 ref)

78-3346 **Amino Acid Sequence Homology Between Polyoma and SV40 Tumour Antigens Deduced from Nucleotide Sequences.** (Eng) Friedmann, T. (Dept. Pediatrics, Sch. Medicine, Univ. California at San Diego, La Jolla, CA, 92093); Doolittle, R. F.; Walter, G. *Nature* 274(5668): 291-293; 1978.

Evidence for amino acid sequence homology between polyoma and simian virus 40 (SV40) tumor antigens was obtained from a study of the nucleotide sequences in the viral DNA's. The deduced amino acid sequences for both polyoma and SV40 tumor antigens were identical in 49/174 positions, the most striking homologies occurring from amino acids 1 to 58 and from amino acids 119 to 159. Eight of the 11 cysteine residues in polyoma virus could be aligned with 8/11 SV40 cysteine residues. The major homology domain from amino acids 1 to 58 was located in the region common to T (mol wt 100,000) and t (mol wt 20,000) antigens (0.65 to 0.60 map units). The second domain from amino acids 119 to 159 was located in the coding region unique for t antigen (0.60 to 0.55 map units). The latter region contained the highly conserved cysteine clusters. The sequence Cys X Cys X X Cys, found in both viral DNA's, also occurs in the α and β subunits of all members of the glycoprotein hormone family, in two immunoglobulin γ-chains, and in one kind of keratin. Another sequence in the Bowman-Birk soybean trypsin inhibitor closely resembles sequences in SV40 t antigen and polyoma t antigen. The data suggest that SV40 and polyoma virus have a common evolutionary origin and that the tertiary structures of their t and T antigens are similar. (20 refs)

78-3347 **Construction of a Viable SV40 Variant Containing Two Functional Origins of DNA Replication.** (Eng) Shenk, T. (Dept. Microbiology, Univ. Connecticut Health Center, Farmington, CT, 06032). *Cell* 13(4): 791-798; 1978.

Viable insertion variants of simian virus 40 (SV40) that contained two functional origins of DNA replication (Or) were constructed. The variants were made by introducing, at 0.175 on the SV40 map, a segment of DNA containing the viral Or. (The region near 0.67 map unit on the SV40 genome contains the original viral Or.) Pulse-labeling and electron microscopy demonstrated that the second Or was functional. The distribution of radioactivity in pulse-labeled closed-circular SV40 (I) DNA was dramatically altered in the variants compared with the parental virus. Electron microscopic examination of viral replicative intermediates indicated that there was one initiation site for DNA synthesis in the parental genome but two in the variant. A deletion could be introduced in the variant that inactivated the original Or. The resulting mutant could still be propagated, and its DNA replication originated at the site of the newly inserted Or. Therefore, the viral genome can tolerate the addition of new nucleotide segments at 0.175 map unit. Other small DNA segments could presumably be inserted at this site and propagated as part of a fully viable genome. (25 refs)

78-3348 **Structural Studies on Defective SV40 Variants Substituted with Monkey DNA Sequences (Meeting Abstract).** (Eng) Wakamiya, T. (Lab. Molecular Biology and Biochemistry, NCI, NIH, Bethesda, MD, 20014); McCutchan, T.; Singer, M.; Rosenberg, M. *Fed Proc* 37(6): 1736; 1978. (no refs)

78-3349 **Construction and Biological Activities of Deletion Mutants of Simian Virus 40.** (Eng) Nathans, D. (Dept. Microbiology, Johns Hopkins Univ. Sch. Medicine, Baltimore, MD, 21205); Lai, C. J.; Scott, W. A.; Brockman, W. W.; Adler, S. P. *Miami Winter Symp* 14: 1-10; 1977.

Experiments on the properties of mutants containing deletions of either the early or late region of the simian virus 40 (SV40) genome are summarized. SV40 deletion mutants were cloned from serially passed virus stocks or constructed by enzymatic manipulation of the DNA. Some of the mutants produced detectable short polypeptides derived from SV40-coded proteins. Mutants in the late region were used to map the junctions between the D and B/C genes. The results suggest that the D gene may overlap with the B/C gene and that, within the *Hin*-E segment preceding the B/C gene proper, there is a signal required for expression of the B/C gene. Late region mutants were also used to define SV40 functions that require only the early or A gene. Mutants lacking portions of both late genes but with an intact early genomic segment were able to induce T antigen in infected cells, replicate their DNA in the absence of helper virus, stimulate thymidine incorporation in cellular DNA, and transform mouse and hamster cells. A cell fusion-complementation rescue procedure was used to show that cells transformed by late deletion mutants contained the mutant genome. Deletion mutants lacking substantial portions of the early genomic region, including those segments where temperature-sensitive A mutants map, lacked all the above activities. (14 refs)

78-3350 **Properties of Simian Virus 40 and BK Virus Tumor Antigens from Productively Infected and Transformed Cells.** (Eng) Simmons, D. T. (Lab. Biology Viruses, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD, 20014); Takemoto, K. K.; Martin, M. A. *Virology* 85(1): 137-145; 1978.

Simian virus 40 (SV40)-specific tumor antigen (T Ag) and BK virus (BKV) T Ag immunoprecipitated from lytically infected and transformed cells were studied. T Ag from SV40-transformed hamster cells had the same electrophoretic mobility in acrylamide gel as the 97,000 (97K)-dalton T Ag isolated from SV40-infected monkey cells. Smaller mol wt forms of SV40 T Ag (76K-90K) were also readily isolated by immunoprecipitation from extracts of infected monkey cells. Experiments using the protease inhibitor L-l-tosylmide-2-phenylethylchloromethyl ketone (TPCK) indicated that the smaller species of SV40 T Ag were derived from the 97K or 94K protein by proteolytic cleavage during their purification from the infected cells. SV40 T Ag could be immunoprecipitated in the presence of either anti-SV40 T serum or anti-BKV T serum, and the various mol wt forms of SV40 T Ag observed with anti-SV40 T serum were also precipitated with anti-BKV T serum. The synthesis of BKV T Ag occurred at significantly later times after infection than did the corresponding SV40 protein. The largest species of labeled SV40 T Ag (97K) comigrated with the largest BKV T Ag in acrylamide gels, as did the 94K protein form of each viral T Ag. The data suggest that the proteins with an apparent mol wt of 97K were the monomer forms of the T Ag in the SV40-infected or -transformed cells and BKV-infected human cells. (24 refs)

78-3351 **Studies of Adenovirus and SV40 Genes Required for In Vitro Transformation.** (Eng) van der Eb, A. J. (Sylvius Labs., Univ. Leiden, Leiden, Netherlands); Lupker, J. H.; Maat, J.; Abrahams, P.; Jochemsen, H.; Houweling, A.; Fiers, W.; Mulder, C.; van Ormondt, H.; de Waard, A. *Miami Winter Symp* 14: 121-137; 1977.

The transforming ability of various adenovirus and simian virus 40 (SV40) restriction fragments was investigated. With adenovirus 2 (Ad2) and Ad5 DNA, fragments with transforming activity were obtained with the restriction endonucleases *Eco* RI, *Bam*HI, and *Hsu* I; these fragments represent the left-terminal fragments of both viral DNA's. Rat cells transformed by *Eco* RI and *Bam* fragments had T-antigen fluorescent patterns similar to those of cells transformed by intact virus. However, cells transformed by the 7% *Hsu* I G fragment of Ad5 DNA had an abnormal distribution T antigen. Examination of five clones of *Hsu* I G transformed rat cells indicated these cells contained viral DNA sequences homologous to 75%-85% of this fragment. Thus, a segment as small as 5.5%-6% of the viral genome contains information for the maintenance of transformation. The proteins coded for by this section of the genome were identified. RNA selected by hybridization to total Ad5 DNA was translated into at least seven polypeptides and five of these [51,000 (51K), 48K, 42K, 38K, and 14K] were encoded by the 7% *Hsu* I G fragment. Further experiments indicated that *Hpa* I fragment E, which contains the left terminal 4.3% of Ad5 DNA, also had transforming activity. Studies of Ad12, Ad3, and Ad7 DNA indicated that a terminal fragment of Ad3 and Ad7, obtained with endonuclease R. *Sma* I, and an *Eco* RI C fragment from Ad12 had transforming activity. The latter fragment also induced tumors upon its injection into newborn hamsters. Studies of SV40 DNA indicated that the 74% *Eco* RI/*Hap* II A, the 59% *Bam* HI/*Hap* II A, endo R. *Eco* RI, *Bgl* I, and *Eco* RI/Taq I A fragments contained transforming activity. (16 refs)

78-3352 **SV40-Adenovirus 2 Hybrid Viruses.** (Eng) Sambrook, J. (Cold Spring Harbor Lab., P.O. Box 100, Cold Spring Harbor, NY, 11524). *Miami Winter Symp* 14: 139-179; 1977.

The isolation of hybrid viruses whose defective 5- to 6-kilobase genomes contain both simian virus 40 DNA and adenovirus 2 (Ad2) DNA is described. Cellular recombination enzymes were used during the construction of these hybrids. The structure of arrangement of these genomes is complex, but in the simplest case, restriction endonuclease digestion and hybridization indicated that the Ad2 DNA was present as a continuous block with a max size of 2.8 kilobases. Different hybrids contained sequences derived from different segments of the Ad2 genome. (33 refs)

78-3353 **Starting Points of Adenovirus DNA Replication (Meeting Abstract).** (Ger) Winnacker, E. L. (Inst. fur Biochemie, Univ. Munchen, Karlstrasse 23, D-8000 Munich 2, W. Germany) Reiter, T.; Weingartner, B. *Hoppe Seylers Z Physiol Chem* 359(3): 339; 1978. (1 ref)

78-3354 **Transfer RNA Methylation in Adenovirus 2 Infected KB Cells (Meeting Abstract).** (Eng) Varricchio, F. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Sehulster, L.; Raska, K. *Fed Proc* 37(6): 1733; 1978. (no refs)

78-3355 **Intracellular Forms of Adenovirus DNA. VII. Excision of Viral Sequences from Cellular DNA in Adenovirus Type 2-infected KB Cells.** (Eng) Baczko, K. (NIH, Bethesda, MD, 20014); Neumann, R.; Doerfler, W. *Virology* 85(2): 557-567; 1978.

Viral sequences were excised from the high-mol-wt DNA of adenovirus type 2 (Ad2)-infected KB cells with three restriction endonucleases of different specificities, *Eco* RI, *Bam* HI, and *Sal* I. Analysis of the restriction endonuclease fragments by gel electrophoresis and DNA-DNA hybridization revealed viral DNA sequences that did not coincide in size with the respective Ad2 virion DNA restriction endonuclease fragments. After cleavage with *Bam* HI or *Sal* I, some viral sequences found in the fragments were larger than the largest virion DNA fragments. Of the viral DNA sequences found after cleavage with *Eco* RI, none was larger than the *Eco* RI A fragment with a mol wt of 13 x 10^6 daltons, which indicated that intact Ad2 DNA does not have a simple oligomeric structure. These results suggest that Ad2 DNA is integrated into cellular DNA in fragments rather than as the intact molecule. This integration pattern is consistent with previous observations that the right end of Ad2 DNA is overrepresented in the 50S-90S class of viral DNA. (19 refs)

78-3356 **Two Regions of the Adenovirus 2 Genome Specify Families of Late Polysomal RNAs Containing Common Sequences.** (Eng) McGrogan, M. (Dept. Pathology, Div. Biology and Biomedical Sciences, Washington Univ. Sch. Medicine, St. Louis, MO, 63110); Raskas, H. J. *Proc Natl Acad Sci USA* 75(2): 625-629; 1978.

Late cytoplasmic RNA's specified by two regions of the adenovirus 2 genome (39.3-51.8 and 70.7-83.4 map units) were analyzed by size fractionation of poly(A)-polysomal ^{3}H-RNA and subsequent hybridization to DNA fragments. Both regions encoded RNA's whose sequence content exceeded the coding capacity of the region. These multiple transcripts are likely to function as messenger RNA's (mRNAs), because they were present on polyribosomes. The messenger DNA segment 39.3-51.8 specified 27S, 22S, and 18S RNA's. The genome sites specifying these three size classes were determined by hybridizations with seven different DNA fragments from this genome region. The 3′ termini of all three size classes were specified by sequences near a common site, position 50.1. The 27S RNA included sequences beginning near 39.3, the 22S RNA sequences from 41.0, and the 18S RNA sequences from approx 45.3 on the unit genome. A second family of four RNA's was transcribed from 70.7-83.4. These 28S, 22S, 18S, and 16S RNA's have a relationship similar to the RNA's transcribed from 39.3-51.8. Sequences near the 5′ ends of these four size classes were specified by different genome sites. However, the 3′ termini of all four classes were localized near map position 80.4. The synthesis of families of RNA may allow the translation of multiple polypeptides from a genome segment that has only one terminator site for mRNA synthesis. (23 refs)

78-3357 **Mapping of Adenovirus Late Promoters with Nascent Mercurated RNA.** (Eng) Weinmann, R. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA, 19104); Aiello, L. O. *Proc Natl Acad Sci USA* 75(4): 1662-1666; 1978.

Nascent RNA molecules were labeled in vivo and elongated in vitro by incubation of the isolated nuclei in the presence of mercurated nucleotides. The RNA molecules initiated and labeled in vivo and elongated in vitro were then selectively purified on a thiopropyl 6-B Sepharose affinity column. The procedure was free of artifacts, since the addition of mercurated nucleotides and retention of the elongated RNAs on the affinity column were mediated by the endogenous RNA polymerase II (nucleoside triphosphate:RNA nucleotidyltransferase; artificial aggregates did not form), sensitive to actinomycin D, and dependent on the presence of all four ribonucleotide triphosphates. This general procedure was applied to the mapping of viral promoters late after adenovirus 2 infection of HeLa cells. RNA purified as described was hybridized to restriction enzyme fragments attached to nitrocellulose filters. The 5′ ends of the nascent RNA chains were located in coordinates 9.5-17 for a rightward transcript, 0-25 for a leftward transcript, and possibly 60-70 for a second rightward transcript. These locations differed clearly from those of the early promoters, suggesting that the transition from early to late functions is controlled at the transcriptional level. (38 refs)

78-3358 **Adenovirus Type 12 Tumor Antigen. I. Separation from DNA Polymerase α and Immunoprecipitation of Tumor-Antigen Polypeptides.** (Eng) Biron, K. K. (Cancer Res. Center, Univ. North Carolina, Chapel Hill, NC, 27514); Morrongiello, M. P.; Raskova, J.; Raska, K. *Virology* 85(2): 464-474; 1978.

Adenovirus type 12 (Ad12) tumor (T) antigen was extracted from Ad12-transformed baby hamster kidney BHK21 cells and partially purified (> 500-fold) by chromatography on double-stranded DNA-cellulose. The antigen contained DNA polymerase α activity that was partially inhibited by antibodies in immune ascitic fluid. The DNA polymerase and T antigen could be completely separated by chromatography on single-stranded DNA-cellulose, to which T antigen did not bind. Antibodies reacting with T antigen had no inhibitory effect on the isolated DNA polymerase. The fraction eluting in the void volume of a single-stranded DNA-cellulose column containing > 1200-fold-purified T antigen markedly stimulated the activity of DNA polymerase α with the native DNA template; with the activated DNA template, the stimulation was less prominent. This stimulation was not significantly affected by T-antigen-reactive antibodies. An analogous fraction of control BHK21 cells had similar stimulatory effects on the isolated DNA polymerase α, indicating that the stimulatory activity is probably not attributable to T antigen. Of the T-antigen polypeptides immunoprecipitated from the > 1200-fold-purified preparations by antibodies from tumor-bearing hamsters, one was identified as a major peptide of mol wt 50,000, the other as a minor species of mol wt 11,000. (35 refs)

78-3359 **Deletions and Insertions in Adenovirus Type 12 DNA after Viral Replication in Vero Cells.** (Eng) Werner, G. (Institut fur Klinische Virologie, Universitat Erlangen-Nurnberg, Loschgestrasse 7, 852 Erlangen, W. Germany); Zur Hausen, H. *Virology* 86(1): 66-77; 1978.

The adaptation of adenovirus type 12 (Ad12; Huie strain) to growth in Vero cells is reported. Infection of the cells was achieved at low input multiplicities; the cells were negative for the expression of simian virus 40 and BK virus T antigens. In contrast to wild-type virus, the adapted virus formed plaques in Vero cells and replicated in primary African green monkey kidney cells. The DNA of the adapted virus was analyzed by heteroduplex mapping, contour length measurement, and cleavage by the restriction endonucleases Bam HI, Eco RI, and Sma I. The data indicated that replication of Ad12 in Vero cells resulted in the accumulation of at least five groups of deletion mutants containing deletions between 0.5 and 1.5 x 10^6 in mol wt. Plaque purification of the same virus preparations in Vero cells resulted in the loss of most of the deleted molecules and permitted the identification of DNA insertions in fragment E of the Bam HI digest. These insertions ranged between 1.2 and 1.8 x 10^5 in mol wt and appeared to represent host cell-specific sequences. (32 refs)

78-3360 **Radiometric Detection of Herpes Simplex Viruses.** (Eng) D'Antonio, N. L. (Johns Hopkins Medical Insts., Baltimore, MD); Tsan, M. F.; Griffin, D. E.; Charache, P. A.; Wagner, H. N. *J Nucl Med* 19(2): 185-190; 1978.

Two radiometric procedures for the detection of herpes simplex virus (HSV) infections were developed in which ^{3}H-thymidine or ^{125}I-iododeoxyuridine is used. Cells infected with HSV-1 or -2 showed increased thymidine incorporation after infection, as did mouse brains infected with HSV-1. Similar results were observed with iododeoxyuridine. This increased incorporation could be inhibited by preneutralization of the virions with immune serum. (22 refs)

78-3361 **Characteristics of the Fc Receptor Induced by Herpes Simplex Virus.** (Eng) Costa, J (Lab. Pathology, Div. Cancer Biology and Diagnosis, NCI, NIH, Bethesda, MD, 20014); Yee, C.; Nakamura, Y.; Rabson, A. *Intervirology* 10(1): 32-39; 1978.

The Fc receptor induced in various cell lines by herpes simplex virus type 1 (HSV-1) from human trigeminal ganglion and HSV- 2 (strain MS) was studied. The 7S early antigen rosette assay revealed that within 20 hr of infection, WI-38 diploid human fibroblasts, Molt lymphoblastoid cells, Y-79 human retinoblastoma cells, Vero African green monkey kidney cells, mouse BALB/c 3T3 cells, SIRC rabbit cornea cells, and LY mouse fibroblasts developed Fc binding sites. A study of the characteristics of the Fc receptor revealed the following: (1) its appearance required viral protein synthesis, but transcription from the input DNA appeared sufficient to generate the receptor; (2) replication of viral DNA was not necessary; (3) 2-deoxy-D-glucose inhibited the appearance of Fc-binding activity; (4) the receptor had a low turnover and was not rapidly shed from the cell surface; (5) the number of cells showing Fc-binding activity was not related to the cell cycle at the time of infection; (6) the receptor was trypsin-sensitive but neuraminidase-resistant; and (7) following trypsin digestion, cells blocked by cytosine arabinoside regenerated the binding activity. (15 refs)

78-3362 **Electrokinetic Alteration of the Surface of Herpes Simplex Virus Infected Cells.** (Eng) Thompson, C. J. (Dept. Microbiology and Cell Biology, S101 Frear Building, Pennsylvania State Univ., University Park, PA, 16802); Docherty, J. J.; Boltz, R. C.; Gaines, R. A.; Todd, P. *J Gen Virol* 39(3): 449-461; 1978.

Whole-cell electrophoresis and isoelectric focusing were used to investigate changes in the ionic composition of the cell surface induced by herpes simplex virus (HSV). Most of the experiments involved HSV type-1 infection of Wistar rat embryo cells. However, experiments were repeated at various times using HSV type-2 and other cell types to establish that a given observation was not unique to the virus or cell type. Cell electrophoretic mobility (a measurement of negative surface charge density) declined concomitantly with the appearance of newly synthesized infectious virus at 6, 9, and 12 hr postinfection. The max decrement in surface charge density following HSV infection of Wistar rat embryo cells was 19.4%. Cell isoelectric focusing pH increased, indicating that the electrokinetic cell surface became ionically more positive. The decline in surface charge density is consistent with the hypothesis that basic amino groups are being introduced to the cell surface. This hypothesis was further tested by reacting cells with the amino-specific reagent 4-acetamide-4′-isothiocyanostilbene-2,2′-disulfonic acid. Infected cells had 2.4 times as many surface charges affected by the amino-specific reagent as did uninfected control cells. A relationship between these virus-induced amino groups and HSV surface antigens was indicated by the similar kinetics of their appearance and electrophoretic neutralization by HSV antibodies. It is suggested that HSV infection adds positive ionogenic groups to the cell surface. (38 refs)

78-3363 **Interference by Herpes Simplex Virus of Tumorigenesis by Murine Sarcoma Virus.** (Eng) Reilly, C. A. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL, 60439); Dale, P. J.; Gutzeit, D. L.; Finkel, M. P. *In: Advances in Comparative Leukemia Research 1977. Proceedings on the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 228-229; 1978.

UV-inactivated herpes simplex virus types 1 and 2 (HSV-1 and HSV-2) were injected into newborn CF#1/Anl mice to determine the effect on FBJ virus-induced osteosarcomas. HSV was injected either 24 hr before, 24 hr after, or at the same time as FBJ virus. There were no significant effects on FBJ-induced tumorigenesis when HSV was given at the same time as or 24 hr after FBJ infection. However, when HSV infection preceded FBJ virus infection, a significant reduction in osteosarcomas was noted. After 1 yr, 54% of the mice pretreated with HSV-1 and 83% of those pretreated with HSV-2 developed osteosarcomas compared with 100% of those treated with FBJ only. When mice pretreated with HSV-1 did develop osteosarcomas, the mean time to death was almost doubled and the mean number of tumors/mouse was halved. With HSV-2-pretreated mice that developed osteosarcomas, the mean life-span was almost quadrupled and the number of tumors/mouse was cut by 40%. No specific pathology appeared in mice treated with HSV only. These results indicate that HSV can interfere with FBJ virus tumorigenesis; the mechanism by which this is accomplished is not known. (1 ref)

78-3364 **Cleavage of Epstein-Barr Virus DNA by Restriction Endonucleases EcoRI, HindIII and BamI.** (Eng) Rymo, L. (Dept. Clinical Chemistry, Univ. Gothenberg, Sahlgren's Hosp., S-413 45 Gothenburg, Sweden); Forsblom, S. *Nucleic Acids Res* 5(4): 1387-1402; 1978.

Cleavage of the DNA's of the B95-8 and P3HR-1 strains of Epstein-Barr virus (EBV) by the restriction endonucleases *Eco*RI, *Hin*dIII, and *Bam*I was investigated using a new technique for quantitative evaluation of the fluorescence of ethidium-stained DNA fragments separated on agarose gels. Most B95-8 DNA fragments produced by *Eco*RI and *Hin*dIII cleavage appeared to be present in one copy per DNA molecule. Digestion of *Bam*I yielded a larger number of fragments, including one with a molecular mass of 2.0 megadaltons that was present in 11 copies and localized to a limited part of the DNA molecule. The same sequences were also present in the P3HR-1 DNA, although in a lower molar ratio. P3HR-1 DNA yielded restriction enzyme cleavage patterns suggesting DNA sequence heterogeneity of P3HR-1 virus. No fragment was present in more than about four copies per molecule of P3HR-1 DNA. Comparison of the restriction enzyme cleavage patterns of P3HR-1 and B95-8 DNA revealed a high degree of structural homology emphasized by nucleic acid hybridization experiments with EBV complementary RNA synthesized in vitro. (22 refs)

78-3365 **Detection of the EBV-determined Nuclear Antigen (EBNA) in Burkitt's Lymphoma and Nasopharyngeal Carcinoma Biopsies by the Acid Fixed Nuclear Binding (AFNB) Technique.** (Eng) Luka, J. (Dept. Tumor Biology, Karolinska Institutet, S 104 01 Stockholm 60, Sweden); Klein, G.; Henle, W.; Henle, G. *Cancer Lett* 4(4): 199-205; 1978.

Epstein-Barr virus (EBV)-carrying biopsy specimens of Burkitt's lymphoma (BL) and nasopharyngeal carcinoma (NPC) patients were used to determine whether EBV-associated nuclear antigen (EBNA) can be demonstrated in these tissues by the acid-fixed nuclear binding (AFNB) technique. Extracts of five BL and five NPC specimens gave a brilliant, EBNA-specific fluorescence after binding to acid-fixed chicken RBC. All BL and NPC extracts gave strong positive nuclear fluorescence with anti-EBNA-positive sera, but not with anti-EBNA-negative sera. Similar extracts of three other African tumors that are not known to carry the EBV genome were negative, even though they were derived from EBV-seropositive patients with relatively high anti-EBV viral capsid antigen antibody titers. In tests with BL and NPC-derived material, the anticomplement immunofluorescence reaction was equally strong with both crude tumor extracts and DNA-cellulose-purified extracts, provided the reaction was performed at 4 C. Incubation of the extracts at 37 C led to a more rapid breakdown of the EBNA in the crude material than in the DNA-cellulose concentrated material. These findings indicate that AFNB can be used to detect EBNA in biopsy specimens. (15 refs)

78-3366 **Epstein-Barr Virus-determined Nuclear Antigen in Malignant Epithelial Cells of Nasopharyngeal Carcinoma.** (Eng) Suzuki, T. (First Dept. Pathology, Niigata Univ. Sch. Medicine, 1-Bancho, Asahimachi-dori, Niigata 951, Japan); Katano, A.; Oboshi, S.; Sairenji, T.; Hinuma, Y. *Gann* 69(1): 133-138; 1978.

A test for the presence of Epstein-Barr nuclear antigen (EBNA) in the malignant epithelial cells of a human nasopharyngeal carcinoma was made by first transplanting tumor tissue in nude mice. Tumor tissue was removed from a lymph node metastasis in a 56-yr-old woman and transplanted sc into two nude BALB/c mice. Optical and electron microscopy indicated that the tumors grown in the mice were identical to the one removed from the woman. Tumor cells from the second passage were dispersed in vitro, and EBNA was detected in smears of the malignant epithelial cells. EBNA was not detected, however, in tumor cell imprints made at passage 1, probably because of inadequate preservation. (10 refs)

78-3367 **Detection of Epstein-Barr Virus (EBV) DNA Sequences Using In Situ Hybridization.** (Eng) Moar, M. H. (Dept. Tumor Biology, Karolinska Inst., 10401 Stockholm 60, Sweden); Klein, G. *Biochim Biophys Acta* 519(1): 49-64; 1978.

Epstein-Barr virus complementary RNA (EBV cRNA) hybridization in situ was used to detect EBV DNA sequences under conditions in which the virus DNA is replicating spontaneously and under conditions in which it is induced to do so following superinfection. Optimum conditions for the in situ detection of EBV DNA in single cells were determined. Preliminary experiments using three Burkitt's lymphoma-derived cell lines (the producer P3HR-1 line, the nonproducer Raji line, and the EBV DNA-negative line BJAB) showed that the hybridization is specific and potentially highly sensitive. Therefore, P3HR-1 cells were used in most of the remaining experiments. The most effective fixatives were methanol/acetic acid (3:1) or methanol/acetone (1:2); and NaOH (0.05 M) was far superior to HCl in denaturing the EBV DNA, which has a high (58%) guanosine + cytosine content. Additional optimal conditions were a temperature of 50 C, a cRNA concentration of 3 nanograms/ml, and a hybridization time of 12 hr. Pretreatment of the cells with RNase did not reduce the percentage of labeled cells or the av number of grain counts/cell, but DNase abolished the autoradiographic reaction completely. ^{3}H-DNA was also an effective probe for the detection of specific DNA sequences in single cells. In experiments with producer, nonproducer, and EBV DNA-negative cells, the percentage of cells positive for EBV DNA correlated with the percentage expressing early antigen. It is concluded that under appropriate conditions, EBV DNA sequences can be detected specifically at the single cell level with a high degree of efficiency. (42 refs)

78-3368 **Lymphotropic Herpesviruses in Hodgkin's Disease.** (Eng) Napel, C. (Clinical Immunology Unit, Dept. Medicine, Univ. Groningen, Groningen, Netherlands); Bijker, J.; De Gast, G. C.; Halie, M. R.; Langenhuysen, M. M.; The, T. H. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds.(New York: Elsevier/North Holland Biomedical Press): 503 pp.; 223-225; 1978.

The lymphocyte reactivity of 24 patients with Hodgkin's disease (HD) to Epstein-Barr virus (EBV) and cytomegalovirus (CMV) was determined and compared with that of age- and sex-matched controls. HD patients showed decreased EBV cellular immunity in association with elevated EBV-viral capsid antigen antibody titers. CMV cellular immunity was also decreased, especially in CMV-early antigen antibody seropositive patients. Lymphocyte reactivity to phytohemagglutinin, concanavalin A, and an antigen cocktail was lower in the patients, but reactivity to pokeweed mitogen was not impaired. In most cases, the general lymphocyte reactivity of the patients was in the normal range. It is suggested that high EBV- and CMV-antibody levels in HD are caused by defective virus specific cellular immunity early in the course of the disease, before disease-associated general depression of lymphocyte function has occurred. (9 refs)

78-3369 **A New Tumour-derived Transforming Strain of Epstein-Barr Virus.** (Eng) Pizzo, P. A. (Infectious Diseases Section, Pediatric Oncology Branch, NCI, Bethesda, MD, 20014); Magrath, I. T.; Chattopadhyay, S. K.; Biggar, R. J.; Gerber, P. *Nature* 272(5654): 629-631; 1978.

A new strain of Epstein-Barr virus (EBV) was isolated from

cell line (AG876) established from the splenic tumor of an
-yr-old Ghanaian boy with Burkitt's lymphoma. Almost all
he cells contain EB nuclear antigen (EBNA), and 5%-20%
nanifest the late viral product, cytoplasmic viral capsid anti-
en (VCA). The AG876 virus has only been propagated in
he cell line derived directly from this splenic tumor. Virus
urified from the AG876 culture medium transformed hu-
nan umbilical cord WBC but failed to induce early antigen
EA) in Raji cells. Purified AG876 DNA banded at a density
f 1.7180 g/cm^3 in a CsCl equilibrium gradient, which is
onsistent with previous analyses of EBV DNA. Association
inetic experiments revealed $\geq$ 90% hybridization of the la-
eled AG876 viral DNA probe with its respective producer
ost cell DNA, as well as with the cellular DNA's from
3HR and B95-8, two other EBV producer cell lines. There
vas virtually 100% homology in the kinetics of hybridization
f AG876 with P3HR1 and B95-8 DNA's, suggesting close
imilarity between the AG876 viral DNA and that of both
3HR and B95-8. AG876 virus seems to be a unique strain
f EBV that is more representative of a wild-type (or putative
tumor virus) strain of EBV than other available strains. (19 refs.)

See also:
*(Rev.): 78-3002, 78-3023, 78-3031, 78-3038, 78-3039, 78-3040, 78-3041, 78-3042, 78-3043, 78-3044, 78-3045, 78-3046, 78-3047, 78-3049, 78-3058.
*(Chem): 78-3193.
*(Phys.): 78-3257.
*(Immun.): 78-3372, 78-3373, 78-3374, 78-3376, 78-3377, 78-3378, 78-3380, 78-3381, 78-3382, 78-3388, 78-3389, 78-3391, 78-3395, 78-3398, 78-3399, 78-3404, 78-3406, 78-3412, 78-3413, 78-3418, 78-3419, 78-3424, 78-3425, 78-3431. 78-3432, 78-3441, 78-3451, 78-3457, 78-3458.
*(Path.): 78-3472, 78-3474, 78-3477, 78-3528.
*(Epid.-Biom.): 78-3579, 78-3584.

78-3370 **Aldehyde Dehydrogenase in 2-AAF-Induced Rat Hepatomas: Characterization of Antigens Recognized by Anti-Hepatoma Aldehyde Dehydrogenase Sera (Meeting Abstract).** (Eng) Lindahl, R. (Univ. of Alabama, University, AL, 35486); Hardman, J. K. *Fed Proc* 37(6):1797; 1978. (no refs)

78-3371 **Heterogeneity of KCl-solubilized Antigens of Chemically Induced Sarcomas (Immunology).** (Eng) Blackstock, R. (Dept. Pediatrics, Hematology-Oncology Service, Univ. Oklahoma, Oklahoma City, OK, 73190); Schimpff, R. D.; Smith, R. T. *Proc Soc Exp Biol Med* 157(3): 354-357; 1978.

KCl-solubilized tumor antigens from female C57BL/6J mice bearing methylcholanthrene-induced (0.5 mg, im) fibrosarcomas were isolated and characterized. Material with both a high ($>$300,000 daltons) and low ($<$30,000 daltons) mol wt inhibited ^{3}H-thymidine incorporation by lymphoid cells taken from tumor-bearing animals. When the inhibitor was removed, however, the remaining material stimulated proliferation in these lymphoid cells over a wide range of mol wts ($<$30,000 to $>$100,000 daltons). (18 refs)

78-3372 **Changes in Levels of H-2 Antigens after RADLV Infection Affect Cellular Immune Responses Involved in Leukemia Resistance (Meeting Abstract).** (Eng) Meruelo, D. (New York University Medical Center, New York, NY, 10016). *Fed Proc* 37(6): 1569; 1978. (no refs)

78-3373 **The Effect of Paul-Bunnell Antigen from Human Malignant Spleens on Polycythemic Friend Virus in BALB/C Mice (Meeting Abstract).** (Eng) Fjelde, A. (Roswell Park Memorial Inst., Buffalo, NY, 14263); Evege, E. *Proc Am Assoc Cancer Res* 19: 146; 1978. (2 refs)

78-3374 **Intracellular and Extracellular Distribution of Antigens from SV40 Transformed Cells (Meeting Abstract).** (Eng) Luborsky, S. W. (NIH, NCI, Bethesda, MD, 20014); Chang, C.; Robinson, C. L.; Mora, P. T. *F*
Proc 37(6): 1561; 1978. (no refs)

78-3375 **Serological Dissection of a Chicken Onco-de**
lopmental Antigen. (Eng) Dietert, R. R. (De
Zoology, Univ. Texas, Austin, TX, 78712); Sanders, B.
Dev Comp Immunol 2(1): 181-184; 1978.

A RBC oncodevelopmental antigen was serologically dissec
ed into eight antigenic determinants. Rabbit antisera pr
pared against RBC from 1-day-old chicks and adsorbed wi
RBC from adult chickens of identical strains were used
detect the presence of antigenic determinants on RBC of se
eral avian species. On the basis of differential adsorptions ar
hemagglutination reactions, eight different antigenic determ
nants were recognized. The expression of these determinan
was compared on RBC of differently aged birds. There w
a loss (or addition) of antigenic determinants with aging. Th
RBC membrane antigens were of phylogenetic interest b
cause of their differential expression among avian specie
RBC from adult and embryonic turkeys expressed only ant
genic determinant 1, whereas RBC from chicken, pheasan
and Japanese quail embryos, which all belong to the sam
family, expressed 1-8, 1-5, and 1-4 antigenic determinant
respectively. Differential expression of the antigenic determ
nants with aging and species of bird should permit studie
of the expression of antigenic determinants with developme
and malignancy. (14 refs)

78-3376 **A Virus-Induced Transformation Specific An**
gen at the Surface of Rous Sarcoma Vir
Transformed Fibroblasts (Meeting Abstract). (Eng) Prat,
(Univ. Torino, 10126 Torino, Italy); Bertini, M.; Comogli
P. M. *Fed Proc* 37(6): 1562; 1978. (no refs)

78-3377 **Effect of Host Immunity on Tumor Cell An**
genic Variability (Meeting Abstract). (En
Pearse, A. (Dept. Pathology, Lenox Hill Hosp., New Yo
NY, 10021); Ioachim, H. L. *Proc Am Assoc Cancer Res*
175; 1978. (no refs)

78-3378 **Immunochemical Characterization of Tum**
Associated Surface Antigens (TASA) On a M

oney Murine Leukemia Virus Induced Lymphoma, MBL-2 (Meeting Abstract).** (Eng) Ng, A. (NCI, NIH, Bethesda, MD, 20014); McIntire, K. R.; Herberman, R. B. *Fed Proc* 7(6): 1563; 1978. (no refs)

8-3379 Effect of the Prolonged Immunization with Tumor-unrelated Antigen on the Tumor Growth. Eng) Targowski, S. P. (Dept. Microbiology, State Univ. New York at Buffalo, Sch. Medicine, Buffalo, NY, 14214); Albini, B.; Milgrom, F. *Proc Soc Exp Biol Med* 157(3): 511-516; 978.

The growth of a methylcholanthrene-induced sarcoma, MCSa-22, in C3H/HeHa mice was investigated after the mice had been immunized for 4-5 mo with bovine serum albumin (BSA) or ovalbumin (OA). A dose of 0.2 mg BSA or OA in 0.2 ml saline was administered iv or ip, respectively. The umor was injected when deposits of immune complexes could be demonstrated in the kidneys. BSA-immunized mice were given 10^4, 10^3, or 10^2 tumor cells. All immunized mice developed tumors within 8-17 days; similar findings were obained in unimmunized controls, except only 6/10 receiving he lowest dose developed tumors, 3 within 17 days, 3 within 43 days. In all groups, tumors from immunized mice weighed significantly more than tumors from controls. OA-mmunized mice were given 10^5, 10^4, or 10^3 cells. Tumors were detected in most immunized and control mice by day 12; however, only 6/12 controls given the lowest dose developed tumors. Tumor wt in mice given the highest OA dose was significantly greater than that in controls. Tumors from BSA- and OA-immunized mice showed no difference in the adsorption of sensitized sheep RBC; sera from the immunized mice markedly inhibited hemadsorption, but sera from normal mice did not. The distribution of immune complexes in mmunized and control mice is outlined. (25 refs)

78-3380 Immunologic Reactivity of Tumor Associated Antigens (TAA) Isolated from Plasma Membranes of Rat Moloney Sarcoma Cells (MST) (Meeting Abstract). (Eng) Leung, K. (Scripps Clinic & Res. Foundation, La Jolla, CA, 92037); Feldman, J. D.; Jones, J. M. *Fed Proc* 37(6): 1561; 1978. (no refs)

78-3381 Altered Moloney Leukemia Virus-Induced Cell Surface Antigens in an Immunoresistant Lymphoma Sub-Line (Meeting Abstract). (Eng) Troy, F. A. Dept. Biological Chemistry, Univ. California Sch. Medicine, Davis, CA, 95616); Fenyo, E. M.; Klein, G. *Fed Proc* 37(6): 569; 1978. (no refs)

78-3382 Immune Response to Epstein Barr Virus Induced Cell Surface Alterations (Meeting Abstract). (Eng) Biron, C. A. (Univ. North Carolina, Chapel Hill, NC, 27514); Hutt, L. M.; Pagano, J. S. *Fed Proc* 37(6): 1561; 1978. (2 refs)

78-3383 Serological Analysis of Cell Surface Antigens of Human Renal Cancer (Meeting Abstract). (Eng) Ueda, R. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Shiku, H.; Whitmore, W. F.; Oettgen, H. F.; Old, L. J. *Proc Am Assoc Cancer Res* 19: 198; 1978. (no refs)

78-3384 Serological Analysis of Cell Surface Antigens of Human Malignant Brain Tumors (Meeting Abstract). (Eng) Pfreundschuh, M. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Takahashi, T.; Oettgen, H. F.; Ransohoff, J.; Old, L. J. *Proc Am Assoc Cancer Res* 19: 198; 1978. (no refs)

78-3385 Tumor Associated Antigens of the Lewis Lung Carcinoma (Meeting Abstract). (Eng) McKolanis, J. R. (West Virginia Univ., Morgantown, WV, 26506). *Proc Am Assoc Cancer Res* 19: 69; 1978. (no refs)

78-3386 Expression of an Antigen Associated with Acute Lymphoblastic Leukemia in Human Leukemia-Lymphoma Cell Lines. (Eng) Minowada, J. (Dept. Immunology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263); Janossy, G.; Greaves, M. F.; Tsubota, T.; Srivastava, B. I.; Morikawa, S.; Tatsumi, E. *J Natl Cancer Inst* 60(6): 1269-1277; 1978.

Thirty human hematopoietic cell lines were studied for the presence of the cell-surface antigen (cALL antigen) associated with the non-T-cell, non-B-cell form of acute lymphoblastic leukemia (ALL). Nine of 20 proven leukemia-lymphoma cell lines were positive for cALL antigen, but all 10 normal Epstein-Barr virus-positive, B-lymphoblastoid cell lines were negative. On the basis of cell markers, the 9 cALL antigen-positive cell lines included 2 non-T-, non-B-cell ALL's, 1 lymphoid-type blast crisis of chronic myelocytic leukemia, 4/6 T-cell leukemia-lymphomas, and 2/10 B-cell lymphomas. Cross-absorption studies established the complete antigenic cross-reactivity of the cALL antigen on these various cell lines and fresh cALL antigen-positive leukemia cells. Although ALL's with a T-cell phenotype (T-cell ALL's) were previously shown to lack the cALL antigen, the antigen was definitely, although weakly, expressed on 7/89 uncultured T-cell leukemias. Serial studies of the cell cultures demonstrated stable expression of the cALL antigen. These results are interpreted in terms of a pluripotential stem cell and/or lymphoid precursor cell association of the cALL antigen. (58 refs)

78-3387 **“Arming” Factor in the Sera of Patients with Prostatic Cancer (Meeting Abstract).** (Eng) Bhatti, R. A. (Cook County Hosp., Chicago, IL, 60612); Ablin, R. J.; Baumgartner, G.; Nagale, V.; Guinan, P. D. *Proc Am Assoc Cancer Res* 19: 3; 1978. (2 refs)

78-3388 **Structure of Globin Gene in Transformed and Neoplastic Cells (Meeting Abstract).** (Eng) Miller, D. M. (NHLBI, NIH, Bethesda, MD, 20014); Axelrod, D.; Nienhuis, A. W. *Proc Am Assoc Cancer Res* 19: 219; 1978. (no refs)

78-3389 **The Effect of Interferon on the Early Functions of Simian Virus 40.** (Eng) Mozes, L. W. (Dept. Pathology, New York Univ. Sch. Medicine, New York, NY, 10016); Defendi, V. *Virology* 85(1): 315-318; 1978.

The ability of interferon (IF) to interfere with the penetration and uncoating of simian virus 40 (SV40) was studied by monitoring the inhibition of SV40-induced tumor antigen (T Ag) in permissive TC7 monkey cells and nonpermissive mouse embryo fibroblasts. Addition of IF to cells previously transformed by SV40 had no effect on T Ag production. However, pretreatment of cells with homologous IF 20 hr prior to SV40 injection resulted in a dose-dependent decrease in the proportion of T Ag-positive cells at 48 hr postinfection. IF addition at increasingly later times after SV40 injection resulted in a progressive reduction in T Ag-positive cells; the sensitivity to inhibition by IF was lost by 12 hr after infection. Penetration and uncoating of SV40 were ruled out as the major site of IF action, since the induction of T Ag by infectious DNA extracted from SV40 virions was also sensitive to IF treatment. Furthermore, addition of IF to cells did not result in resistance to viral infection until several hours had elapsed. These findings suggest that the inhibitory effect of IF is primarily at a step between SV40 uncoating and integration of viral DNA into the host cell genome. (16 refs)

78-3390 **Circulating Immune Complexes and Complement Levels in Human Neuroblastoma-bearing Nude Mice (Meeting Abstract).** (Eng) Brandeis, W. E. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Helson, L.; Khan, A.; Liu, Y. P.; Day, N. K. *Pro Am Assoc Cancer Res* 19: 80; 1978. (no refs)

78-3391 **In Vivo Functional Analysis of the Leukocyte Infiltrating Regressing Moloney Virus Induce Sarcomas (Meeting Abstract).** (Eng) DeFreitas, E. (Dept Microbiology, M. S. Hershey Medical Center, Hershey, PA 17033); Kreider, J. W.; Bartlett, G. L. *Proc Am Assoc Cance Res* 19: 196; 1978. (no refs)

78-3392 **Immune Macrophage Functions in High and Low Cancer Incidence Strains of Mice. A Comparative Study (Meeting Abstract).** (Eng) Gallily, R. (Dept. Immunology, Lautenberg Center for General and Tumor Immunology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Haran-Ghera, N. *Fed Proc* 37(6): 1841; 1978. (no refs)

78-3393 **In Vivo and In Vitro Effects of Tumor-enhancing IgG$_2$ on Macrophage-mediated Alloimmune Responsiveness (Meeting Abstract).** (Eng) Gilmore, J. C. (Univ. Mississippi Medical Center, Jackson, MS, 39216); Cruse, J. M.; Lewis, R. E. *Fed Proc* 37(3): 413; 1978. (no refs)

78-3394 **Cross-reactivities of Ethylnitrosourea-induced Neurogenic Rat Tumors Studied by In Vitro Assays for Cell-mediated Immunity.** (Eng) Oda, Y. (Dept. Neurosurgery, Kyoto Univ. Medical Sch., Shogoin-Kawaracho 54, Sakyo, Kyoto 606, Japan); Handa, H.; Kieler, J. *Eur J Cancer* 14(5): 491-499; 1978.

The cross-reactivities of neurogenic tumors induced transplacentally in Wistar rats by ethylnitrosourea (ENU; 60 mg/kg ip to the pregnant mother) were studied by various in vitro tests for cell-mediated immunity. Migration-inhibition tests (MIT) were performed using neurogenic tumor extracts, a spontaneous mammary carcinoma, and various normal adult and fetal tissues. An indirect ^{51}Cr-release assay and an indirect DNA-inhibition test were performed with culture supernatants from mixed cultures of neurogenic tumor cells and sensitized or nonsensitized lymphoid cells. All three assays revealed tumor-associated antigens with cross-reactions between various neurogenic tumors. Cross-reactions were also demonstrated by MIT between neurogenic tumors and fetal tissues. A peripheral neurinoma cross-reacted with adult brain, and a trigeminal nerve neurinoma cross-reacted with the spontaneous mammary carcinoma. None of the neurogenic tumors cross-reacted with adult liver. Thus, ENU-induced neurogenic tumors share one or several common antigens that are not tumor-specific. The reactivity of lymphoid cells from tumor-bearing rats was considerably weaker than that of lymphoid cells from hyperimmunized rats. The reactivity of lymphoid cells from the thymus and lymph nodes was considerably stronger than that of splenocytes. (24 refs)

78-3395 **Immune Responses to Weakly Immunogenic Virally Induced Tumors. 1. Overcoming Low Responsiveness by Priming Mice with a Syngeneic In Vitro Tumor Line or Allogeneic Cross-reactive Tumor.** (Eng) Galili, N. (Lautenberg Center General and Tumor Immunology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Devens, B.; Naor, D.; Becker, S.; Klein, E. *Eur J Immunol* 8(1): 17-22; 1978.

Attempts were made to overcome the immunologic unresponsiveness of A strain mice to YAC tumor, a syngeneic Moloney virus-induced lymphoma, and of C57BL/6 mice to RBL5 tumor, a Rauscher virus-induced lymphoma. Significant cytotoxic responses could be generated against the YAC tumor by immunizing A mice with YAC-1, a tissue culture line of YAC, and culturing the primed spleen cells from these immunized mice for 6 days. Spleen cells from A mice inoculated with tumor cells from the in vivo tumor line failed to generate a syngeneic cytotoxic response. Spleen cells from A mice that were inoculated with the cross-reactive allogeneic tumor RBL5 could generate significant cytotoxic responses to the syngeneic tumors YAC and YAC-1. Similarly, spleen cells from C57BL/6 mice inoculated with the cross-reactive allogeneic tumor YAC-1 could generate a significant cytotoxic response to the syngeneic tumor RBL5. RBL5, in contrast to the YAC tumor, was immunogenic in its in vivo form, and subsequent culturing did not improve its immunogenicity. Thus, cross-reactive allogeneic tumors can stimulate syngeneic cytotoxicity. (32 refs)

78-3396 **Idiotypic (ID) Determinants on Human Myeloma Bone Marrow T Cells. Modulation of ID Synthesis and Secretion of Plasma Cells by Anti-ID Activated T Cells (Meeting Abstract).** (Eng) Abdou, N. I. (Univ. Kansas, Kansas City, KS, 66103); Sagawa, A.; Abdou, N. L. *Proc Am Assoc Cancer Res* 19: 217; 1978. (no refs)

78-3397 **Is Protein Synthesis Necessary for the Commitment of Lymphocyte Transformation?** (Eng) Milner, J. (Dept. Biochemistry, Univ. Cambridge, Cambridge, England). *Nature* 272(5654): 628-629; 1978.

Studies were conducted to determine whether protein synthesis is necessary for the initial commitment of cells into the mitotic cell cycle following concanavalin A (Con A) stimulation of resting lymphocytes. Mitogenesis was measured by the incorporation of ^{3}H-thymidine into cellular DNA during the period 48-50.5 hr after exposure to Con A. Anisomycin, an inhibitor of protein synthesis, had no effect on the transformation of spleen cells from BALB/c mice when added to the culture medium at 3.8 nM; 10 nM anisomycin reduced transformation considerably, and at 38 nM it completely blocked Con A-induced transformation. The inhibitory effect of anisomycin could be reversed by replacing the medium containing anisomycin plus Con A with fresh medium plus Con A or with fresh medium only. The ability of lymphocytes to transform following removal of both anisomycin and Con A was similar to that observed when only the anisomycin was removed. The Con A-induced transformation of splenic lymphocytes is thus sensitive to very low concentrations of anisomycin, which block the increase in protein synthesis in con A-stimulated cells, as determined by measuring the incorporation of ^{35}S-methionine into proteins. The observation that lymphocytes become independent of extracellular Con A for transformation (after removal of both anisomycin and Con A) implies that commitment of lymphocytes to enter the mitotic cycle may be independent of protein synthesis. (8 refs)

78-3398 **Immunological Reactivity of BALB/c Mice During Moloney Sarcoma Virus Infection.** (Eng) Marshall, G. D. (Dept. Biology, Texas A&M Univ., College Station, TX); Foster, B. G. *FEMS Microbiol Lett* 3(1): 43-46; 1978.

Daily nonspecific immune responses were studied in BALB/c mice infected with Moloney murine sarcoma virus (MSV: 0.1 ml of a stock soln diluted to 0.62 log focus-forming unit/ml, im). Lymphocyte responses to phytohemagglutinin-p (PHA-P) indicated the existence of a generalized immunocompetence until day 12, when the MSV-induced tumors reached their max size. After declining on days 12-15, the PHA-P responses returned to normal on day 21, concomitant with tumor regression. Responses to concanavalin A increased exponentially after day 13 and remained elevated until day 7. A significant decline in the response to pokeweed mitogen on day 13 was repaired within 1 day. Mixed lymphocyte reaction responses paralleled the PHA-P responses, indicating a decrease in cell-mediated immunity on days 14-15. The results are discussed in terms of the relative maturation steps from uncommitted lymphocytes to immunologically mature T cells. (19 refs)

78-3399 **Are JHV Marek's Disease Tumor Cells B-Cells? (Meeting Abstract).** (Eng) Lam, K. M.

(Temple Univ., Philadelphia, PA, 19140); Spaulding, E. H.; Linna, T. J. *Fed Proc* 37(6): 1453; 1978. (no refs)

78-3400 **Evidence for Lymphocyte Cell Surface Receptors on Friend Erythroleukemic Cells (Meeting Abstract).** (Eng) Parker, C. L. (Dept. Anatomy, Bowman Gray Sch. Medicine, Wake Forest Univ., Winston-Salem, NC, 27103); Hooper, W. C. *In Vitro* 14(4): 388; 1978. (no refs)

78-3401 **Inhibition of Concanavalin A-mediated Lymphocyte Stimulation by Extracts of Lung Carcinomas (Meeting Abstract).** (Eng) Roth, J. A. (Div. Oncology, Dept. Surgery, Sch. Medicine, Univ. California, Los Angeles, CA, 90024); Chee, D. O.; Morton, D. L.; Holmes, E. C. *Proc Am Assoc Cancer Res* 19: 135; 1978. (no refs)

78-3402 **Abnormalities of Lymphocyte Transformations in Women with Condylomata Acuminata.** (Eng) Seski, J. C. (M.D. Anderson Hosp. and Tumor Inst., Houston, TX, 77025); Reinhalter, E. R.; Silva, J. *Obstet Gynecol* 51(2): 188-192; 1978.

The antigen-induced lymphocyte transformation responses of 16 women with recalcitrant condylomata acuminata were compared with those of 24 matched controls to determine if the growths are a reflection of an underlying defect in cell-mediated immunity. Lymphocyte transformation responses to phytohemagglutinin, concanavalin A, and pokeweed mitogen were significantly lower for the patients than for the controls. There was no significant difference between the two groups in lymphocyte transformation in Candida or streptokinase-streptodornase antigens. Approx 33% of each group took oral contraceptives; of these women, the mitogenic responses of the patients remained lower than those of the control group. In the patient group, 2 with diabetes mellitus, 1 with lupus erythematosis, and 1 with intraepithelial carcinoma of the vulva, vagina, and cervix had the lowest mitogenic responses. It is suggested that a refractory course of condylomata acuminatum in some women may reflect an underlying immunosuppressed state. (21 refs)

78-3403 **Allograft Cytotoxicity Co-Operation Between Alloimmune T Cells and Macrophages.** (Eng) Jones, B. (Dept. Immunology, Arthur Stanley House, Middlesex Hosp. Medical Sch., 40-50 Tottenham St., London W1P 9PG, England); Jones, T. C. *Immunology* 34(2): 247-255; 1978.

The ability of spleen cells from C57BL/10 (H-2b) mice to cooperate directly with normal macrophages in the destruction of target cells was investigated using a micro-culture system. Mice were immunized by ip injection of 2.5 x 10^7 P815Y (H-2d) cells, and 7-12 days later, spleen cells from these mice cooperated with an adherent component of nonimmune starch induced peritoneal cells in the cytostasis of P815Y target cells; no activity was expressed against an EL4 (H-2b) lymphoma. Although significant values for synergy could be obtained using the ^{125}I-iododeoxyuridine-incorporation assay to measure cytostasis, normal peritoneal cells were incapable of cooperating with T cells in cytolysis, as measured by ^{51}Cr release from prelabeled target cells. Initially, the synergistic interaction was immunologically specific, but nonspecific activity could be induced by challenge with specific antigen. Furthermore, both the direct specific synergistic reaction and the indirect nonspecific reaction required T cell function. (12 refs)

78-3404 **The Immunodepressive Effect of Friend Virus. IV. Effects on Spleen B Lymphocytes.** (Eng) Dracott, B. N. (Immunology Div., Dept. Pathology, Royal Coll. Surgeons England, Tennis Court Road, Cambridge CB2 1QP, England); Wedderburn, N.; Doenhoff, M. J. *Immunology* 34(4): 679-687; 1978.

Splenic immune responses having varying dependence on accessory cell cooperation were studied after the infection of mice with Friend virus (FV). Infection had no effect on cell proliferation or antibody production in cultures stimulated with *Escherichia coli* lipopolysaccharide. The in vivo response to type III pneumococcal polysaccharide was depressed only moderately. The response to sheep RBC was depressed severely both in vivo and in vitro. However, the latter was greatly reduced by costimulation of the cultures with *Escherichia coli* lipopolysaccharide. The depletion of potential suppressor lymphocyte populations by irradiation or adult thymectomy did not ameliorate the depression of responses to sheep RBC or pneumococcal polysaccharide as a result of FV infection. However, when mice were subjected to thymectomy, irradiation, and marrow restoration, FV infection had no significant effect on the responses of T-lymphocyte-depleted mice. It is suggested that precursor B-lymphocyte depletion is not responsible for immune depression by FV, at least after short infection intervals. It is also suggested that FV acts on inductive rather than effector processes and that the greater the resulting depression, the greater the dependence of the latter on cooperation, especially by T lymphocytes. Conversely, precursor B-lymphocyte subpopulations that respond in the absence of cooperation may be affected less or not at all by FV. (44 refs)

78-3405 **Induction of Aryl Hydrocarbon Hydroxylase (AHH) in Human T-Lymphocytes (Meeting Ab-**

stract). (Eng) Alfred, L. (Charles R. Drew Medical Sch., Los Angeles, CA, 90059); Venkatesan, N.; Kouri, R. E. *Proc Am Assoc Cancer Res* 19: 74; 1978. (no refs)

78-3406 **Studies on the Specificity and Control of the Cytolytic T Lymphocyte Response to Murine Leukemia Virus-Induced Tumors (Meeting Abstract).** (Eng) Plata, F. (Albert Einstein Coll. Medicine, Bronx, NY, 10461); Lilly, F. *Fed Proc* 37(6): 1569; 1978. (no refs)

78-3407 **Immunochemical Characterization of Two Antigenic Components on Human Normal and Cultured T Lymphocytes (Meeting Abstract).** (Eng) Ades, E. W. (Univ. Alabama Medical Center, Birmingham, AL, 35294); Balch, C. M.; Zwerner, R. K.; Acton, R. T.; Durant, J. R. *Proc Am Assoc Cancer Res* 19: 137; 1978. (1 ref)

78-3408 **Immunopathology of Fatal Infectious Mononucleosis in an X-Linked Recessive Lymphoproliferative Syndrome (Meeting Abstract).** (Eng) Purtilo, D. T. (Dept. Pathology, Univ. Massachusetts Medical Center, Worcester, MA, 01505); Bhawan, J. *Lab Invest* 38(3): 361; 1978. (no refs)

78-3409 **Abrogation of Syngeneic Transplantable Ib Tumors with DNP-Conjugated Anti-Ib Tumor Cell Antibodies (Meeting Abstract).** (Eng) Paque, R. E. (Dept. Microbiology, Univ. Texas Health Science Center, San Antonio, TX, 78284); Duffey, P. S.; Nealon, T. *Proc Am Assoc Cancer Res* 19: 25; 1978. (no refs)

78-3410 **Blocking of Specific Binding to Tumor Antigens by Fibrinolysin-Cleaved Antibody (Meeting Abstract).** (Eng) Nathanson, S. D. (Dept. Surgery, Univ. California, Los Angeles, CA, 90024); Kempner, D. *Proc Am Assoc Cancer Res* 19: 133; 1978. (no refs)

78-3411 **Isolation and Characterization of Antitumor Antibodies from Cancer Patient Sera by Affinity Chromatography (Meeting Abstract).** (Eng) Gupta, R. K. (Div. Oncology, Dept. Surgery, Sch. Medicine, Univ. California, Los Angeles, CA, 90025); Silver, H. K.; Reisfeld, R. A.; Morton, D. L. *Proc Am Assoc Cancer Res* 19: 133; 1978. (no refs)

78-3412 **Prevalence of Neutralizing Antibodies Against Adenoviruses at Lucknow.** (Eng) Mathur, A. (Upgraded Dept. Pathology and Bacteriology, K.G. Medical Coll., Lucknow-226003, India); Singh, G.; Singh, U. K.; Mehrotra, R. M.; Chaturvedi, U. C. *Indian J Med Res* 67: 19-26; 1978.

Blood samples from 400 healthy subjects of Lucknow, India were analyzed for the presence of antibodies (ab) to adenovirus (Ad) types 2, 7, and 8. Ab against one or more of the Ad's were found in 103 sera, 14 of which contained ab to more than one type of Ad. Most of the sera were positive for Ad2 (55 samples), followed by Ad7 (40) and Ad8 (23). The highest incidence of Ab was seen in children < 10 yr of age. (19 refs)

78-3413 **Protective Effect of Antibodies Against Isologous Aggregated Immunoglobulins in Experimental Leukemia.** (Rus) Kul'berg, A. Ia. (N. F. Gamaleia Inst. Epidemiology and Microbiology, Moscow, USSR); Chernousova, L. N.; Shevliagin, V. Ia.; Morgunova, T. D. *Dokl Akad Nauk SSSR* 239(5): 1233-1234; 1978.

To test whether elimination of the specific blocking factor (the factor that defends tumor cells from T lymphocytes, or killer cells) would result in regression of tumor growth, BALB/c mice inoculated with Rauscher virus were immunized with antiserum (MAAS) against aggregated immunoglobulins. MAAS antiserum was obtained from BALB/c mice immunized with isologous immunoglobulins that had been heated at 63 C for 20 min. The antiserum (0.1 ml) was injected ip daily for the first 4 days, every other day for the next 22 days. Mice in the control group received injections of normal serum from BALB/c mice. Although all mice developed leukemia, the lifespan of mice treated with MAAS antiserum was 46.5 days, compared with 30.9 days in the controls. (11 refs)

78-3414 **Humoral Antibodies in Patients with Leukemia and Infectious Mononucleosis.** (Rus) Vyadro, M. M. (Lab. Experimental Tumor Therapy, P. A. Gertsen Scientific Res. Cancer Inst., Moscow, USSR); Bergolts, V. M.; Korzhenkova, M. P. *Pediatriia* (8): 47-50; 1977.

To determine whether the serum from patients with infectious mononucleosis (IM) contains cytotoxic anti-leukemic antibodies, 20 serum specimens from 17 children with IM were tested in a direct cytotoxicity assay with cell targets from 10 leukemic patients (6 had acute lymphoblastic leukemia, 3 chronic lymphoblastic leukemia, and 1 chronic myeloid leukemia). Cytotoxic antileukemic antibodies were absent in all 20 sera, but some sera contained noncytotoxic 7S antibodies, which could be detected in an indirect cytotoxicity assay. Preincubation of the serum with leukemic cell targets enhanced the cytotoxic effect of anti-7S serum. (12 refs.)

78-3415 **Specificity of Antibody Induced in Sarcoma Patients Immunized with Allogeneic Sarcoma Cells (Meeting Abstract).** (Eng) Saxton, R. E. (Div. Oncology, Dept. Surgery, Sch. Medicine, Univ. California, Los Angeles, CA, 90024); Golub, S. H.; Morton, D. L. *Proc Am Assoc Cancer Res* 19: 135; 1978. (no refs)

78-3416 **In Vitro Assays of Monocyte (M) Function in a Human Tumor System (Meeting Abstract).** (Eng) Mukherji, B. (Tufts-New England Medical Center Hosp., Boston, MA, 02111); Nathanson, L. *Proc Am Assoc Cancer Res* 19: 141; 1978. (no refs)

78-3417 **Human Monocyte-induced Tumor Cell Cytotoxicity.** (Eng) Rinehart, J. J. (Hematology Section, Veterans Admin. Hosp., 54th St. and 48th Ave., Minneapolis, MN, 55417); Lange, P.; Gormus, B. J.; Kaplan, M. E. *Blood* 52(1): 211-220; 1978.

Monocyte-induced tumor cell cytotoxicity (MITC) was examined in vitro using monocytes obtained from normal human donors and four human tumor cell lines: CAK-5, a renal cell carcinoma; T-24, a transitional bladder cell carcinoma; Malme-3M, a melanoma; and SK-MES-1, an epidermoid lung carcinoma. Cocultivation of monocytes and tumor cells, as observed by light microscopy, resulted in marked monocyte-tumor cell interaction and a reduction in tumor cell number. Excellent correlation was obtained between the percentage of cytotoxicity determined by direct cell counts and by ^{3}H-thymidine incorporation. MITC increased with monocyte/tumor cell ratios in three tumor cell lines but not in SK-MES-1. Monocytes were significantly more cytotoxic for Malme-3M than for the other three lines (80% cytotoxicity at a monocyte/target cell ratio of 32:1 vs 38%-50% for the other three lines). MITC was not due to a soluble cytotoxin, serum complement, or putative "natural" tumor cell antibodies, and it was not a nonspecific effect of cell-cell interaction. MITC was not mediated by reduction in space available for tumor cell adherence and it was not an artifact due to media depletion. These results suggest that human monocytes have a cytotoxic potential against tumor cells in vivo. (21 refs)

78-3418 **IgM and IgG Antibody-Dependent Cell-Mediated Cytotoxicity in the Moloney Sarcoma Virus System: No Requirement for the Fifth Component of Complement (Meeting Abstract).** (Eng) Fuson, E. W. (Birmingham, AL, 35294); Lamon, E. W.; Alford, C. A. *Fed Proc* 37(6): 1381; 1978. (no refs)

78-3419 **Antibody-Dependent Cell-Mediated Cytotoxicity Against Syngeneic Polyoma-Transformed Tumor Cells (Meeting Abstract).** (Eng) Walia, A. S. (Birmingham Veterans Admin.Hosp., Birmingham, AL, 35294); Lamon, E. W. *Fed Proc* 37(6): 1381; 1978. (no refs)

78-3420 **Multicell Spheroids as a Model Target for In Vivo Immunity to Solid Tumor Allografts (Meeting Abstract).** (Eng) Lord, E. M. (Univ. Rochester Cancer Center, Rochester, NY, 14642); Sutherland, R. M. *Proc Am Assoc Cancer Res* 19: 71; 1978. (no refs)

78-3421 **Development of In Vitro and In Vivo Anti-Tumor Cytotoxicity in Noncytotoxic, Tumor-bearer Spleen Cells (Meeting Abstract).** (Eng) Mokyr, M. B. (Dept. Microbiology, Univ. Illinois Medical Center, Chicago, IL, 60612). *Proc Am Assoc Cancer Res* 19: 201; 1978. (no refs)

78-3422 **Antibody-dependent Cellular Cytotoxicity (ADCC) to EL-4: Inhibition by Autologous Lymphoid Cells (Meeting Abstract).** (Eng) Pollack, S. B. (Cancer Res. Center, Seattle, WA, 98104); Herrick, M. V.; Hutchinson, F. *Proc Am Assoc Cancer Res* 19: 53; 1978. (no refs)

78-3423 **Derepression of Nuclear Template Restrictions for DNA Synthesis by the Immunostimulator Pyran Copolymer.** (Eng) Mohr, S. J. (Div. Urology, Univ. Colorado Medical Center, Denver, CO, 80220); Massicot, J. G.; Chirigos, M. A. *Cancer Res* 38(6): 1610-1616; 1978.

The effect of the immunostimulator pyran copolymer on in vitro DNA synthesis was investigated using mouse liver nuclei. Pyran interacted with the repressed genome of isolated murine liver nuclei and allowed DNA synthesis to take place. This interaction occurred immediately and was influenced by the magnesium concentration, pyran concentration, and mol wt of the pyran. Furthermore, pyran could reverse the histone-induced inhibition of a pure poly(deoxyadenylate-deox-

thymidylate) template primer for DNA synthesis. Parallel
esults were obtained with both intact nuclei and nuclei that
ad undergone disruption, indicating that no interaction with
ie nuclear membrane was involved in these results. In addi-
on, morphological swelling of the nuclei was seen in the
resence of pyran concentrations that were maximally
imulatory to DNA synthesis. Pyran, therefore, is able to
elease the DNA template of murine liver nuclei from restric-
ons that normally would prevent them from serving as a
seful template for DNA synthesis. It is not known whether
ie ability of pyran to derepress the nuclear DNA template
related to its action in potentiation of vaccine, macrophage
imulation, and interferon induction. (33 refs)

8-3424 **Immunosurveillance and Immunostimulation of Primary Murine Rous Sarcoma (Meeting Ab-**
tract). (Eng) Haughton, G. (Univ. North Carolina Medical
ch., Chapel Hill, NC, 27514); Banks, R.; Babcock, G.; Whit-
iore, A. C. *Fed Proc* 37(6): 1280; 1978. (no refs)

8-3425 **Ability of Tumor Factors to Enhance Growth of an SV40-induced Tumor in Hamster (Meeting**
bstract). (Eng) Strickland, F. (Dept. Pathology, Univ.
hicago, Chicago, IL, 60637); Hunter, R. *Fed Proc* 37(7):
107; 1978. (no refs)

8-3426 **Stimulation of Tumor Colony Formation by Peritoneal Exudate Cells (PEC) (Meeting Ab-**
tract). (Eng) Jessup, J. M. (Basic Res. Program, NCI-
rederick Cancer Res. Center, Frederick, MD, 21501). *Proc*
m Assoc Cancer Res 19: 31; 1978. (no refs)

8-3427 **A Stimulatory Factor in Human Cancer Ascites Fluid (Meeting Abstract).** (Eng) Badger, A. M.
Boston Univ. Sch. Medicine, Boston, MA, 02118). *Proc Am*
ssoc Cancer Res 19: 72; 1978. (no refs)

8-3428 **Failure to Demonstrate Immunodepression as a Contributing Component in Radiation**
eukomogenesis of the RFM Mouse (Meeting Abstract).
Eng) Perkins, E. H. (Biology Div., Oak Ridge Natl. Lab.,
ak Ridge, TN, 37830); Cacheiro, L. H. *Fed Proc* 37(6):
280; 1978. (no refs)

78-3429 **Preferential Depression of IgM Producing Immunocytes during Mastocytoma-induced Immunosuppression.** (Eng) Friedman, H. (Dept. Microbiology, Albert Einstein Medical Center, Philadelp ., PA, 19141); Kamo, I. *Proc Soc Exp Biol Med* 157(3): 381-385; 1978.

The effects of immunosuppressive PB815X mastocytoma cells and cell-free extracts of this tumor on the appearance of IgM vs IgG antibody-producing cells were examined in DBA/2 mice after primary or secondary immunization. Immunologic impairment was noted in mice bearing PB815X tumors and in mice inoculated with cell-free extracts and/or ascites fluid from the tumor-bearing animals. The 19S IgM antibody response to sheep RBC was preferentially affected. Although there were 60%-80% fewer IgM plaque-forming cells (PFC) in mice given a primary injection with sheep RBC, there was only a slight effect on the appearance of indirect 7S IgG PFC. Mice primed with sheep RBC and then implanted with mastocytoma cells showed a marked impairment of the secondary IgM response but no depression of the IgG response. Injection of cell-free ascites fluid from tumor-bearing mice into normal recipient mice before primary or secondary immunization with sheep RBC resulted in marked depression of the IgM response only. These results suggest that PB815X cells preferentially affect the immunocytes producing IgM hemolytic antibody. (10 refs)

78-3430 **In Vitro Depression of Lymphocyte Response to PHA by Acute Phase Reactants (Meeting Abstract).** (Eng) Israel, L. (Univ. Paris XIII, CHU Bobigny, 93000, France); Edelstein, R.; Samak, R. *Proc Am Assoc Cancer Res* 19: 10; 1978. (no refs)

78-3431 **Biphasic Immunosuppression in Chickens Infected with Marek's Disease Virus (Meeting Abstract).** (Eng) Lee, L. F. (United States Dept. Agriculture, ARS, Regional Poultry Res. Lab., E. Lansing, MI, 48823); Sharma, J. M.; Nazerian, K.; Witter, R. L. *Fed Proc* 37(6): 1357; 1978. (no refs)

78-3432 **Suppression of Migration Inhibition Factor (MIF) Production by Macrophages from Mice Bearing MSV-Induced Tumors (Meeting Abstract).** (Eng) Varesio, L. (NCI, NIH, Bethesda, MD, 20014); Jarrell, E. M.; Landolfo, S.; Holden, H. T. *Fed Proc* 37(6): 1354; 1978. (no refs)

78-3433 **Ontogeny of an In Vitro Generated Suppressor Cell (Meeting Abstract).** (Eng) Rollwagen, F.

M. (Memorial-Sloan Kettering Cancer Center, New York, NY, 10021); Stutman, O. *Fed Proc* 37(3): 559; 1978. (no refs)

78-3434 **Suppressor Cell Activity in Immunodepressed Lung and Breast Cancer Patients (Meeting Abstract).** (Eng) Jerrells, T. R. (Litton Bionetics, Inc., Kensington, MD, 20795); Dean, J. H.; Richardson, G. L.; Vadlamudi, S.; Herberman, R. B. *Proc Am Assoc Cancer Res* 19: 73; 1978. (no refs)

78-3435 **A Humoral Suppressor Factor in Tumor-bearing Mice (Meeting Abstract).** (Eng) Landes, R. G. (Univ. Minnesota, Minneapolis, MN, 55455); Ristow, S. S.; McKhann, C. F. *Proc Am Assoc Cancer Res* 19: 231; 1978. (no refs)

78-3436 **Macrophages and Resistance To Tumours. I. Inhibition of Delayed-Type Hypersensitivity Reactions by Tumour Cells and by Soluble Products Affecting Macrophages.** (Eng) Nelson, M. (Kolling Inst. Medical Res., Royal North Shore Hosp., St. Leonards NSW 2065, Australia); Nelson, D. S. *Immunology* 34(2): 277-290; 1978.

Tumor cell inhibition of delayed-type hypersensitivity (DTH) reactions was examined. A/J mice were footpad-inoculated with mixtures of sheep RBC to which they had developed DTH and syngeneic 3-methylcholanthrene-induced fibrosarcoma cells. DTH reactions were associated with a depression of subsequent tumor growth compared with that in control animals. The degree of depression was proportional to the intensity of the reactions. Conversely, the presence of fibrosarcoma cells was associated with depression of DTH reactions. Serum-free culture supernatants of various mouse, human, and rat tumors depressed DTH reactions in mice, the depressive activity being apparent even at dilutions > 1:100. Supernatants from cultures of nonmalignant cells had little or no depressive activity. Protein synthesis, but not DNA synthesis, was required for the production of the responsible factors. In vitro, the active supernatants markedly inhibited macrophage migration, either spontaneously or in response to a chemotactic factor, but they had much less effect on mitogen-stimulated or unstimulated lymphocyte cultures. The activity was decreased or lost after treatment with proteolytic enzymes, ribonuclease, neuraminidiase, or hyaluronidase. Membrane ultrafiltration showed that the activity was associated with materials of mol wt > 1,000. However, materials of mol wt > 10,000 and < 10,000 had different in vivo effects, with the larger factors being more depressive of the later phases of the reaction and the smaller factors being more depressive of the earlier. Injection of concentrated supernatants from the tumors together with fibrosarcoma cells led to more rapid initial tumor growth. (36 refs)

78-3437 **Immunity to Transplantable Carcinogen-induced Fibrosarcomas in B2/B2 Chickens. III. Tumor Growth Inhibition by Local Delayed Hypersensitivity Reactions to Unrelated Antigens.** (Eng) Palladino, M. A. (Dept. Pathology, New York Univ. Sch. Medicine, 550 First Ave., New York, NY, 10016); Thorbecke, G. J. *Eur J Immunol* 8(4): 257-262; 1978.

In SC chickens, the growth of transplantable fibrosarcomas (SCFS), which were derived from 7,12-dimethylbenz(a)anthracene-induced primary tumors, was suppressed by delayed hypersensitivity (DH) reactions to human immunoglobulin (Ig), *Corynebacterium parvum*, or allogeneic cells at the site of tumor cell injection. Spleen cells of SC chickens sensitized to human Ig or *C. parvum* suppressed SCFS growth only when injected simultaneously with the sensitizing antigen. This suppression did not occur in irradiated recipients. SCFS I cells injected into wattles of tumor-immune chickens provoked a local DH reaction. Spleen cells from donors sensitized to SCFS I adoptively transferred immunity to both SCFS I and SCFS II when cells of the two tumors were mixed together before injection, but not when SCFS II cells were injected alone. Tumor-specific and non-tumor-specific DH may be essential for the local suppression of fibrosarcoma growth in chickens. (31 refs)

78-3438 **Natural Killer Cells May Be the Only Cells in Normal Mouse Lymphoid Cell Populations Endowed with Cytolytic Ability for Antibody-coated Tumour Target cells.** (Eng) Ojo, E. (Dept. Immunology, Uppsala Univ. Biomedical Centre, Box 582, Uppsala, S-751 23, Sweden); Wigzell, H. *Scand J Immunol* 7(4): 297-306; 1978.

Normal mouse lymphoid cells were analyzed for their ability to act as natural killer (NK) cells against an NK-sensitive tumor target (YAC), to act as effector cells against IgG-coated P815 cells, and to function as effector cells against IgG-coated chicken RBC. NK activity and antibody-dependent cell cytotoxicity (ADCC) against IgG-coated P815 cells were found to vary in parallel with respect to the age, organ source, and genotype of the effector cells. On the other hand, ADCC against chicken RBC was largely performed by effector cells distinct from those functioning as NK cells or in ADCC against P815 cells. Preincubation of the cells at 37 C for 4 hr decreased the lytic efficiency of ADCC anti-P815 and NK activity significantly, indicating that both were temperature-sensitive, in contrast to ADCC against chicken RBC. Likewise, ip inoculation of mice with *Corynebacterium parvum* reduced ADCC against chicken RBC, but it increased ADCC against P815 and NK activity. Blocking experiments using 'cold' inhibitor cells in cytolytic assays indicated that NK cells and effector cells against IgG-coated P815 cells were the same. It is thus concluded that NK cells in the mouse also have the ability to express killer cell activity against IgG-coated tumor target cells; NK cells may be the only cell type in the mouse equipped with cytolytic potential for antibody-coated murine nucleated cells. (32 refs)

78-3439 Possible Involvement of Natural Killer Cells in Bone Marrow Graft Rejection. (Eng) Lotzova, E. (Dept. Developmental Therapeutics, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); Savary, C. A. *Biomedicine [Express]* 27(9/10): 341-344; 1977.

The similarities between natural killer (NK) cells and hematopoietic resistance effector (HR-E) cells were investigated. Young mice did not express NK or HR-E cells when challenged with parental bone marrow grafts; adult mice were good responders to both types of cells. A dose of 1,100 R whole-body irradiation given 24 hr before in vitro cytotoxicity assays did not abrogate or decrease NK-cell cytotoxicity; HR-E cells were previously shown to be radioresistant. Both populations could be inhibited by cyclophosphamide, silica, carrageenan, and *Corynebacterium parvum*. Mice tolerant to bone marrow grafts and therefore accepting parental marrow transplants showed decreased NK-cell reactivities. These findings suggest, but do not prove, that NK and HR-E cells represent the same population. (29 refs)

78-3440 Natural Killing in Immunodeficient Patients. (Eng) Koren, H. S. (Div. Immunology, Duke Univ. Medical Center, Durham, NC, 27710); Amos, D. B.; Buckley, R. H. *J Immunol* 120(3): 796-799; 1978.

To identify the effector cell for natural killing (NK), NK capacity was evaluated in the peripheral blood mononuclear cells from 14 patients with well-defined primary immunodeficiency (ID) disorders, as these disorders have distinct lymphocyte subpopulation membrane receptor and/or functional abnormalities. In addition, the antibody-dependent cell-mediated cytotoxicity (ADCC) of these cells against antibody-coated RBC and lymphoid or tumor targets was assayed. The patients comprised 5 with X-linked agammaglobulinemia (Xa γ), 6 with common variable agammaglobulinemia (CVA γ), 2 with severe combined ID (SCID), and 1 with ID with hyper-IgM (HypM). NK activity was normal but ADCC against lymphocyte targets was grossly impaired in the HypM and XAγ patients. This selective inability to lyse antibody-coated nonerythroid target cells suggests that (1) two separate subpopulations of effector cells may exist for the two types of killing, (2) one effector cell may exist for both systems, but the effector functions are mediated by two distinct cell-surface recognition structures, or (3) one effector cell exists for both systems but ADCC is more susceptible than NK to abnormalities affecting lymphoid cell function. Cells from the CVAγ patients had demonstrable NK and ADCC activity. The degree of reactivity among these patients varied widely, and it probably reflects the heterogeneous nature of the ID in this group. The only ID state in which lymphocyte NK activity was found to be lacking was SCID. SCID patients showed no ADCC against nonerythroid targets, but they showed normal ADCC against erythroid targets. It is suggested that monocytes and K lymphocytes are capable of mediating ADCC against erythroid targets but that only K lymphocytes can lyse antibody-coated nonerythroid targets. The fact that NK is lacking in the most severe form of ID indicates that NK may be among the more primitive of the host-defense and surveillance mechanisms. (23 refs)

78-3441 Cell Proliferation and the In Vitro Secondary Cytotoxic Response to Syngeneic Mouse Tumor Cells (Meeting Abstract). (Eng) Stiller, R. A. (Lab. Immunodiagnosis, NCI, NIH, Bethesda, MD, 20014); Holden, H. T. *Proc Am Assoc Cancer Res* 19: 112; 1978. (no refs)

78-3442 Oral Administration of Syngeneic or Allogeneic Tissues Alters Growth Rate of an Allotransplantable Tumor in Mice (Meeting Abstract). (Eng) Breyere, E. J. (Dept. Biology, American Univ., Sibley Memorial Hosp., Washington, DC, 20016); Heath, J. R. *Proc Am Assoc Cancer Res* 19: 51; 1978. (no refs)

78-3443 Murine Colon Adenocarcinoma: Immunobiology (Meeting Abstract). (Eng) Goldrosen, M. H. (Dept. Surgical Oncology, Roswell Park Memorial Inst., Buffalo, NY, 14263); Tan, M. H.; Leveson, S. H. *Proc Am Assoc Cancer Res* 19: 233; 1978. (no refs)

78-3444 Tumor Induction in Host-Versus-Graft Disease (Meeting Abstract). (Eng) Cornelius, E. (Yale Univ., New Haven, CT, 06510). *Fed Proc* 37(3): 412; 1978. (no refs)

78-3445 Unique and Common Tumor-specific Transplantation Antigens of Chemically Induced Mouse Sarcomas. (Eng) Hellstrom, K. E. (Div. Tumor Immunology, Fred Hutchinson Cancer Res. Center, Seattle, WA); Hellstrom, I.; Brown, J. P. *Int J Cancer* 21(3): 317-322; 1978.

Nine transplantable lines of sarcomas induced in female BALB/c mice by hind leg injection of 0.1 mg 3-methylcholanthrene (3-MC) were tested for their ability to induce transplantation immunity against themselves and against other syngeneic 3-MC induced sarcomas. The mice were immunized by sc injection of 5 x 10^7 x-irradiated tumor cells. Each of the nine tumor lines could induce a stronger tumor-specific transplantation resistance against itself than against other tumors. However, there was also a weak cross-reaction between tumors in the transplantation test, indicative of common tumor-specific transplantation antigens (TSTA). Cross-reactions were seen significantly more often when the immunizing tumor expressed murine leukemia virus (MuLV) antigens, and this was irrespective of the MuLV expression of the challenge tumor. Thus, not only unique, but also common TSTA were found in these lines. (19 refs)

78-3446 **The Deciduo-Placental Barrier Blocks Transmission of a Rat Malignant Lymphoma-Leukemia (Meeting Abstract).** (Eng) Hass, G. M. (Rush-Presbyterian-Saint Luke's Medical Center, Chicago, IL, 60612); McCreary, P. A.; Laing, G. H.; Galt, R. M. *Fed Proc* 37(3): 680; 1978. (no refs)

78-3447 **Characterization of Hereditarily Asplenic-Athymic (LASAT) Mice (Meeting Abstract).** (Eng) Lozzio, B. B. (Center Health Sciences, Univ. Tennessee Memorial Res. Center, Knoxville, TN, 37920); Machado, E. A.; Lair, S. V.; Aggio, M. C.; Ichiki, A. T. *Proc Am Assoc Cancer Res* 19: 64; 1978. (no refs)

78-3448 **Mononuclear Cell Accumulation Stimulated by an Extract of EL4 Leukemia Cells (Meeting Abstract).** (Eng) Berd, D. (Fox Chase Cancer Center, Philadelphia, PA, 19111); Wilson, E. J. *Proc Am Assoc Cancer Res* 19: 125; 1978. (no refs)

78-3449 **Cell-mediated Immunity (CMI) Against a Methylcholanthrene Induced Sarcoma (METH-A) by Detergent Solubilized Tumor Associated Antigen (Meeting Abstract).** (Eng) Padarathsingh, M. L. (Litton Bionetics, Inc., Kensington, MD, 20795); Dean, J. H.; Northing, J. W.; McCoy, J. L.; Lavrin, D. H.; Natori, T.; Law, L. W. *Proc Am Assoc Cancer Res* 19: 208; 1978. (no refs)

78-3450 **Allogeneic Nude Mice Used as Recipients in a Winn Assay (Meeting Abstract).** (Eng) Lill, P. H. (Sch. Medicine, Univ. South Carolina, Columbia, SC, 29208). *Proc Am Assoc Cancer Res* 19: 127; 1978. (no refs)

78-3451 **Analysis of the Humoral Immune Response in Mice Following the Spontaneous Regression of Friend Virus-Induced Erythroleukemia (Meeting Abstract).** (Eng) Dietz, M. (Dept. Biology, Michigan Cancer Foundation, Detroit, MI, 48201); Hines, D.; Rich, M. A.; Furmanski, P. *Proc Am Assoc Cancer Res* 19: 156; 1978. (no refs)

78-3452 **Effect of Different Modalities of Active Immunization on Incidence of Tumors in A/J Mice Sarcoma Model (Meeting Abstract).** (Eng) El Domeiri, A. (Dept. Surgery, Univ. Illinois Medical Center, Chicago, IL, 60612); Sabet, T. Y.; Hsia, W. C. *Proc Am Assoc Cancer Res* 19: 123; 1978. (no refs)

78-3453 **Immunologic Mechanisms Involved in the Establishment and Maintenance of the Tumor Dormant State (Meeting Abstract).** (Eng) Weinhold, K. J. (Thomas Jefferson Univ., Philadelphia, PA, 19107); Wheelock, E. F. *Proc Am Assoc Cancer Res* 19: 95; 1978. (no refs)

78-3454 **Surface Immunoglobulins in Relation to Other Characteristics of Transplantable, Spontaneous, Reticulum-Cell Sarcomas Type B of BALB/c Mice (Meeting Abstract).** (Eng) Merwin, R. M. (NIH, Bethesda, MD, 20014); Asofsky, C.; Campbell, P. S. *Proc Am Assoc Cancer Res* 19: 222; 1978. (no refs)

78-3455 **The Source of Immume RNA Capable of Transferring Tumor-Specific Immunity (Meeting Abstract).** (Eng) Wang, B. S. (Harvard Medical Sch., Boston, MA, 02115); Onikul, S. R.; Mannick, J. A. *Fed Proc* 37(3): 412; 1978. (no refs)

78-3456 **Further Characterization of Immune RNA Capable of Transferring Tumor Immunity (Meeting Abstract).** (Eng) Wang, B. S. (Harvard Medical Sch., Boston, MA, 02115); Mannick, J. A. *Proc Am Assoc Cancer Res* 19: 21; 1978. (no refs)

78-3457 **Serum Factors Modulating Host Immunity to SV40 Tumor Cells.** (Eng) Lausch, R. N. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA, 17033); Prather, S. O. *Fed Proc* 37(7): 2095-2096; 1978.

The immune response of LSH hamsters to isografts of simian virus 40 (SV40)-transformed tumor cells (PARA-7) was investigated. When spleen cells from tumor-bearing animals were tested immediately, they were not cytotoxic to PARA-7 target cells, but they became cytotoxic when they were incubated in vitro overnight. Spleen cells from SV40-sensitized hosts or from animals whose tumor had been surgically removed, were active on direct testing. These findings suggest that the tumor cells released a substance that could inhibit the activity of sensitized spleen cells and that this phenomenon could be reversed. Study of the serum factors modulating host tumor immunity indicated that progressive growth of PARA-7 transplants was accompanied by a demise of serum antibody-dependent cell cytotoxicity (ADCC), an increase in serum blocking, and a loss of spleen cell cytotoxicity. Events occurring during incubation that resulted in reversal of tumor-mediated suppression of cellular cytotoxicity were examined next. During overnight incubation of unreactive spleen cells, a substance was released into the medium that could block cell-mediated immunity. This medium could not mediate ADCC. However, medium overlying effector cells active on direct testing had ADCC but no blocking activity. Antigen extracted from PARA-7 tumor cells and admixed with sensitized effector cells specifically blocked cellular cytotoxicity. Incubation of antigen with serum or medium mediating ADCC resulted in neutralization of such activity and concomitant formation of a factor whose blocking properties in the microcytotoxicity assay were similar to that of tumor bearer serum. Antigen by itself did not block at the level of the target cell. (7 refs)

78-3458 **Inhibitory Effect of BCG Vaccine on Specific Antitumor Immunity of Syrian Hamsters.** (Rus) Agibalova, L. V. (Cancer Res. Center, Moscow, USSR). *Vestn Akad Med Nauk SSSR* (2): 42-46; 1978.

The effect of 'wet' and lyophilized BCG vaccine on nduced specific antitumor immunity was studied in Syrian hamsters. The animals were immunized with a single ip injection of simian virus 40 (SV40: 1 ml) or of 10^5-10^7 irradiated (12,000 rads) tumor cells (SV40-nduced sarcoma ICV or spontaneous hepatoma Gt-11B). Antitumor immunity was assessed by in vivo transplantation tests. Sc injection of 'wet' BCG vaccine, at doses ranging from 0.25 to 1.0 mg per 0.2 ml, reduced specific resistance of immunized hamsters to SV40. This did not occur with the lyophilized form. In experiments with irradiated tumor cells, again only the 'wet' BCG vaccine decreased the antitumor resistance of inoculated hamsters both to sarcoma ICV and hepatocellular carcinoma. (18 refs)

78-3459 **Transplantation Resistance to Line-10 Hepatoma After Growth and Excision (Meeting Abstract).** (Eng) Canti, G. F. (NIH, Bethesda, MD, 20014); Hunter, J. T.; Zbar, B.; Rapp, H. J. *Proc Am Assoc Cancer Res* 19: 90; 1978. (no refs)

78-3460 **Cell-mediated Anti-Tumour-associated Immunity in Patients with Genitourinary Malignancy: Carcinoma of the Urinary Bladder (Meeting Abstract).** (Eng) Guinan, P. D. (Cook County Hosp., Chicago, IL); Bhatti, R. A.; Ablin, R. J. *Fed Proc* 37(3): 412; 1978. (no refs)

78-3461 **Augmentation of Immune Response Against a Weakly Immunogenic Fibrosarcoma (T241) and Its Effect on Tumor Growth and Pulmonary Metastases (Meeting Abstract).** (Eng) Gautam, S. C. (Cleveland Clinic, Cleveland, OH, 44106). *Proc Am Assoc Cancer Res* 19: 98; 1978. (no refs)

78-3462 **Glucan: Inhibition of Tumor Growth and Enhancement of Survival in Four Syngeneic Murine Tumor Models (Meeting Abstract).** (Eng) Di Luzio, N. R. (Dept. Physiology, Tulane Univ. Sch. Medicine, New Orleans, LA, 70112); Cook, J. A.; Williams, D. L. *Fed Proc* 37(3): 680; 1978. (no refs)

78-3463 **Regional Lymph Node Phagocytic Activity Inhibition by Mouse and Human Mammary Carcinoma (Meeting Abstract).** (Eng) Mosley, J. G. (Dept. Surgery, Royal Postgraduate Medical Sch., London, England); Agwunobi, T. C.; Shama, D.; Boak, J. L. *Br J Surg* 65(5): 370; 1978. (no refs)

78-3464 **Influence of Serial Transplantation on the Immunological-Clinical Correlates of BALB/cfC3H Mouse Mammary Tumors.** (Eng) Hager, J. C. (Dept. Medicine, Roger Williams General Hosp., 825 Chalkstone Ave., Providence, RI); Miller, F. R.; Heppner, G. H. *Cancer Res* 38(8): 2492-2500; 1978.

An attempt was made to characterize clinically a set of serially transplanted BALB/cfC3H mammary tumors in terms of their latency periods, growth rates, invasiveness, and capacity to metastasize and, simultaneously, to test their ability to induce cell-mediated immunity, serum-blocking factors, and lymphocyte-dependent antibody. Sixty tumors representing up to 10 passage generations of 7 spontaneously arising mammary adenocarcinomas growing sc in BALB/cfC3H mice were used. No significant correlation was observed between any of the clinical characteristics. Cell mediated immunity was associated with slower growth rates and the presence of metastases. Serum-blocking factors were detected more frequently in mice exposed to tumors with shorter latency periods and faster growth rates. They were also detected more frequently with nonmetastatic implants than with tumors capable of metastasizing to the lungs or visceral organs. Lymphocyte-dependent antibody was associated with slow-growing and noninvasive tumors. Thus, different types of immune reactivity are associated with different aspects of clinical behavior. The data were further analyzed to assess the influence of serial transplantation on the behavioral and immunological characteristics of the seven mammary tumor populations. Although the results were illustrative of the principles of progression described previously, there was a marked fluctuation of all seven characteristics from generation to generation. This fluctuation is interpreted in the context of the existence within one tumor cell population of distinct subpopulations of tumor cells heterogeneous with regard to growth and malignant potential. (27 refs)

78-3465 **Clinical and Immunogical Behavior of Serially Transplanted Strain BALB/CfC$_3$H Mouse Mammary Tumors (Meeting Abstract).** (Eng) Hager, J. C. (Dept. Medicine, Roger Williams General Hosp., Providence, RI, 02912); Griswold, D. E.; Heppner, G. H. *Proc Am Assoc Cancer Res* 19: 16; 1978. (no refs)

78-3466 **Cell Mediated Immunity Directed to Breast Cancer Associated Antigens in Family Members of Breast Cancer Patients (Meeting Abstract).** (Eng) Yonemoto, R. H. (City Hope Natl. Medical Center, Duarte, CA, 91010); Waldman, S. R. *Proc Am Assoc Cancer Res* 19: 366; 1978. (no refs)

See also:
*(Rev.): 78-3031, 78-3048, 78-3049.
*(Chem.): 78-3126, 78-3183, 78-3191, 78-3192, 78-3200, 78-3208.
*(Viral): 78-3270, 78-3277, 78-3281, 78-3288, 78-3301, 78-3302, 78-3310, 78-3313, 78-3317, 78-3318, 78-3320, 78-3323, 78-3324, 78-3325, 78-3344, 78-3345, 78-3365, 78-3368.
*(Path.): 78-3466, 78-3478, 78-3482, 78-3483, 78-3484, 78-3513, 78-3522, 78-3543.

8-3467 **A Specific Chromosome Breakpoint Associated with Mouse Plasmacytomas.** (Eng) Shepard, J.
. (Dept. Pathology, Dartmouth Medical Sch., Hanover,
H, 03755); Pettengill, O. S.; Wurster-Hill, D. H.; Sorenson,
. D. *J Natl Cancer Inst* 61(1): 255-258; 1978.

ncultured cells of the near-diploid mouse plasmacytoma
IOPC-31C, which contains 44 chromosomes, were studied
sing chromosome-banding techniques. An unexpected find-
g was the absence of the two markers, derived from a recip-
ocal translocation between chromosomes 12 and 15, that
ere consistently present in three uncultured near-tetraploid
yelomas and also in a diploid mouse myeloma. In the
ear-diploid mouse plasmacytoma, chromosome 15 par-
cipated in a reciprocal translocation with chromosome 6,
orming two different markers (m3 and m5) unique to this
mor. However, the location of the breakpoint in chromo-
ome 15 was the same as that required for the formation of
e reciprocal translocation between chromosomes 12 and 15
 the other tumors. This common breakpoint is likely to be
nportant in the development of mouse plasmacytomas. It
suggested that the relationship of the breakpoint in
hromosome 15 to plasmacytoma in the mouse may be analo-
ous to the relationship between the Philadelphia chromo-
ome and chronic myelogenous leukemia in man. (26 refs)

8-3468 **Chromosomal Aberrations and Morphological Transformation of Hamster Embryonic Cells
nduced by L-Tryptophan Pyrolyzate In Vitro.** (Eng) Tsuda,
I. (Biological Res. Center, Japan Tobacco and Salt Public
orp., Hatano, Kanagawa-ken, 257, Japan); Kato, K.; Mat-
umoto, T.; Yoshida, D.; Mizusaki, S. *Mutat Res* 49(1): 145-
48; 1978.

he ability of the pyrolyzate of L-tryptophan (TP) to induce
hromosomal aberrations and morphological transformation
 Syrian hamster embryo cells was determined. Primary or
econdary cultures were exposed for 24 hr to TP concentra-
ions of 1-100 μg/ml and assayed for chromosomal aberra-
ions. Because of cytotoxicity, metaphase cells were not de-
ected at TP concentrations > 75 μg/ml. At 30 and
0 μg/ml, a significant increase in aberrations was
bserved, with breaks and exchanges being the most
requent changes. Polyploidy and diplochromosomes
ere often seen with 50 μg/ml. Exposure of cells to
0, 30, or 50 μg/ml significantly enhanced the fre-
uency of morphological transformation. The mor-
hology of the transformed cells was indistinguishable from that of cells treated with N-methyl-N'-nitro-N-nitrosoguanidine. The health hazard resulting from environmental contamination by pyrolysis products should be investigated. (10 refs)

78-3469 **Mutation in Cultures Human Lymphoid Lines (Meeting Abstract).** (Eng) Bennett, S. (Div. Genetics, Coll. Physicians and Surgeons, Columbia Univ., New York, NY, 10032); Bloom, A.; Nakamura, F.; Spence, S. *Mutat Res* 53(1): 120; 1978. (no refs)

78-3470 **Chromosome Studies of Trichloroethylene Workers.** (Ger) Konietzko, H. (Institut fur Arbeitsmedizin der Universitat, Wilhelmstrasse 27, D-7400 Tubingen, W. Germany); Haberlandt, W.; Heilbronner, H.; Reill, G.; Weichardt, H. *Arch Toxicol (Berl)* 40(3): 201-206; 1978.

On the basis of lymphocyte cultures, chromosome studies were carried out on 28 employees involved in the purification of trichloroethylene (TCE) who were exposed from 1 to 21 yr to various concentrations of TCE. Nine of these subjects showed elevated numbers of hypodiploid cells, but otherwise they had normal karyotypes; one subject had the karyotype 47,XY,+mar (small metacentric extrachromosome). For these nine employees, it was found that the significantly higher chromosome aberration rate correlated with TCE concentration level but not with duration of exposure. (9 refs)

78-3471 **Chromosomal Aberrations in Heavy Smokers.** (Eng) Obe, G. (Institut fur Genetik, Freie Universitat Berlin, Arnimallee 5-7, D-1000 Berlin 33, W. Germany); Herha, J. *Hum Genet* 41(3): 259-263; 1978.

Lymphocyte chromosomes from 20 heavy smokers and 71 controls were examined after 48-hr culture for the frequency of dicentric chromosomes, ring chromosomes, and chromatid translocations. There was significant excess of chromosome and chromatid aberrations among the smokers. No chromosome exchanges were noted in 5/20, 2 of whom allegedly did not inhale. (21 refs)

78-3472 Changes in Shape and Orientation of Cell Nuclei due to Neoplastic Transformation. (Rus) Samoilov, V. I. (State Univ., Moscow, USSR); Rovenskii, Iu. A.; Slavnaia, I. L.; Slovachevskii, M. S. *Tsitologiia* 20(3): 321-328; 1978.

The ability to elongate and the contact orientation reaction of cell nuclei were studied in nine fibroblastlike transformed cell lines cultured on curved and flat polymer surfaces with radii of 333, 75, or 61 μm. The cell lines tested included a murine line L transformed by methylcholanthrene in vitro, murine lines C40 and C-khim (sarcomas induced by implantation of plastic material), murine line M22 transformed by simian virus 40 (SV40) virus in vitro, Syrian hamster cell line 20/90 transformed by SV40 virus in vitro, Syrian hamster cell line KhEK-40 transformed by cytomegalovirus in vitro, Syrian hamster cell line KP transformed by polyoma virus in vitro, rat cell line K2 transformed spontaneously in vitro, and human cell line ChETR transformed by SV40 virus in vitro. Normal embryonic fibroblasts of the respective species were used as controls. The elongation of the cell nuclei was reduced in the human, rat, Syrian hamster, and one of the murine cell lines cultured on flat substrate, compared with the corresponding controls. The elongation of the nuclei of the transformed cells failed to increase on the curved surface during the first 24 hr of culture, but a slight increase was observed during the following 24 hr. Contact orientation, ie, as the deviation of the orientation of the long axes of the cell nuclei from that of the axis of the cylinder, was absent or considerably reduced in the transformed cell lines, compared with the normal fibroblasts. (9 refs)

78-3473 Membrane Alterations During the Carcinogenic Process. (Eng) Walborg, E. F. (Science Park Res. Div., Univ. Texas System Cancer Center, Smithville, TX); Starling, J. J.; Davis, E. M.; Glenny, J. R.; Hixson, D. C. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 26 pp.; 1978. (no refs)

78-3474 Chromosome Changes (Trisomies #15 and 17) Associated with Tumor Progression in Leukemias Induced by Radiation Leukemia Virus. (Eng) Wiener, F. (Dept. Tumor Biology, Karolinska Institutet, 104 01 Stockholm 60, Sweden); Ohno, S.; Spira, J.; Haran-Ghera, N.; Klein, G. *J Natl Cancer Inst* 61(1): 227-237; 1978.

Chromosome changes associated with tumor progression in leukemias induced by radiation leukemia virus (RadLV) were investigated in C57BL/6 mice. Males were inoculated by various routes with RadLV variant-induced preleukemia a
leukemia cells from female inbred C57BL/6 mice. Since
donors were females and all recipients were males, the s
chromosome complements (XX and XY) were used to disti
guish the preleukemia and leukemia cells from those of h
origin. G-banding analyses revealed that $>50\%$ of the mal
that developed leukemia possessed tumor stem lines of
chromosomes with a trisomy of chromosome 15 (5/14 mi
that had received dependent RadLV-induced preleukem
cells and 12/14 mice that had received autonomo
RadLV-induced preleukemia cells). In animals inoculat
with overt leukemia cells and in which tumor progressi
occurred, G-banding analyses revealed an additional trisom
of chromosome 17. The cytogenetic evidence strongly su
gests that diploid preleukemia cells injected into syngene
recipients gain an additional chromosome 15 in the first sta
of leukemia development (during their transformation in
fully autonomous tumor cells). The results also indicate th
in some animals inoculated with overt leukemia cells, pr
sumably with a chromosome constitution of 41XX and tris
my of chromosome 15, leukemia progression is mark
cytogenetically by the further gain of genetic material in t
form of trisomy of chromosome 17. At present, the signi
cance of this second trisomy cannot be assessed. (33 refs)

**78-3475 Conversion of Polycythemia vera to Refractor
Anemia with Hyperplastic Bone Marrow.** (En
Mitrou, P. S. (Zentrum der Inneren Medizin, Abteilung f
Hamatologie, Johann Wolfgang Goethe-Universitat, The
dor-Stern-Kai, D-6000 Frankfurt am Main 70, W. Ge
many); Fischer, M.; Krehan, L.; Martin, H. *Blut* 36(1): 41-4
1978.

Five years after his last chemotherapeutic treatment (busu
fan) for polycythemia vera, a 46-yr-old man developed pa
cytopenia or refractory anemia with hyperplastic bone ma
row. The morphologic changes and the presence of blast cel
in bone marrow and peripheral blood suggested a prele
kemia state. The patient died within a year fro
bronchopneumonia. The nomenclature associated wi
this condition (pancytopenia, chronic erythroleukem
preleukemia) is not uniform, but the morpholog
findings are virtually identical. Some patients su
sequently develop acute leukemia. The prognosis
refractory anemia with hyperplastic bone marro
following polycythemia vera is, independent of the su
sequent acute leukemia, invariably terminal. (14 re

**78-3476 A Fatal Myeloproliferative Syndrome in
Family with Thrombocytopenia and Platel
Dysfunction.** (Eng) Luddy, R. E. (Dept. Pediatrics, Uni
Maryland Sch. Medicine, 22 S. Greene St., Baltimore, M
21201); Champion, L. A.; Schwartz, A. D. *Cancer* 41(5
1959-1963; 1978.

A family is described in which three children died of a myeloproliferative syndrome following a lifelong history of thrombocytopenia and a bleeding disorder. Two of the children had a disorder resembling juvenile chronic myelogenous leukemia and the third had acute monocytic leukemia. Hematologic abnormalities in the mother and five other siblings included thrombocytopenia, abnormal platelet function, elevated Hb F or serum vitamin B_{12} concentrations, and low WBC alkaline phosphatase scores. These leukemia-associated abnormalities in a family with multiple cases of nonlymphocytic leukemia appear to represent a previous undescribed familial myeloproliferative syndrome. (11 refs)

78-3477 **Thirteen Cases of Leukemia in a Family.** (Eng) Gunz, F. W. (Dept. Medical Res., Kanematsu Memorial Inst., Sydney Hosp., Sydney, New South Wales 2000, Australia); Gunz, J. P.; Vincent, P. C.; Bergin, M.; Johnson, F. L.; Bashir, H.; Kirk, R. L. *J Natl Cancer Inst* 60(6): 1243-1250; 1978.

Thirteen cases of leukemia, 12 of them acute, occurred in three generations of a family comprising 293 members. Individual cases could not be linked to the possession of any of a range of genetic markers. Cytogenetic studies showed no constitutional chromosome abnormalities. Preliminary results of virologic studies suggested the presence of oncornaviruses in at least one leukemic patient whose DNA contained sequences related to the RNA genome of baboon endogenous virus. This aggregation of leukemia cases likely resulted from a genetic, probably polygenic, predisposition in association with the activity of leukemogenic factors whose nature remains to be clearly defined. (21 refs)

78-3478 **Acute Leukemia in a Kidney Transplant Patient Treated with Immunosuppressants.** (Fre) Olmer, M. (Service de Nephrologie, Hopital de la Conception, 136, rue Saint-Pierre, F 13335 Marseille Cedex 4, France); Robert, A. *Nouv Presse Med* 7(1): 49; 1978.

Acute leukemia developed in 44-yr-old man 3 yr after he received a kidney transplant accompanied by treatment with immunosuppressants. The immunosuppressive therapy consisted of antilymphocyte serum for 15 days after the transplant and total doses of 4.5 g mercaptopurine and 57 g azathioprine. The patient was maintained on cortisone because of evidence of transplant rejection. Petechial purpura appeared suddenly, and a hematogram showed anemia, 2,500/mm^3 WBC, 10% polynuclear neutrophils, and the presence of blast cells. A myelogram showed a massive infiltration of leukoblasts (60%), most of which were monocyte precursors; the others were immature granulocytes. The patient died 9 days later, and autopsy confirmed the presence of leukemia with widespread hemorrhage. There was no history of contact with a chemical carcinogen, and serological tests for viral infection were negative except for the persistence of the Australia antigen. Possible mechanisms for the development of the leukemia in this patient are discussed. (6 refs)

78-3479 **Leukemia in Identical Twins.** (Eng) Fujimoto, T. (Dept. Pediatrics, Kyushu Univ. Sch. Med., Fukuoka, Japan); Hasegawa, K.; Take, H.; Miyazaki, S.; Sasaki, K.; Kishida, K.; Goya, N. *Acta Haematol Jpn* 41(1): 57-62; 1978.

Cases of concordant acute lymphocytic leukemia occurring in 3.5-yr-old identical twin boys are reported. Only one other report of concordant acute leukemia in identical twins has appeared in the Japanese literature. (17 refs)

78-3480 **Hibernoma: A Possible Model of Brown Fat Histogenesis.** (Eng) Dardick, I. (Dept. Lab. Medicine, Ottawa Civic Hosp., 1053 Carling Ave., Ottawa, Ontario K1Y 4E9, Canada). *Hum Pathol* 9(3): 321-329; 1978.

Hibernomas are composed of cells that resemble those of brown adipose tissue. A surgically resected hibernoma is described that contained cells at all stages of maturation from immature, relatively lipid-free cells to multiloculated and, finally, uniloculated adipocytes. The ultrastructural features of the adipocytes were compared with the previously reported features of differentiating preadipocytes and lipid-depleted mature adipocytes from human white adipose tissue. This comparison confirmed the existence of distinct mitochondrial and cytoplasmic membrane component differences at all developmental stages. Thus, brown and white adipose tissue may be two distinct histologic types. (34 refs)

78-3481 **Malignant Histiocytomas.** (Eng) Langsam, L. B. (Dept. Surgery, St. Vincent Charity Hosp., 2351 E. 22nd St., Cleveland, OH, 44115); Fine, G.; Ponka, J. L. *Arch Surg* 113(4): 473-476; 1978.

A review of 16 patients (13 men, 3 women aged 12-75 yr) with malignant histiocytomas in the extremities, head, chest wall, retroperitoneum, lung, spermatic cord, and lower abdomen is presented. Various methods of treatment were used; local recurrence developed in 29% treated with local excision. Ten patients are alive and well, two are alive with metastatic disease. These heterogeneous diseases probably arise from the histiocyte. (10 refs)

78-3482 **Possible Reversion of Malignancy In Vivo.** (Eng) Revilla, M. C. (Dept. Investigacion en Medicina Experimental, Lab. Biomembranas, Mexico DF, Mexico); Gonzalez, M. T.; Balderas, M. Z.; Romero, G. *Nature* 272(5652): 454-455; 1978. (5 refs)

The L5178Y lymphoma line, which is stable when passaged in syngeneic BALB/c mice and has a diploid chromosome number of 39 ± 1, was used to study the reversion of malignancy in vivo. When serially allotransplanted for up to eight passages in CD1 mice, the cells lost their tumorigenicity; they did not induce tumors when transplanted to BALB/c mice. The reversion state was associated with an increase in chromosome number from diploid to subtetraploid or tetraploid between the first and fifth transplantations, with a decrease to diploid or subdiploid between the sixth and eighth passages. The stability of the reverted cells is attributed to the balance between chromosome factors that determine the expression (E) and suppression (S) of transformed properties; ie, an excess of S over E. (5 refs)

78-3483 **Clonal Origin of a T Cell Lymphoproliferative Malignancy.** (Eng) Stryckmans, P. A. (Institut Jules Bordet, 1, rue Heger Bordet, Brussels, Belgium); Debusscher, L.; Heyder-Bruckner, C.; Heimann, R.; Mandelbaum, I. M.; Wybran, J. *Blood* 52(1): 69-76; 1978.

An attempt was made to determine the clonal origin of the circulating neoplastic T-cells of a 34-yr-old black woman with a T-cell lymphoproliferative malignancy and heterozygosity at the X Cap "ex" chromosome-linked locus for glucose-6-phosphate dehydrogenase (G-6-PD) isoenzymes. Light and electron microscopy revealed the lymphoid nature of the circulating tumor cells, which were identified as T cells by an erythrocyte (E) rosette assay. When the patient's peripheral WBC count reached 95 x 10^9/liter, 88% of the cells formed E rosettes. Electrophoresis showed equal staining of G-6-PD isoenzymes (A and B) in the RBC, the isolated peripheral blood mononuclear cells, the polymononuclear cells, and the T-cell-poor lymphocyte population. In contrast, heavy staining of G-6-PD-B and only traces of G-6-PD-A could be seen in the T-cell-rich population. After treatment of the patient, equal staining of both isoenzymes was seen in all cellular populations, including the T-cell-rich population. The observation that the G-6-PD was almost exclusively of the B type in the circulating neoplastic cells strongly suggests the monoclonal origin of this T-lymphocyte-derived malignancy. (29 refs)

78-3484 **Immune Function in Healthy Relatives of Patients with Malignant Disease.** (Eng) Dworsky, R. (Edmondson Res. Building, 1840 N. Soto St., Los Angeles, CA 90032); Baptista, J.; Parker, J.; Chandor, S.; Noble, G.; Herrmann, K.; Henle, W.; Henderson, B. *J Natl Cancer Inst* 60(1): 27-30; 1978.

Measures of humoral and cell-mediated immunity were compared in healthy members of 18 different families grouped according to their history of neoplasia. There were four groups. Group 1 had 22 members of 5 families with 2 or more cases of lymphoproliferative disease in first- and/or second-degree relatives, Group 2 had 11 members of 6 families in which the propositus had a lymphoreticular neoplasm and other cancers occurred in first- and/or second-degree relatives, Group 3 had 18 members of 7 families with no known history of cancer, and Group 4 consisted of 7 laboratory personnel and 4 spouses of participating families. Group 1 had a significantly lower response to concanavalin A (Con A) than Group 2 or 4. The difference in response to *Candida albicans* antigen between Groups 1 and 3 were also significant. Group 1 had a significantly higher IgM level; IgM was positively correlated to WBC counts. A negative correlation was found between Con A responses and both IgM level and Epstein-Barr virus-viral capsid antigen (EBV-VCA) titers. However, IgM and EBV-VCA were not strongly correlated with each other. EBV-VCA titers and measles antibody titers were correlated. EBV-VCA titers showed an inverse correlation with responses to phytohemagglutinin A and Con A mitogens. These findings indicate that healthy members of families with multiple cases of lymphoma may have decreased responses to mitogen stimulation compared with members of families with other forms of cancer. (21 refs.)

78-3485 **Melanogenesis in Genetically Determined Pigment Cell Tumors of Platyfish and Platyfish-Swordtail Hybrids: Correlation Between Tyrosinase Activity and Degree of Malignancy.** (Eng) Vielkind, U. (Genetisches Institut, Justus-Liebig-Universitat Giessen, Heinrich-Buff-Ring 58-62, D-6300 Lahn-Giessen, W. Germany); Schlage, W.; Anders, F. *Z Krebsforsch* 90(3): 285-299; 1977.

Tyrosinase activity was determined in the soluble and particulate fractions of tumors in platyfish (*Platypoecilus maculatus*) and platyfish-swordtail (*Xiphophorus helleri guentheri*) hybrids. A close correlation was observed between the level of tyrosinase activity and the degree of malignancy. Spot patterns consisting of completely differentiated (benign) tumor gene (Tu)-transformed cells showed no tyrosinase activity. Premelanomas containing a few incompletely differentiated (malignant) Tu-transformed cells in addition to many differentiated ones showed moderate tyrosinase activities. Melanomas which contained increasing numbers of incompletely differentiated cells with increasing growth rates showed high to extremely high tyrosinase activities. Thus, tyrosinase levels can be used as indicators of both the degree of differentiation and degree of malignancy of neoplastically transformed pigment cells. (49 refs)

78-3486 **Actual Doubling Time Values of Pulmonary Metastases from Malignant Melanoma.** (Eng)

Plesnicar, S. (Inst. Oncology, Vrazov trg 4, 61000 Ljubljana, Yugoslavia); Klanjscek, G.; Modic, S. *Aust NZ J Surg* 48(1): 23-25; 1978.

The doubling times of small- and large-diameter pulmonary metastases were compared in 18 patients (9 men, 9 women 24-89 yr old) with pulmonary metastases from histologically proved malignant melanoma. From chest radiograms, 81 metastases were measured 51 consecutive times and 120 doubling-time values were obtained. These values were calculated according to the change in tumor diameter. Metastases with diameters ≤15.0 mm grew significantly faster (mean doubling time 24.9 days) than metastases with diameters > 15.0 mm (mean doubling time 51.0 days). Eleven values were > 100 days (mean: 182.2 days); the diameters of the metastases ranged from 100 to 397.8 mm. The group rate appears to depend on the size of the metastatic growth, since with increasing volume of metastases the growth rates seemed to decrease. (11 refs)

78-3487 **Isolation and Metastatic Properties of Detachment Variants of B16 Melanoma Cells.** (Eng) Briles, E. B. (Div. Hematology, Washington Univ. Sch. Medicine, Box 8125, 660 South Euclid, St. Louis, MO, 63110); Kornfeld, S. *J Natl Cancer Inst* 60(6): 1217-1222; 1978.

Variant clones of B16 melanoma cells (B16F1 and B16F10 lines) were studied for the relationship between their in vitro adhesive properties and their in vivo tumor-related properties. Variant clones were isolated that detached from plastic tissue culture dishes either more or less readily than the parent lines upon treatment with EDTA or ethyleneglycol-bis(b-aminoethylether)-N,N-tetraacetic acid. Cells that detached more readily were spindlier in culture than parent cells, covered less surface area per cell, and formed fewer pulmonary tumors when injected iv into syngeneic C57BL/6J mice. Cells that detached less readily were flatter in culture and formed more pulmonary tumors when injected iv than the parent cells. After detachment with EDTA, the cells reattached to culture dishes at the same rate. This result is discussed in terms of the concept that initial attachment and subsequent establishment of cell-to-substrate adhesions (ie, cell spreading) are independent events. (21 refs)

78-3488 **Effects of Enzymes and Polyelectrolytes on the Aggregation Properties of Metastatic Melanoma Cells (Meeting Abstract).** (Eng) Madyastha, K. R. (Div. Medical Oncology, Medical Coll. Virginia, Richmond, VA, 28298); Phondke, G. P.; Madyastha, P. R.; Barth, R. F. *Proc Am Assoc Cancer Res* 19: 221; 1978. (no refs)

78-3489 **A Case of Oculocutaneous Albinism Complicated by Malignant Melanoma.** (Jpn) Yoshioka, J. (Dept. Dermatology, Nara Medical Univ., Kashihara, Japan); Kitamura, W. *Jpn J Clin Dermatol* 32(5): 355-364; 1978.

A 32-yr-old woman with oculocutaneous albinism, an autosomal recessive condition, developed a malignant melanoma. She had been treated with dopa-R (dihydroxyphenylalanine-R) and melanin for the albinism. Types II and III melanosomes were found among the tumor cells. Eight literature cases of benign and malignant tumors developing in human albinos are also reported. (24 refs)

78-3490 **Amyloidoma: A Clinicopathologic Study of Four Cases (Meeting Abstract).** (Eng) Lipper, S. (Dept. Pathology, Univ. Cape Town Medical Sch., Cape Town, South Africa); Kahn, L. B. *Lab Invest* 38(3): 354-355; 1978. (no refs)

78-3491 **Precursor Lesions in Familial Melanoma: A New Genetic Preneoplastic Syndrome.** (Eng) Reimer, R. R. (Dept. Hematology-Oncology, Cleveland Clinic Foundation, 9500 Euclid Ave., Cleveland, OH, 44106); Clark, W. H.; Greene, M. H.; Ainsworth, A. M.; Fraumeni, J. F. *JAMA* 239(8): 744-746; 1978.

Examination of seven melanoma-prone families revealed that 18/20 melanoma patients and 24/43 first-degree relatives had prominent pigmented cutaneous lesions. The lesions were flat, frequently > 10 mm in diameter, and haphazardly colored. The number of lesions ranged from 10 to > 100 per person. They were most common over the "horse collar" area of the trunk and upper extremities, but they were also present on unexposed areas. Examination of six persons with this syndrome showed level 1 or 2 superficial spreading melanomas arising from one of the lesions. The detection rate of melanoma among first-degree relatives with the syndrome was 14%. This syndrome appears to represent an autosomal dominant trait, and it may serve as a cutaneous marker to identify persons at high risk for melanoma. (12 refs)

78-3492 **Osteogenesis and Chondrogenesis in Transplants of Dunn and Ridgway Osteosarcoma Cell Cultures.** (Eng) Hanamura, H. (Dept. Surgery, Div. Orthopedic Surgery, UCLA Medical Sch., Los Angeles, CA); Urist, M. R. *Am J Pathol* 91(2): 277-298; 1978.

The effects of Dunn and Ridgway osteosarcoma cells and cell products on the genetic program of normal connective tissue cells were determined. Dunn osteosarcomas synthesized 2 times more alkaline phosphatase than Ridgway osteosarcomas, 3 times more than HeLa cells, and 4-5 times more than rat or mouse fibroblast cell cultures. Implants of killed freeze-dried Dunn cell cultures into the thigh muscles of mice were resorbed and replaced by normal cartilage, bone, and bone marrow tissue. Implants of freeze-dried Ridgway cells were resorbed and replaced by fibrous tissue only. Outgrowths of normal muscle septum connective tissue cells into the stroma of Ridgway tumors differentiated into fibrous tissue. Cultures of either tumor on a substratum of bone matrix stroma prepared from normal bone proliferated, assumed a spherical shape, and perpetuated the transformed osteoblast-like cell without forming attachments or adapting to the contour of the substratum. Outgrowths of muscle mesenchymal cells on the Dunn tumor stroma differentiated into cartilage. Dunn osteosarcoma cell cultures proliferated on the inside and produced deposits of normal bone (not tumorous bone) on the outside of diffusion chambers. Killed freeze-dried cell cultures produced transfilter deposits of normal bone and bone marrow, but in significantly lower quantity. Outgrowths of muscle connective tissue, on a substratum of cellulose acetate, differentiated into cartilage when cell-free Dunn stroma was present under the organ culture grid. Tumorigenesis and normal cartilage and bone morphodifferentiation were antithetic; however, tumor cells transferred a bone morphogen similar to the bone morphogenetic protein (BMP) of normal bone matrix. BMP recruited mesenchymal cells to proliferate and differentiate into cartilage and bone. (32 refs)

78-3493 **Separation of Cells from a Murine Fibrosarcoma on the Basis of Size. II. Differential Effects of Cell Size and Age on Lung Retention and Colony Formation in Normal and Preconditioned Mice.** (Eng) Grdina, D. J. (Section Experimental Radiotherapy, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., 6723 Bertner Ave., Houston, TX, 77030); Peters, L. J.; Jones, S.; Chan, E. *J Natl Cancer Inst* 61(1): 215-220; 1978.

The influence of cell size and cell age on the intrinsic clonogenicity and retention efficiency of tumor cells in relation to lung colony formation (LCF) was investigated in female C3Hf/Bu mice inoculated iv with fibrosarcoma cells (2 x 10^5 viable cells). Three different lung colony assays were used to study the influence of these parameters in normal untreated mice and in mice in which the efficiency of the assay was increased by pretreatment (24 hr before inoculation) with cyclophosphamide (CP) (250 mg/kg, sc) or whole-body irradiation (WBI: 1,000 rads). Mice receiving WBI also received 10^6 heavily irradiated cells (10,000 rads) in the viable cell inoculum. In untreated mice, cell retention and LCF were positively correlated, with the larger cells, predominantly in G_2, being the most efficiently retained and yielding the most lung colonies. No effect of cell size or relative position in the cell cycle on LCF was seen in preconditioned mice. The efficiency of LCF was increased by 10- and 30-fold in CP and WBI-treated mice, respectively. Cell arrest patterns correlated with LCF in WBI-treated mice for all cell sizes, but no such correlation was found for the smallest cells (G_1 phase) in CP-pretreated mice. It is concluded that the intrinsic clonogenic ability of a fibrosarcoma cell does not depend on its size or its relative position in the cell cycle. Inasmuch as the metastatic ability of a cell is a function of both its intrinsic clonogenic ability and its relationship to host-modifying factors, the parameters of cell size and age can, however, become important determinants in metastasis formation, depending on the conditions imposed on the host. (15 refs)

78-3494 **Pathology of Immunologic Regression of Tumor Metastases in the Lymph Nodes.** (Eng) Kodama, T. (Lab. Pathology, Cancer Inst., Hokkaido Univ. Sch. Medicine, Kita-15-jo, Nishi-7-chome, Kita-ku, Sapporo 060, Japan). *Gann Monogr Cancer Res* (20): 83-91; 1977.

The pathology of a transplantable lymphosarcoma (WGT4) induced by Gross virus in WKA/Mk rats was investigated. Injection of 5 x 10^6 tumor cells into the right foot of Gross-tolerant rats resulted in death from tumor growth in approx 16 days. However, injection of tumor cells into normal rats showed temporary growth followed by regression. All WGT-4 tumors regressed completely in an av of 16 days. The right popliteal, lumbar, and inguinal lymph nodes draining the tumor site in Gross-tolerant rats enlarged with tumor growth and reached a max size at death. Lymph nodes from normal rats reached a max size on day 8 and returned to normal after approx 16 days. By day 6 after tumor cell injection, the regional lymph nodes were completely occupied by tumor in both Gross-tolerant and normal rats; however, lymph node metastases disappeared in normal rats by day 8. Histopathologically, infiltration and proliferation of lymphoid cells, reticulum cells, and fibrocytes occurred in the regressing metastatic tumor and in the regressing transplanted tumor in the foot. Uninfected rat lymph nodes, in which metastases regressed, had a 'starry sky' appearance. These findings suggest that regression of transplanted or metastatic tumors in syngeneic rats is due to an immunological reaction by the host and that immunological factors may be responsible for the 'starry sky' appearance of the node. (19 refs)

78-3495 **Regulation of Development of Embryo-derived Teratocarcinomas (Meeting Abstract).** (Eng) Damjanov, I. (Hahnemann Medical Coll., Philadelphia, PA, 19102); Caldwell, J. W. *Lab Invest* 38(3): 384; 1978. (no refs)

78-3496 **Isolation and Characterization of Chromatin from Embryoid Bodies and Teratomas: Role of Nonhistone Chromosomal Proteins in Differentiation (Meeting Abstract).** (Eng) Carter, D. B. (Lab. Environmental Toxicology, NIEHS, Research Triangle Park, NC, 27709). *Environ Health Perspect* 20: 238-239; 1977. (no refs)

78-3497 **Histogenesis of Dermatofibrosarcoma Protuberans. An Ultrastructural Study.** (Eng) Alguacil-Garcia, A. (Section of Publications, Mayo Clinic and Mayo Foundation, Rochester, MN 55901); Unni, K. K.; Goellner, J. R. *Am J Clin Pathol* 69(4): 427-434; 1978.

Three original and two recurrent lesions of dermatofibrosarcoma protuberans (DFP) from five patients were studied ultrastructurally. The basic tumor cell was spindle-shaped, with long slender cytoplasmic projections (CP). The nuclei were usually fusiform, and they showed some convolutions and indentations. In cross section, the nuclei were ovoid, or less frequently, very lobulated or cerebriform. The cytoplasmic membrane was smooth. The cytoplasm contained scant profiles of flat rough endoplasmic reticulum, a few vesicles of smooth endoplasmic reticulum, mitochondria, and free ribosomes and polyribosomes. Variable numbers of tumor cells showed fragments of basal lamina, small focal cytoplasmic densities peripherally, and, rarely, maculae adherens-type intercellular junctions. The latter were usually seen between the CP. The tumor cells and their CP were often arranged in a circular fashion around the CP of other tumor cells. This sheath-like arrangement was very characteristic of DFP. The tumor cells had a strong resemblance to pericytes and perineural cells, as well as marked ultrastructural similarities to neurofibroma. These findings suggest that DFP arises from a primitive fibroblastic cell with a sheath-forming cell differentiation simulating perineural cells. (37 refs)

78-3498 **Xeroderma Pigmentosum. Clinical Course and Ultrastructure of Tumor-free Skin Regions Not Exposed to Sun.** (Ger) Haustein, U. F. (Hautklinik des Bereichs Medizin, Karl-Marx-Universitat, Liebigstrasse 21, DDR-701 Leipzig, E. Germany). *Dermatol Monatsschr* 164(4): 255-270; 1978.

In a 29-yr-old woman with xeroderma pigmentosum (XP), the poikilodermatic, precancerous stage of the disease was observed in parallel with the neoplastic stage. Since childhood, the patient had developed many epidermal, mesenchymal, and pigment-forming tumors that had been treated surgically, radiologically, or with ointments containing 5-fluorouracil. Electron microscopy of tumor-free skin that had not been exposed to the sun revealed the following: (1) distinct changes of the keratinocytes and basal cells; (2) polymorphism as well as elevated and premature synthesis of (pre)melanosomes; and (3) vacuolization of the upper corium and changes resembling elastosis senilis in fibroblasts with an otherwise normal structure. These changes are comparable to those observed after UV irradiation. Elevated melanin synthesis may be an attempt to compensate for the UV effect by morphologic changes parallel to the development of lentigo maligna. (53 refs)

78-3499 **Histologically Proved Basal Cell Carcinomas in Identical Twins.** (Eng) Liddell, K. (District General Hosp., Eastbourne, E. Sussex BN21 2UD, England). *Br Med J* 2(6130): 97; 1978.

Identical twins (65 yr old) presented with at virtually the same time with erythematous, slightly crusted lesions of the back, which were diagnosed histologically to be superficial basal cell carcinoma. Common epidemiological factors (possibly, arsenical salts taken in tonics as children) and inherited biochemical defect are suggested as possible causes. (4 refs)

78-3500 **Multiple Bowen's Disease Observed in Former Workers of a Poison Gas Factory in Japan, with Special Reference to Mustard Gas Exposure.** (Eng) Inada, S. (Dept. Dermatology, Hiroshima Univ. Sch. Medicine, Kasumi, Hiroshima 734, Japan); Hiragun, K.; Seo, K.; Yamura, T. *Jpn J Dermatol B* 5(2): 49-60; 1978.

Five cases of multiple Bowen's disease in former employees of a poison gas factory in Okunojima, Japan, were studied. The patients, all men, ranged in age from 63 to 70 yr (mean, 66 yr), and they had been employed in the production of mustard gas for 3-15.3 yr (mean, 9.3 yr). The period between commencement of employment in the factory and diagnosis of Bowen's disease ranged from 31 to 46 yr (mean, 39.2 yr). A total of 23 lesions of Bowen's disease were identified on the five patients: 10 on the trunk, 3 on the upper extremities, and 10 on the lower extremities. Although the workers wore protective clothing, the mustard gas permeated the clothing and apparently dissolved in the sweat to affect the skin. In addition to the Bowen's lesions, all patients had numerous pigmented and depigmented spots and hyperkeratotic papular eruptions on the skin. These data suggest that the Bowen's disease in these workers was caused by exposure to mustard gas, although there are no literature reports of this association. (35 refs)

78-3501 **Mycosis Fungoides: Can Continuous Antigen Stimulation Be Its Cause?** (Fre) Bazex, J. (Clinique des Maladies Cutanees et Syphilitiques, Hopital Purpan, Place du Dr. Baylac, F 31052 Toulouse Cedex, France). *Nouv Presse Med* 22(16): 1410; 1978.

Three patients developed mycosis fungoides several years after the implantation of metallic bone prostheses. The symptoms disappeared a few weeks after the implants were removed. The findings suggest continuous antigen (metal, in this case) stimulation as the possible cause of mycosis fungoides in these patients. (4 refs)

78-3502 **Malignant Meningioma: Ultrastructure and Observations on Histogenesis.** (Eng) Pietruszka, M. (Dept. Pathology, Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA); Salazar, H.; Pena, C. *Pathology* 10(2): 169-173; 1978.

A malignant meningioma was studied by light and electron microscopy. Histologically, the tumor was composed predominantly of spindle shaped cells arranged in interlacing bundles with numerous mitoses and frequent nuclear atypia. Tonguelike projections of tumor infiltrated the cerebral cortex, dura mater and blood vessels. Necrotic foci were conspicuous. Ultrastructurally, the main findings included moderate plasma membrane interdigitation, many intracytoplasmic filaments, and junctional attachments of three kinds: desmosomes, hemidesmosomes, and zonulae adherens. These subcellular features are suggestive of an arachnoidal cell origin. (9 refs)

78-3503 **Subependymoma. A Case Report with Ultrastructural Study.** (Eng) Azzarelli, B. (Inst. Pathology, Case Western Reserve Univ., Cleveland, OH 44106); Rekate, H. L.; Roessmann, U. *Acta Neuropathol (Berl)* 40(3): 279-282; 1977.

Light and electron microscopic examination of a subependymoma in a 68-yr-old man showed that it probably arose by replication of mature subependymal tissue. The designation subependymoma is indicative of both its geographic and cytogenetic origin. It should not be designated an astrocytoma or an ependymoma. (9 refs.)

78-3504 **Selection and Altered Properties of Brain-colonising Metastatic Melanoma.** (Eng) Brunson, K. W. (Dept. Developmental and Cell Biology, Univ. California, Irvine, CA, 92717); Beattie, G.; Nicolson, G. L. *Nature* 272(5653): 543-545; 1978.

Sequential in vivo selection for brain-preferring B16 melanoma variant lines yielded cell lines that became more brain-specific with selection. The B16-F1 line (selected once in the lung), yielded a large number of pulmonary tumors, some extrapulmonary tumors, and a few brain tumors. Brain-selected B16-B5 and B16-B10n lines formed larger numbers of brain lesions and few to almost no lung tumors or lesion at other sites. Cell-surface differences between brain-selecte and lung-selected B16 melanoma lines were found when exposed surface proteins were analyzed by lactoperoxidas catalyzed iodination. Autoradiograms made after sodiu dodecyl sulfate-polyacrylamide gel electrophoresis of ^{125}I labeled surface proteins from the B16 lines revealed that th brain-selected lines had a predominance of two exposed surface proteins of mol wt 95,000 (95K) and 100K, compare with the parental B16-F1 line. The degree of exposure of th 95K and 100K proteins correlated with the degree of brai colonization and preference in 5/5 experiments (B16-B10 > B16-B5 > B16-B1 = (approx) B16-F1). Because bot surface proteins were labeled on the B16-F line, th indicates possible differences in the display or quantit of these surface molecules on the brain-colonizing B1 B lines. (36 refs)

78-3505 **Primary Intracranial Tumours in Black and Indian Children, 1960-1975.** (Eng) Quinn, R. J (Dept. Paediatrics and Child Health, Univ. Natal, Durban S. Africa); Scragg, J. N.; Rubidge, C. J. *S Afr Med J* 53(6) 211-213; 1978.

The results of a survey of primary intracranial tumors occurring in children admitted to King Edward VIII Hospital i Durban between 1960-1975 are presented. Of 60 childre identified, 42 were black and 18 were Indian to give respectiv incidences of 0.5 and 2.1/1,000 admissions. Comparison wit the number of children admitted with other malignant diseases showed cerebral tumors to be the most common form of solid neoplasms in children <12 yr of age. Thirty tumor were supratentorial and 29 were infratentorial; the exact primary site could not be established in 1. Histological type wa determined in 49 cases, with astrocytoma (26) and medulloblastoma (12) being the most common tumors. The age distribution, sex ratio, and tumor site of these patients were simila to those found in recent studies of other racial groups. (8 refs)

78-3506 **Medulloblastoma: Example of Late Adult Onset with Statistical Correlation Relative to Etiology.** (Eng) Seitz, W. S. (Inst. Medical Analysis, Bockgasse 4.9.9, A-1120 Vienna, Austria); Operschall, E. J. *Radiol Clin (Basel)* 47(4): 295-309; 1978.

A unique case of advanced adult onset of medulloblastoma in a 88-yr-old Chinese woman is reported, and the literature on 33 histologically verified cases of medulloblastoma is reviewed. The patient underwent craniotomy and excision of a posterior fossa cerebellar tumor that demonstrated ovoid or elongated tumor cells with hyperchromatic nuclei in a configuration suggesting a classical medulloblastoma without typical rosette formation. The patient died 8 mo after surgery. A review of the literature and data compiled by the California

Tumor Registry reveal that this case represents the oldest example of medulloblastoma, which appears to occur most frequently around the age of 5 yr. The male to female ratio is 2.04 for all age groups. Since 1971, the av survival has increased from 25 to 27 mo, possibly as a result of improved therapeutic techniques. The changing survival expectancy with surviving interval is 10 mo/survival yr. There is a relationship between prognosis and histological type, tumor location, age, and sex. The literature also reveals that there is a trend for adult lateral and child midline development, lending support to the etiological concept of migration of primitive medulloblasts upward and laterally to form the external granular layer. There is a statistically significant tendency for midline tumors to differentiate along classical lines and lateral tumors along desmoplastic lines. (25 refs)

78-3507 **Grave Cranio-cerebral Trauma 30 Years Ago as Cause of the Brain Glioma at the Locus of the Trauma. Particulars of the Case.** (Eng) Mrowka, R. (Klinika Neurochirurgii, Slaskiej Acad. Medicine, ul. Medykow 14, P1-40752 Katowice 6, Poland); Bogunska, C.; Kulesza, J.; Bazowski, P.; Wencel, T. *Zentralbl Neurochir* 39(1): 57-64; 1978.

A glioblastoma multiforme developed in a 50-yr-old man 30 yr after he suffered a craniocerebral trauma caused by the penetration of artillery missile splinters in the left parietal region. A 3-cm bone defect was located directly above the tumor, and there were close cicatrical connections among the dura, brain tissue, and tumor. (18 refs)

78-3508 **Colon Polyps, Sebaceous Cysts, Gastric Polyps, and Malignant Brain Tumor in a Family.** (Eng) Binder, M. K. (Div. Gastroenterology, Dept. Medicine, Harbor General Hosp., 1000 W. Carson St., Torrance, CA, 90509); Zablen, M. A.; Fleischer, D. E.; Sue, D. Y.; Dwyer, R. M.; Hanelin, L. *Am J Dig Dis* 23(5): 460-466; 1978.

A family with a distinct syndrome of adenomatous colonic polyps and sebaceous cysts without osseous or soft-tissue abnormalities is presented. One member had benign gastric polyps limited to the upper half of the stomach and another had a medulloblastoma of the cerebellum. A histologic feature in the colon of the propositus was the presence of atypical epithelium in flat mucosa as well as in the colonic polyps. The atypical epithelium, which was noted after subtotal colectomy, may be the earliest stage of new polyp formation and further malignant potential. The grandfather in this family had colon cancer, and a granduncle had gastric carcinoma. (15 refs)

78-3509 **The Relation of Nervous Elements to Intradermal Nevi. An Electron Microscopic Study.** (Eng) Jurecka, W. (Institut fur Mikromorphologie und Elektronenmikroskopie, Universitat Wien, Alser Strasse 4, A-1090 Vienna, Austria); Lassmann, H.; Gebhart, W. *Arch Dermatol Res* 261(3): 219-230; 1978.

Intratumoral nerves of dermal nevi were investigated by electron microscopy in order to determine the role of neural elements in the development of these tumors. Intratumoral nerves exhibited an increased cellularity, but the number of myelinated fibers was reduced. Types B and C nevus cells were frequently present within the nerve fascicles. Unmyelinated axons were found in close relationship to nevus cells. Schwann cells showed proliferations similar to the early stages of neurofibroma, and they could be differentiated from unpigmented tumor cells by their relation to axons only. These findings indicate that identical cell populations are present within the intratumoral nerves and the tumor itself. Therefore, the theory of a neural component in the histogenesis of intradermal nevi is supported. (26 refs)

78-3510 **Ultrastructural Study of Cellular Relationships in Squamous Cell Carcinomas (Meeting Abstract).** (Eng) Gigoux, C. (Univ. Rochester, Rochester, NY); Johansen, E.; Norante, J. D. *J Dent Res* 57(A): 261; 1978. (no refs)

78-3511 **Distant Metastases in Nasopharyngeal Carcinoma.** (Eng) Khor, T. H. (Dept. Radiotherapy, Singapore General Hosp., Singapore 3); Tan, B. C.; Chua, E. J.; Chia, K. B. *Clin Radiol* 29(1): 27-30; 1978.

The records of 352 consecutive patients with nasopharyngeal carcinoma (NPC) were analyzed to determine the pattern of distant metastases, which were diagnosed on the basis of clinical signs and radiography. Ninety-nine patients (28.1%) developed distant metastases. There were no age or sex differences in risk. Skeletal metastases occurred in 48.5% of metastatic cases. Lung and liver involvement followed at frequencies of 30.3% and 29.3%, respectively. Of the 21 double organ combinations, bone and lung (7 cases) and bone and liver (6 cases) were the most common. The extent of the primary tumor and the cervical lymph node status both exerted a significant influence on the probability of metastases developing. Sixty-five (65.6%) patients with subsequent distant metastases had no local disease clinically, either in the nasopharynx or the cervical lymph nodes. The prognosis of NPC patients with metastases is poor, with a median survival under 4 mo and a mortality rate of 91% within a year of the first metastasis. (12 refs)

78-3512 **Investigations on the Occurrence and Stages of Various Forms of Thyroid Carcinoma in an En-**

demic Goiter Area (Meeting Abstract). (Ger) Lohrs, U. (Munich, W. Germany); Permanetter, W.; Spelsberg, F.; Beitinger, M. *Zentralbl Allg Pathol* 122(1/2): 144; 1978. (no refs)

78-3513 **Association of Thymoma and Thymic Lymphoid Hyperplasia (Meeting Abstract).** (Eng) Madarnas, P. (Dept. Pathology, Univ. Sherbrooke, Sherbrooke, Quebec, Canada). *Lab Invest* 38(3): 355; 1978. (no refs)

78-3514 **Teratomas of the Neck in Adults.** (Eng) Colton, J. J. (Dept. Otorhinolaryngology, Univ. Michigan Hosp., Ann Arbor, MI, 48109); Batsakis, J. G.; Work, W. P. *Arch Otolaryngol* 104(5): 271-272; 1978.

The 10th case of malignant teratoma of the neck in an adult (77-yr-old man) is reported. The clinicopathologic features of these tumors are different in adults and in neonates/infants. The tumors may arise from germ cells and/or displaced multipotential embryonic tissue. A proposed pathogenesis is that tissue about the primitive streak and notochord lose its governing external influences and develop into tumors. The reason for the long dormant period is not known. (7 refs)

78-3515 **Enhancement of the Tumorigenicity of Preneoplastic Mammary Nodule Lines by Enzymatic Dissociation.** (Eng) Medina, D. (Dept. Cell Biology, Baylor Coll. Medicine, Texas Medical Center, Houston, TX, 77030); Shepherd, F.; Gropp, T. *J Natl Cancer Inst* 60(5): 1121-1126; 1978.

The effects of enzymatic dissociation on the tumor potentials were examined in three preneoplastic mammary nodule lines (D1, D2, and C4) isolated from BALB/cCrgl mice. Treatment with collagenase, hyaluronidase, and pronase or with collagenase and hyaluronidase produced high yields of viable single cells that were injected (10^5 cells) into the cleared mammary fat pads of syngeneic BALB/c mice. In 11 experiments, all dissociated nodule cell lines showed a marked increase in tumorigenicity, compared with the same tissues transplanted as 1-mm^3 pieces. The results could not be explained on the basis of differences between the amounts of cells transplanted in the two procedures. In a second series of experiments, normal mammary cells from virgin, pregnant, or lactating mice were mixed in different ratios with 10^5 nodule cells and injected into the mammary fat pads. The presence of the normal cells reversed the marked increase in the tumorigenicity of the enzymatically dissociated nodule cells to a level equal to or less than the tumorigenicity of control transplants (1-mm^3 pieces). The growth of 10^5 mammary tumor cells was not inhibited when the cells were mixed with 3 x 10^5 normal cells and transplanted into the mammary fat pads. These results showed that enzymatic dissociation can increase the tumor potential of preneoplastic mammary nodule lines. They support the hypothesis that nodule cells, but not neoplastic cells, are sensitive to the growth-inhibitory effects of normal mammary cells. (18 refs)

78-3516 **Enhanced Formation of Hyperplastic Alveola Nodules in Mammary Glands of Yellow (Avy/A) Virgin Female (C3H x VY)F-1 Hybrid Mice (Meetin Abstract).** (Eng) Wolff, G. L. (National Center Toxicologica Res., Jefferson, AR, 72079); Medina, D. *Proc Am Assoc Cancer Res* 19: 58; 1978. (no refs)

78-3517 **Intravascular Mucinosis and Thrombosis Accompanying Adenocarcinomas (Meeting Abstract).** (Eng) Pratt-Thomas, H. R. (Dept. Pathology, Medical Univ. South Carolina, Charleston, SC, 29403); Smith, R. M.; McMaster, K. R.; Earlywine, G. R. *Lab Invest* 38(3): 361; 1978. (no refs)

78-3518 **Cytomorphological Grading and Feulgen DNA-Analysis of Metaplastic and Neoplastic Bronchial Cells.** (Eng) Nasiell, M. (Dept. Pathology, Sabbatsberg Hosp., Box 6401, S-113 82 Stockholm, Sweden); Kato, H.; Auer, G.; Zetterberg, A.; Roger, V.; Karlen, L. *Cancer* 41(4): 1511-1521; 1978.

Quantitative determinations of nuclear DNA were made in sputum cytology specimens with diagnoses of metaplasia; metaplasia with borderline, mild, moderate, or severe atypia; carcinoma in situ; and invasive carcinoma. An attempt was made to determine if progressive atypia and neoplasia are related to changes in DNA content. The cellular material was processed by the destaining diagnostic smears and processed by destaining the diagnostic smears and restaining them by the Feulgen method. Nuclear DNA content was determined with a scanning microspectrophotometer. There was good correlation between increasing degree of aneuploidy and increasing degree of morphologic nuclear atypia. The data also indicate a progressive aberration from normality of nuclear DNA content in squamous metaplastic and neoplastic cells exhibiting a progressive degree of atypia. This implies that squamous bronchial carcinoma is preceded by sequential changes in cellular composition that can be detected by quantitative DNA analysis and cytology. (15 refs)

78-3519 **Endogenous Factors in Bronchial Carcinoma.** (Ger) Kohout, J. (Medizinische Abteilung, Wilhelminenspital, Montleartstrasse 37, A-1171 Vienna, Austria). *Wien Med Wochenschr* 128(4): 1-8; 1978.

Bronchial carcinoma and its possible relationships with other diseases were studied in a series of 224 patients. Lung carcinoma was found to be associated with chronic bronchitis in 195/224 patients, but there were no significant differences in the incidence of chronic bronchitis among the different histological types of the carcinoma. Chronic bronchitis was found in 87/99 cases of squamous cell carcinoma, 37/42 cases of adenocarcinoma, 33/37 cases of small cell carcinoma, 35/41 cases of giant cell carcinoma, and 3/5 cases of alveolar cell carcinoma. Lung carcinoma was associated with chronic gastric or duodenal ulcer in 39 cases, with other chronic gastrointestinal diseases in 39, with chronic pulmonary tuberculosis in 21, with chronic cholecystopathy in 21, with liver cirrhosis or chronic hepatitis in 19, and with chronic sinusitis, tonsillitis, or otitis in 18. The distribution of these chronic diseases was independent of the histological type of the carcinoma. The skin window test revealed weakened and delayed macrophage activity in the carcinoma patients, independent of the histological type. These findings and the fact that the recent increase in lung cancer incidence is mainly due to an increase in the incidence of squamous cell carcinoma indicate the absence of an evident causal relationship between lung carcinoma and any accompanying chronic diseases. The reduced macrophage activity observed in both the lung carcinoma and these diseases may be a common cause of their chronic course and frequent association. (26 refs)

78-3520 **Unusual Coincidence of Carcinoma of the Stomach and Bronchi.** (Cze) Zdichynec, B. (Vnitrni oddeleni, nemocnice s poliklinikou Pocataky, OUNZ, Pelhrimov, Czechoslovakia). *Cesk Gastroenterol Vyz* 31(8): 554-556; 1977.

Carcinomas of the bronchi and stomach with metastases to the liver, adrenals, kidneys, and pericardium were found in a 52-yr-old man who smoked > 40 cigarettes a day. The incidence of gastric and bronchial carcinoma in relatives of the same generation was 5/16 and 2/16, respectively. (6 refs)

78-3521 **Morphologic Aspects of Experimental Canine Bronchial Carcinogen Exposure.** (Eng) Cohen, A. H. (Dept. Pathology, Harbor General Hosp., 1000 W. Carson St., Torrance, CA, 90509); Shors, E.; Okita, M.; Matsumura, K.; Jensen, T.; Benfield, J. R. *Eur J Cancer* 14(4): 401-409; 1978.

Alterations in canine bronchial epithelium were studied during the course of recurrent localized carcinogen exposure. Five groups of dogs were used. Group 1 (7 mongrels) received submucosal injections of 3 ml of a solution containing 15 mg/ml benzo(a)pyrene (BP) attached to a ferric acid carrier for a total dose of 810 mg over 32 mo. Group 2 (8 mongrels) received submucosal injections of 3 ml of a solution containing 10-15 mg/ml N-methyl-N-nitrosourea (MNU) for a total dose of 810-945 mg over 28 mo. Group 3 (6 mongrels) received BP similar to Group 1 for 11 mo and then mucosal application of 0.5 ml of a solution containing 10 mg/ml NMU for 16 mo. Group 4 (12 inbred beagles) and Group 5 (21 inbred beagles) received submucosal injections of 0.5 ml of a solution containing 30 mg/ml BP for 7 mo (total dose 315 mg) and mucosal application of 0.5 ml of a solution containing 30 mg NMU for 17 mo (total dose 135 mg). Group 5 dogs were treated with azathioprine and prednisolone to achieve chronic immunosuppression. The initial lesion was hyperplasia of the columnar cells with occasional intraepithelial gland formation. Basal cell hyperplasia and squamous metaplasia ensued and were frequently coexistent. Atypical epithelial maturation was noted in all dogs, especially those with squamous metaplasia. The immunosuppression regimen indicated that antibody responses were depressed within 2 mo, skin graft rejection was prolonged, and cutaneous responses to injected antigens were suppressed, compared to Group 4. Thus, the dog is an ideal model for studying the sequence of events during the induction of bronchial carcinomas. (20 refs)

78-3522 **Enhancement of Artificial Lung Metastases in Tumor-Bearing Mice (Meeting Abstract).** (Eng) Ando, K. (M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Hunter, N.; Peters, L. J. *Proc Am Assoc Cancer Res* 19: 172; 1978. (no refs)

78-3523 **Physiological and Molecular Markers During Carcinogenesis.** (Eng) Farber, E. (Dept. Pathology, Univ. Toronto, Toronto, Canada); Ogawa, K.; Cameron, R. G.; Laishes, B.; Solt, D.; Lin, J. C. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 20-21 pp.; 1978. (no refs)

78-3524 **Isoniazid and Bile Duct Cancer (Letter to Editor).** (Eng) Lowenfels, A. B. (Westchester County Medical Center, Valhalla, NY); Norman, J. *JAMA* 240(5): 434-435; 1978.

Two men (55 and 71 yr old) developed bile duct cancer 18 and 2 yr after receiving isoniazid (total dose, 122 and 36 g, respectively) for pulmonary tuberculosis. The short interval in the latter patient may have been due to the fact that he was an alcoholic. These were the only bile duct cancers seen at this hospital from 1970 to 1977. (3 refs)

78-3525 **Management of Congenital Choledochal Cyst with Intrahepatic Involvement.** (Eng) Todani, T. (Dept. Surgery, Okayama Univ. Medical Sch., Okayama, Japan); Narusue, M.; Watanabe, Y.; Tabuchi, K.; Okajima, K. *Ann Surg* 187(3): 272-280; 1978.

Observations of 75 patients (17 personal cases and 58 from the literature) with choledochal cysts with intrahepatic involvement (Type IV-A) indicated that surgical treatment must achieve bile drainage from both choledochal and intrahepatic sites. To prevent cancer at a later age, hepaticoenterostomy at the porta hepatis with partial resection of the wall of the intrahepatic cyst combined with excision of the intrahepatic cyst is recommended. Six of the 75 patients developed cancer. (34 refs)

78-3526 **Retrograde Lymphatic Spread of Colonic Carcinoma to the Liver.** (Eng) Kumar, R. K. (Sch. Pathology, Univ. New South Wales, Kensington, New South Wales 2033, Australia). *Experientia* 34(6): 768-769; 1978.

A case is presented of retrograde lymphatic permeation of the liver by a colonic carcinoma. A 63-yr-old man underwent hemicolectomy for a moderately differentiated adenocarcinoma of the cecum. Three months later, multiple metastatic nodules were found in the abdominal wall and mesentery. He died 1 mo later. Autopsy findings indicated that the carcinoma had recurred at the site of previous anastomosis. There was no evidence of contiguous spread of the tumor. The liver contained no metastatic nodules, but the carcinoma had spread along the neurovascular-ductal bundles arising from the porta hepatis and had involved lymph nodes in the porta hepatis. Microscopic examination revealed a moderately differentiated adenocarcinoma that had infiltrated the submucosal, muscular, and serosal layers of the intestine at the anastomotic sites. In the liver, there was extensive malignant infiltration of the major portal tracts. It is concluded that this case illustrates the typical features of retrograde lymphatic permeation of the liver by a malignant tumor. (4 refs)

78-3527 **Non-tropical "Idiopathic Splenomegaly": a Follow-up Study of Ten Patients Described in 1969.** (Eng) Dacie, J. V. (Dept. Haematology, Royal Postgraduate Medical Sch., Ducane Road, London W12 0HS, England); Galton, D. A.; Gordon-Smith, E. C.; Harrison, C. V. *Br J Haematol* 38(2): 185-193; 1978.

The history of 4/10 patients who were reported in 1969 as suffering from nontropical idiopathic splenomegaly is given. Of the remaining six patients, two had been lost to follow-up and four had died: two from lymphoma 4.5 yr and 8 mo, respectively, after splenectomy and two from unrelated causes. Two of the four living patients developed malignant lymphomas 6 and 2 yr, respectively, after splenectomy. The other two have lived for 17 and 15 yr, respectively, without developing any signs of a malignant tumor. One of the lymphoma patients was treated successfully by x-radiation; the other patient was initially treated successfully by x-radiation and chemotherapy and is still alive, although the tumor is now resistant to chemotherapy. The histology of the patients' spleens was reviewed in the light of their clinical history, but no criteria were found that would be of prognostic value. However, some cytological abnormality was recognized in the spleens of 3/4 patients who developed malignant lymphomas. It appears that unless the cell population in both the red and white pulp is strictly normal, the patient is likely to develop a malignant lymphoma. The tumors that subsequently developed had no special or characteristic features. (5 refs)

78-3528 **Effects of Culture Conditions on the Growth and Differentiation of Transformed Rat Adrenocortical Cells.** (Eng) Auersperg, N. (Cancer Res. Center, Univ. British Columbia, Vancouver, British Columbia, V6T 1W5, Canada). *Cancer Res* 38(7): 1872-1884; 1978.

Depending on culture conditions, normal rat adrenocortical (AC) cells grown in vitro either proliferate and are fibroblast-shaped, with myoid cell characteristics and limited steroidogenic activity, or they do not replicate and are epitheliallike and similar to AC secretory cells structurally and steroidogenically. The effects of oncogenic transformation on these cellular responses were investigated in the Kirsten murine sarcoma virus-transformed rat AC line TRA. Transformation eliminated the differential proliferative response of the cells to culture variables and resulted in similar growth rates and high cell densities under all conditions. Horse serum, conducive to epitheliallike growth of untransformed cells, caused aggregation, but fetal calf serum, conducive to fibroblastic growth of untransformed cells, caused dispersed growth in TRA cultures. Variations in dissociation procedures had little effect on growth patterns. Most ultrastructural characteristics of myoid as well as AC differentiation were reduced in TRA cells; basement membranes and extracellular matrix were produced, but under different conditions than those in untransformed cultures. Like normal AC cells, TRA cells were capable of 20α-reduction and Δ^5-3-β-dehydrogenation of ^{14}C-pregnenolone but, in contrast to untransformed cells, they did not respond to variables in culture conditions by qualitative changes in pregnenolone metabolism, they did not produce deoxycorticosterone or corticosterone, and they secreted progesterone, which was not recovered from untransformed cultures. TRA cells showed no increase in steroidogenic differentiation in response to culture environment factors that normally modulate fibroblast-shaped AC stem cells to more differentiated steroid-secreting cells. (33 refs)

78-3529 **Adrenocortical Carcinoma in Two Children with Subsequent Primary Tumors.** (Eng) Levine, G. W. (Dept. Pathology, Children's Hosp., 1056 E. 19th Ave., Denver, CO, 80218). *Am J Dis Child* 132(3): 238-240; 1978.

The case reports of two unrelated 13-mo-old children with adrenocortical carcinoma who subsequently developed another primary tumor are presented. In both patients, the tumor was removed surgically, and no specific antitumor therapy was given. In one child an intracerebellar medulloblastoma was diagnosed at age 5, and in the other a poorly differentiated ileocecal lymphocytic lymphoma was diagnosed at age 8. Since neither child had a history suggestive of exposure to environmental agents and iatrogenic causes can be excluded as a factor in the tumorigenesis, it is suggested that these patients had a genetic predisposition to tumor development. None of the parents exhibited an immune defense deficit. (17 refs)

78-3530 **Wilms Tumor in a Mother and Child.** (Eng) Tebbi, K. (Dept. Pediatrics, Case Western Reserve Univ., Rainbow Babies & Childrens Hosp., 2101 Adelbert Road, Cleveland, OH, 44106); Gross, S. *J Pediatr* 92(6): 1026-1027; 1978.

The occurrence of Wilms' tumor in a mother and son is reported. Both had undergone left nephrectomy, at ages 1 yr and 5 yr, respectively, for the tumor. No abnormalities were noted in three siblings. The son is free of recurrent disease 2 yr later. Neither the mother nor the child had identifiable chromosomal abnormalities. It has been suggested that for hereditary Wilms' tumor, a mutation in the germinal cells of one parent is inherited by the infected offspring, but a second mutation in the somatic cells is necessary for tumor development. (7 refs)

78-3531 **Inflammatory Oncotaxis.** (Eng) DerHagopian, R. P. (Div. Surgical Oncology, Univ. Miami, Sch. Medicine, P.O. Box 520875, Miami, FL, 33152); Sugarbaker, E. V.; Ketcham, A. *JAMA* 240(4): 374-375; 1978.

The presence of long disease-free intervals prior to cancer recurrence supports the concept of the dormant neoplastic cell. Clinical and experimental observations suggest that tissue trauma and subsequent inflammation enhance the implantation and growth of circulating cancer cells at trauma sites or that dormant neoplastic cells at these sites may undergo metamorphosis and growth. Brief reports of four recent cases of tumor recurrence following trauma and inflammation are offered as examples. The term oncotaxis is used to convey the overall picture of cancer cell attraction to points of inflammation. Inflammation is oncotactic because it facilitates entrapment of tumor cells and provides capillary stickiness and disruption in the intercellular endothelial barrier. Whether there are actual substances similar to chemotactic products that attract WBC is speculative. Inflammation is only one potential cause for facilitating metastases. (9 refs)

78-3532 **Highly Differentiated Gastric Adenocarcinoma Originating from the Normal, Non-metaplastic Gastric Epithelium. An Ultrastructural Study of a Case.** (Eng) Teglbjoerg, P. S. (Inst. Pathology, Odense Univ. Hosp., DK-5000 Odense, Denmark). *Acta Pathol Microbiol Scand [A]* 86(1): 87-89; 1978.

Light microscopic and ultrastructural features of a highly differentiated gastric adenocarcinoma are presented. The specimen was collected during a total gastrectomy performed on a 69-yr-old man who had complained of increasing dyspepsia for 6 mo. By light microscopy, the tumor was classified as a pylorocardiac gland carcinoma. Incomplete microvilli could be observed by electron microscopy at the luminal cytoplasmic membrane. There were plentiful spherical, electron-dense secretory granules in the luminal part of the cytoplasm. Identical secretory granules were seen in the pyloric glands of normal nonneoplastic mucosa removed from another stomach. The cells of these glands also had incomplete microvilli. This ultrastructural study provides considerable evidence that the tumor had its origin in the normal, nonmetaplastic gastric epithelium, possibly the pylorocardiac glands. (10 refs)

78-3533 **Cancer of the Cardia Misdiagnosed as Hiatal Hernia.** (Fre) Ballivet, M. (Centre Hospitalier Princesse Grace, av. Pasteur, Monaco, Principality Monaco); Fissore, A.; Fissore, O.; Campora, J. L.; Annonier, C. *Sem Hop Paris* 54(3): 155-156; 1978.

Cancer of the cardia of the stomach was discovered in a 70-yr-old man who underwent surgery for a hiatal hernia. An extreme narrowing of the esophagus at the esophagogastric junction had prevented endoscopic examination of the stomach. The original diagnosis was hiatal hernia with peptic esophagitis. (no refs)

78-3534 **Genetic Aspects of Gastritis-Cancer Relationship.** (Eng) Varis, K. (Gastroenterological Unit-ed Second Dept. Medicine, Meilahti Hosp., SF-Helsinki, Finland); Ihamaki, T.; Lehtola, J.; Sipponen, F.; Kekki, M.; Isokoski, M.; Saukkonen, M.; Siurala, M. *Dtsch Z Verdau Stoffwechselkr* 38(2): 51-54; 1978.

Genetic studies of three family samples comprising first-degree relatives of probands with severe atrophic body gastritis, pernicious anemia, and gastric carcinoma were performed to evaluate the genetic background and precancerous significance of atrophic gastritis. The studies indicated that the tendency to develop severe atrophic body gastritis is influenced by one genetic factor and that this genetically caused type of gastritis is connected with a higher risk of developing gastric carcinoma. There is, however, no conclusive evidence that atrophic gastritis alone leads to gastric carcinoma. It is possible that it makes the gastric mucosa more prone to the effects of factors that initiate the promotion phase of the malignant process. It is also possible that atrophic gastritis and gastric carcinoma are parallel phenomena with some common genetic factors.(7 refs)

78-3535 **Genetic and Dynamic Views on Gastritis-Cancer Relationship (Meeting Abstract).** (Eng) Varis, K. (Gastroenterological Unit, Second Dept. Medicine, Meilahti Hosp., Univ. Helsinki, Helsinki, Finland); Kekki, M.; Lehtola, J.; Ihamaki, T.; Sipponen, P.; Isokoski, M.; Saukkonen, M.; Siurala, M. *Digestion* 16(3): 281-282; 1977. (no refs)

78-3536 **Cancer Risk of the Stomach Resected for Ulcer: The Influence of the Gastroenteric Anastomosis (Meeting Abstract).** (Eng) Dahm, K. (Chirurgische Universitatsklinik, Hamburg, W. Germany); Eichfuss, H. P.; Koch, W. *Digestion* 16(3): 236; 1977. (no refs)

78-3537 **Follow-Up of Patients with Chronic and Atrophic Gastritis for the Question of Precancerous State (Meeting Abstract).** (Eng) Bergemann, W. (Geriatric Dept., Second Internal Dept., General Hosp., Berlin-Wilmersdorf, W. Germany). *Digestion* 16(3): 230-231; 1977. (no refs)

78-3538 **Gastric Lesions in Familial Adenomatosis Coli. Their Incidence and Histologic Analysis.** (Eng) Watanabe, H. (Second Dept. Pathology, Faculty Medicine, Kyushu Univ., 60, Fukuoka 812, Japan); Enjoji, M.; Yao, T.; Ohsato, K. *Hum Pathol* 9(3): 269-283; 1978.

The frequency of accompanying gastric lesions in familial adenomatosis coli (FAC) was investigated in 22 patients from 14 families. Various gastric lesions were confirmed in 15 patients belonging to 13 families. Histologically, the lesions were classified as adenoma (9), fundic gland polyposis (6), carcinoma (3), and microcarcinoid (2). Fundic gland polyposis consisting of simple hyperplasia of the fundic glands may be the gastric lesion specific to FAC, since polyposis has not been reported in any other disease. FAC affects not only the colon and rectum, but is capable of inducing tumorigenicity in other organs. The stomach is one of the organs in which extracolonic lesions occur. (35 refs)

78-3539 **Malignant Transformation of Hyperplasiogenic Polyps of the Stomach. Case Report.** (Eng) Remmele, W. (Pathologisches Inst., Kliniken der Landeshauptstadt Wiesbaden, Schwalbacher Strasse 62, D-6200 Wiesbaden, W. Germany); Kolb, E. F. *Endoscopy* 10(1): 63-65; 1978.

Examination of hyperplasiogenic gastric polyps from a 71-yr-old woman revealed the presence of adenocarcinoma in two polyps. It is suggested that a biopsy diagnosis of hyperplasiogenic polyp be followed by removal of the whole polyp to prevent or halt malignant transformation. In view of this instance of transformation, the classification of hyperplasiogenic polyps as hyperplastic should be reevaluated. (8 refs)

78-3540 **Duodenal Ulcer Associated with Pyloric Microgastrinoma and Antropyloric Focal Gastrinosis.** (Fre) Berger, G. (Laboratoire d'Anatomie Pathologique de l'UER, Alexis Carrel Hopital Cardiologique, B. P. Lyon Monchat, F 69394 Lyon Cedex 3, France); Chayvialle, J. A.; Berger, F. *Nouv Presse Med* 7(22): 1937-1940; 1978.

A small benign gastrinoma of the pylorus and focal gastrinosis were discovered in the vagotomy-antrectomy specimen of a 59-yr-old man with symptoms of duodenal ulcer. The ulcer symptoms were of 20 yr duration and had recently increased in intensity. X-rays and gastric endoscopy revealed a duodenal ulcer. Despite medical treatment, the symptoms persisted. Initial exploration of acid secretion and blood gastrin levels in response to iv secretin (3 IU/kg/hr) showed only a slight elevation in gastrin levels and no abnormalities in acid secretion. A second exploration 3 mo later showed a moderate but stabilized increase in blood gastrin levels and acid secretion that led to the tentative diagnosis of gastrinoma or hyperplasia of the gastrin-producing antral cells. No pathology was observed during vagotomy-antrectomy; after the surgery, the and blood gastrin level fell to below normal levels. Examination of the surgical speciman showed a tiny (0.5 cm) submucosal carcinoid tumor (gastrinoma) with an ulcerated surface. It had not infiltrated the muscle layer. The gastrin cells in the tumor were revealed by immunocytochemical tests with antigastrin serum. Gastrin cells were also observed in the antropyloric region. The role of these minimal gastrin cell lesions in the pathogenesis of duodenal ulcer and their relationship to the Zollinger-Ellison syndrome are discussed. (12 refs)

78-3541 **Adenocarcinoma of the Lower Third of the Duodenum. Diagnosis by Endoscopy and Radiology.** (Fre) Debray, C. (Clinique des Maladies de l'Appareil digestif, Service de Gastro-enterologie A, Hopital Bichat, 170, boulevard Ney, 75877 Paris Cedex 18, France); Charleux, H.; Gouerou, H.; Richard, P.; Marche, C. *Sem Hop Paris* 54(3): 172-175; 1978.

A 35-yr-old woman developed an adenocarcinoma of the lower third of the duodenum, a rare location (0.033%-0.05% at autopsy). Endoscopy with 11 biopsies and 2 x-ray series were necessary to diagnose the 3-cm neoplasm, which was located 9 cm from the pylorus. The patient had a duodenectomy, a pancreatectomy of the head of the pancreas, and a cholecystectomy, and she was well 1 yr after surgery. (no refs)

78-3542 **Three Antral Lesions: Two Neoplasms (One on the Site of an Ulcer Scar) and an Accessory Pancreas.** (Fre) Debray, C. (Service de Gastro-enterologie A, Clinique des Maladies de l'Appareil digestif, Hopital Bichat, 170 boulevard Ney, 75877 Paris Cedex 18, France); Leymarios, J.; Benhamou, G.; Marche, C. *Sem Hop Paris* 54(3): 163-167; 1978.

The resected stomach of a 70-yr-old man had three lesions in the antral region. The first lesion was a differentiated carcinoma superimposed on a gastric ulcer, the second was a differentiated adenocarcinoma, and the third was a 9-mm accessory pancreas. (no refs)

78-3543 **Induction of Pancreatic Ductulitis in Syrian Hamsters Transplanted with Pancreatic Adenocarcinoma (Meeting Abstract).** (Eng) Runge, R. G. (Eppley Inst. Cancer Res., Univ. Nebraska Medical Center, Omaha, NB, 68105); Pour, P.; Takahashi, M. *Proc Am Assoc Cancer Res* 19: 184; 1978. (1 ref)

78-3544 ***Streptococcus bovis* Endocarditis and Colonic Cancer (Letter to Editor).** (Eng) Noble, C. J. (Dept. Medical Microbiology, King's College Hosp., London, SE5 8RX, England); Uttley, A. H.; Falk, R. H.; Richardson, P. J. *Med J Aust* 1(8067): 766; 1978.

A 60-yr-old man presented with a short history of wt loss, rectal bleeding, and altered bowel habit 5 mo after admission for *Streptococcus bovis* endocarditis. Secondary hepatic deposits of well-differentiated adenocarcinoma were found, and the primary lesion was assumed to be the large bowel. In view of the eight literature cases of this association, it is suggested that *S. bovis* endocarditis patients be screened for carcinoma of the colon. (1 ref)

78-3545 **Promotion of Intestinal Neoplasia by Postoperative Mucosal Hyperplasia (Meeting Abstract).** (Eng) Williamson, R. C. (Bristol Royal Infirmary, Bristol, England); Bauer, F. L.; Ross, J. S.; Malt, R. A. *Digestion* 16(3): 284-285; 1977. (no refs)

78-3546 **Intestinal Polyposis: A Genetic Disease.** (Fre) Plauchu, H. (Service de Genetique, Hotel Dieu, F 69288 Lyon Cedex 1, France); Bigay, D.; Casteu, J. G.; Guillemin, G.; Robert, J. M. *Lyon Med* 239(1): 21-30; 1978.

The genetic and clinical aspects of intestinal polyposis are discussed based on a study of 32 cases in 22 families. A study of four family trees revealed variable expression of the responsible gene. It is suggested that familial polyposis coli has a genetic link with Gardner's syndrome and, perhaps, with Turcot's syndrome. (47 refs)

78-3547 **The Significance of Cholecystectomy in Colonic Carcinogenesis (Meeting Abstract).** (Ger) Werner (Hamburg, W. Germany); de Heer; Mitschke *Zentralbl Chir* 103(6): 378; 1975. (no refs)

78-3548 **Familial Colon Cancer.** (Fre) Sarroca, C. (Department de Chirurgie, Hopital Central des Forces Armees, Montevideo, Uruguay); Quadrelli, R.; Praderi, R. *Nouv Presse Med* 22(16): 1412; 1978.

Familial colonic cancer without polyposis was found in four generations of one family. The cancer was associated with cancer of the skin, pancreas, stomach, and endometrium and with acanthosis of the lips in some members. (no refs)

78-3549 **Human Ovarian Tumours Heterotransplanted to "Nude" Mice.** (Eng) Kullander, S. (Dept. Obstetrics and Gynaecology, Univ. Lund, Lund, Sweden); Rausing, A.; Trope, C. *Acta Obstet Gynecol Scand* 57(2): 149-159; 1978.

The acceptance, growth, and histology of 22 different human ovarian tumors, which were heterotransplanted sc to female nude mice, were studied. When small tissue pieces were used, 10/16 tumors grew. When tumor cell suspensions were used, however, all 11 trials failed. Metastatic or infiltrative growth was not seen in mice observed for up to 25-40 days. Material from each tumor was transplanted to four separate sites in two mice. The successful grafts largely retained the original morphological features. As measured by ^{3}H-thymidine incorporation, DNA synthesis was also similar in the original tumors and the growing grafts. Preliminary lactic dehydrogenase isoenzyme analysis of mouse venous blood several weeks after grafting showed that the original human tumor isoenzyme picture was mirrored in the mouse blood. Only one of the tumors from six previously treated patients grew. Irradiation and the cytostatic drugs might directly or indirectly have influenced cellular multiplication, DNA synthesis, and the possibilities of these damaged cells growing in the nude mouse. (22 refs)

78-3550 **The Ultrastructure of Endometrioid Carcinomas of the Ovary.** (Eng) Fenoglio, C. M. (Columbia Univ. Coll. Physicians and Surgeons, New York, NY, 10032); Puri, S.; Richart, R. M. *Gynecol Oncol* 6(2): 152-164; 1978.

Tissue samples from 12 histologically documented cases of human ovarian endometrioid carcinoma were examined by light microscopy, scanning electron microscopy, and transmission electron microscopy. Eight of the 12 cases were well- or moderately well-differentiated neoplasms composed of cells arranged in a glandular pattern resembling malignant glands of the uterine endometrium. Seven of these contained cell types other than classical endometrial cells: 4 contained areas of clear cells, 3 were associated with squamous metaplasia (adenoacanthoma), 2 contained mucinous or serous components, and in 1 case there was associated endometriosis. The ultrastructural features of these cases is described in detail. In general, the cells were pleomorphic and arranged irregularly, and most of the cell surfaces were covered with abundant and well-developed microvilli. The most characteristic feature of the endometriallike cells was bundles of perinuclear microfilaments and intracytoplasmic glycogen. The dominant cytoplasmic organelles were free ribosomes. Mitochondria, lysosomes, lipid droplets, and multivesicular bodies were also present. The plasma membranes in the apical regions were usually straight and reinforced by numerous desmosomes, but in the basal region they were irregular and connected with fewer and less well-developed desmosomes. A well-developed basal lamina was present. The nuclei were enlarged and ovoid, with well-defined nuclear membranes, solitary nucleoli, and chromatin material clumped at the periphery. Light microscopy can be used to diagnose well- and moderately well-differentiated lesions, but electron microscopy should be used for poorly differentiated cancers. (14 refs)

78-3551 **Uterine Choriocarcinoma Fourteen Years Following Bilateral Tubal Ligation.** (Eng) Lathrop, J. C. (Dept. Gynecology, Rhode Island Hosp., Providence, RI); Wachtel, T. J.; Meissner, G. F. *Obstet Gynecol* 51(4): 477-482; 1978.

A 42-yr-old woman developed a choriocarcinoma of the uterus 14 yr after bilateral tubal ligation and resection and 15 yr after the birth of her last (fourth) child. Chromosomal studies of cultured metastatic cells from a metastatic lung nodule and the skin revealed an extra number 8 chromosome in a subpopulation of cells. The tumor could have arisen from a new pregnancy, but no fistula between the peritoneal cavity and tube or endometrial cavity could be demonstrated or it could have arisen de novo as a nongestational lesion. However, it is most likely that the lesion resulted from the activation of chorionic cells that had probably been present since the patient's last pregnancy. (15 refs)

78-3552 **Squamous Cell Dysplasia and Carcinoma In Situ After Prenatal Exposure to Diethylstilbestrol: Examination and Follow-up of 1,424 Women (Meeting Abstract).** (Eng) Prat, J. (Dept. Pathology, Massachusetts General Hosp., Boston, MA, 02114); Robboy, S. J.; Barnes, A. B.; Scully, R. E.; Welch, W. R. *Lab Invest* 38(3): 360-361; 1978. (no refs)

78-3553 **Histology of Precancerous and Early Stages of Cervical Carcinoma (Meeting Abstract).** (Ger) Mobius, G. (Pathologisches Institut, Bezirkskrankenhaus, DDR-27 Schwerin, E. Germany). *Fortschr Med* 96(77): 350-350; 1978. (no refs)

78-3554 **Precancerous Potential of Condyloma of the Cervix and the Vagina (Meeting Abstract).** (Eng) Commisso, J. (Dept. Cytology, Magee-Womens Hosp., Pittsburgh, PA); Kanbour, A.; Fisher, E.; Klionsky, B. *Acta Cytol (Baltimore)* 21(6): 786; 1977. (no refs)

78-3555 **The Possible Role of Hemidesmosomes in Neonatally Estrogen-induced Selection of a Permanently Altered Abnormal Vaginal Epithelium.** (Eng) Cunha, G. R. (Anatomy Dept., Sch. Medicine, Univ. Colorado, Denver, CO, 80262); Lee, A. K. *J Exp Zool* 203(3): 361-370; 1978.

The involvement of hemidesmosomes in estradiol-induced vaginal epithelial cell selection was studied in neonatal BALB/c Crgl mice inoculated daily with 20 μg estradiol-17β (E). The number of hemidesmosomes per basal cell cross section (HD/bccs) of the vaginal epithelium increased gradually in both untreated and E-treated mice from 0.07 on day 1 to 2-3 on day 4. It then increased markedly on day 5, plateauing at 8 days at about 6-7 HD/bccs in the untreated mice and 14-15 in the E-treated mice. Approx 20 HD/bccs were observed in the vaginas of 57-day-old E-treated mice. Five-day-old E-treated mice contained two types of vaginal epithelial cells: basophilic cells containing about 19 HD/bccs and light cells containing only about 5 HD/bccs. The results suggest that electron-lucent basal cells with few HC/bccs are replaced in E-treated mice by basophilic, HD-rich cells. The data are consistent with the view that hormone-induced selection of vaginal epithelial cells whose expression of cornification and proliferation is independent of estrogen stimulation is accomplished by a selective increase in the numbers of hemidesmosomes in certain basal epithelial cells. (18 refs)

78-3556 **Hemopoietic Tissue Reactions Associated with the Growth of Transplanted Syngeneic Murine Tumors.** (Rus) Osipova, T. V. (Lab. Virology, Cancer Res. Center, Moscow, USSR); Bukhman, V. M.; Belianchikova, N. I.; Svet-Moldavskii, G. Ia. *Biull Eksp Biol Med* 85(3): 346-349; 1978.

The leukemoid reactions induced by transplanted syngeneic solid tumors were studied in (CBA x C57BL/6j) mice. Animals were inoculated sc with hemangiopericytoma cells (10^6 tumor cells/mouse), urinary bladder carcinoma cells, or hepatoma cells (the last two at 0.5 ml of 50% homogenate). Hemangiopericytoma transplantation resulted in a marked increase in spleen wt, spleen cell cellularity, and the number of colony-forming units. These changes were first observed on day 2 after tumor transplantation. Tumor growth was correlated with an increase in WBC (the number of mature neutrophils showed a 10-fold increase, and the number of monocytes showed a 10- to 20-fold increase). The leukemoid reaction was less pronounced in mice with the transplanted hepatoma. Animals with the transplanted urinary bladder tumor did not show any increase in spleen wt and cell cellularity. The only change noted was an increased number of mature granulocytes (60%, compared to 10%-20% in controls). (12 refs)

78-3557 **Cytological Changes due to Urinary Calculi. A Consideration of the Relationship Between Calculi and the Development of Urothelial Carcinoma.** (Eng) Beyer-Boon, M. E. (Dept. Cytopathology, Univ. Medical Centre, Leyden, Netherlands); Cuypers, L. H.; de Voogt, H. J.; Brussee, J. A. *Br J Urol* 50(2): 81-89; 1978.

Cytological abnormalities that may occur during the presence of urinary calculi and after their removal were investigated in 62 lithiasis patients, and the relationship between calculi and the development of urothelial carcinoma was studied in 92 patients with cancer of the renal pelvis and ureter. In 11/62 lithiasis patients, the calculi induced urothelial cellular abnormalities comparable with those associated with malignancy. Severe cellular changes, comparable with those of carcinoma in situ, were observed in the epithelium adjacent to a calculus. Squamous metaplasia was frequently observed in cases with staghorn stones in the renal pelvis. The abrasive effect of the calculus could have accounted for the many multinucleated cells found in the sediment. Upon removal of the calculi, the cytological atypia and the multinucleation disappeared. To date, none of these patients has developed urothelial carcinoma. According to the hospital records of 92 patients with cancer of the renal pelvis and ureter, 14 had either concomitant or previous lithiasis. This finding indicates that lithiasis is more common than would be expected in patients with renal pelvic and ureter cancer, suggesting a relationship between these conditions. (34 refs)

78-3558 **Morphologic Characterization of Two Established Cell Lines, T24 and MGH-U1, Derived from Human Bladder Carcinoma.** (Eng) Kato, T. (Dept. Urology, Akita Univ. .Sch. Medicine, Akita, Japan); Ishikawa, K.; Nemoto, R. *Jpn J Urol* 69(1): 40-46; 1978.

The morphology of two established cell lines, T24 and MGH-U1, derived from human bladder transitional cell carcinomas was investigated. Light microscopy revealed that both lines retained their epithelial features and grew in a multilayered pattern. Scanning electron microscopy indicated that the cells had microvilli as well as cytoplasmic protrusions on the cell surface. Transmission electron microscopy revealed junctional complexes between adjacent cells and well-developed cytoplasmic organelles. Viruslike particles were not detected. In the proliferating phase, the cells had a polymorphic appearance; electron microscopy failed to detect morphologically distinct cells. In spite of the high degree of similarity between the two lines, T24 had slightly more spindle-shaped cells and more relatively well-developed microvilli, compared with MGH-U1 cells. T24 cells had a modal chromosome number of 91, but MGH-U1 cells had one of 85. These findings indicate that T24 cells underwent a change in their morphologic appearance following long-term culture. (14 refs)

78-3559 **Occult Urinary Bladder Cancer Associated with Prostatic Hyperplasia or Cancer (Meeting Abstract).** (Eng) Kahan, A. (Dept. Pathology, Montefiore Hosp. and Medical Center, Bronx, NY); Schreiber, K.; Koss, L. G. *Acta Cytol (Baltimore)* 21(6): 788; 1977. (no refs)

78-3560 **Carcinosarcoma of the Urinary Bladder.** (Ita) Martinelli, G. (Istituto de anatomia e istologia pathologica, II Cattedra, Universita di Bologna, Bologna, Italy); Bazzocchi, F.; Piccaluga, A.; Reggiani, A.; Buli, P.; Bercovich, E. *Pathologica* 69(998): 613-622; 1977.

Carcinoma intermingled with chondrosarcoma was found in the urinary bladder of a 59-yr-old man. The combination carcinosarcoma is assumed to have originated from a single embryonic germ cell with potential epithelial and mesenchymal evolution. (10 refs)

78-3561 **The Natural History of Nephrogenic Adenoma of the Urinary Bladder.** (Eng) Cremer, H. (Pathologisches Institut, Universitat Bonn, Postfach 2120, D-5300 Bonn 1, W. Germany); Adolphs, H. D. *Z Krebsforsch* 91(1): 49-53; 1978.

The natural history of a nephrogenic adenoma of the urinary bladder is presented based on four biopsies from a 64-yr-old patient. The adenoma was composed of both villous and glandular structures. The two-layered surface epithelium consisted of an eosinophilic cell layer covering a clear cell layer. The latter rested on a delicate basal layer. The clear cells disappeared toward the tip of the villi. There was a 200-μm zone of transition between the urinary bladder epithelium and the nephrogenic adenoma. Scattered nodules of glandular structures were found at the base of the villi, and they were lined by eosinophilic cells or by clear cells. These epithelial cells were derived from the corresponding surface epithelial cells from which they had separated by active downward growth. (8 refs)

78-3562 **The Association Between Vesical Carcinoma and Urinary Obstruction.** (Eng) Fellows, G. J. (Dept. of Urology, General Infirmary, Leeds, England). *Eur Urol* 4(3): 187-188; 1978.

Signs of bladder outflow obstruction were recorded in 199 male patients with carcinoma of the bladder and in an age-matched control group. The incidence of obstruction in the patients was significantly higher than that in controls ($p < 0.05$) and it increased with age in both groups. The most common cause of obstruction in this series was benign prostatic hyperplasia. (3 refs)

78-3563 **Adenomatoid Tumour of the Epididymis: Histochemical and Ultrastructural Study of 2 Cases.** (Eng) Nistal, M. (Departamento de Morfologia, Facultad de Medicina, Universidad Autonoma, c/Arzobispo Morcillo 4, Madrid 34, Spain); Contreras, F.; Paniagua, R. *Br J Urol* 50(2): 121-125; 1978.

The histochemical and ultrastructural characteristics of adenomatoid epididymal tumors that occurred in two patients (46 and 51 yr old) are described. Acid mucopolysaccharides were present in the lumen of the tubular structures. The epithelial cells had numerous microvilli in the free surface, junction complexes, wide intercellular channels, and a well-developed Golgi complex. Abundant bundles of microfilaments and intracellular spaces were observed in the cytoplasm. Both lymphocytes and histiocytes were identified in the tubular lumen and between the epithelial cells. The stroma of the tumor was composed of dense connective tissue with abundant bundles of elastic fibers. These findings support the concept that these tumors are of mesothelial origin. This is the first time that intracytoplasmic spaces in the epithelial cells and elastic fibers in the stroma have been observed. (3 refs)

78-3564 **Unusual Incidence of Testicular Tumors.** (Eng) Gordon, J. O. (Urology Professional Assoc., 605 Garfield St., Tupelo, MS, 38801); Elliott, J. P.; Evans, J. W.; Platt, L. O.; Gordon, A. M. *South Med J* 71(4): 372-373; 1978.

The case of three men, ages 27, 49, and 36, with seminoma are presented. Although this is a rare tumor, all three men were seen by the same physician within 18 mo. Follow-up of the patients revealed no evidence of recurrence or metastases following radiotherapy and tumor excision. (1 ref)

See also:

*(Rev.): 78-3019, 78-3023, 78-3024, 78-3033, 78-3034, 78-3035, 78-3036, 78-3051, 78-3054, 78-3055.
*(Chem.): 78-3064, 78-3067, 78-3070, 78-3086, 78-3095, 78-3097, 78-3101, 78-3107, 78-3110, 78-3112, 78-3115, 78-3119, 78-3136, 78-3137, 78-3140, 78-3148, 78-3154, 78-3159, 78-3161, 78-3170, 78-3174, 78-3182, 78-3184, 78-3201, 78-3213, 78-3217, 78-3234.
*(Phys.): 78-3242, 78-3251, 78-3265.
*(Viral): 78-3331.
*(Immun.): 78-3388, 78-3402, 78-3408, 78-3427, 78-3443, 78-3459, 78-3461, 78-3465.
*(Epid.-Biom.): 78-3566, 78-3567, 78-3572, 78-3578, 78-3584.

78-3565 **Mortality from Carcinoma of the Cervix.** (Eng) Yule, R. (Christie Hosp., Holt Radium Inst., Withington, Manchester M20 9BX, England). *Lancet* 1(8072): 1031-1032; 1978.

Although deaths from cervical cancer in England and Wales decreased in the period 1970-1976, the number of deaths in women > 35 yr old doubled. This increase is attributable to the earlier onset of sexual activity and to the increased use of contraceptive pills or intrauterine devices, which offer no protection against the transmissible agent (virus or spermatozoa) thought to be responsible for the disease. Widespread and frequent cytotesting is necessary to ensure the detection of precancerous cervical conditions in younger women. (no refs)

78-3566 **Pancreatic Cancer: Three Connecticut Families in Which an Adult Child Presented with the Disease Before a Parent (Meeting Abstract).** (Eng) Sangree, M. H. (Waterbury Hosp., Waterbury, CT). *Gastroenterology* 74(5, part 2): 1143; 1978. (2 refs)

78-3567 **Familial Hodgkin's Disease. Epidemiological Considerations on Three Cases Diagnosed in Brothers During Infancy (Meeting Abstract).** (Spa) Gomez Pereira, C. (Servicio Hematologia-Hemoterapia, C.S. "Reina Sofia" de la Cordoba, Cordoba, Spain); Gomez Garcia, P.; Martinez Guibelalde, F.; Manzanares, R.; Garcia Castellano, J. M.; Velasco, F.; Aljama, R.; Torres Gomez, A. *Sangre (Barc)* 23(2): 247; 1978. (no refs)

78-3568 **Epidemiology of Bladder Cancer Deaths in Japan.** (Eng) Ohno, Y. (Dept. Preventive Medicine, Nagoya Univ. Sch. Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466, Japan); Aoki, K. *Gann* 68(6): 715-729; 1977.

To explore the epidemiological and etiological implications of bladder cancer mortality in Japan, national mortality statistics for 1947-1974 were analyzed. The age-adjusted death rate for bladder cancer increased in both sexes prior to the early 1960's, after which the rate leveled off in men and declined in women. Almost identical risks for bladder cancer deaths were noted in men born after 1910 and women born after 1900. The following trends were also identified: an apparent decrease in bladder cancer mortality among the young and an increase in older age groups; a decrease in mean age at death of 5.7 yr in men and 12.7 yr in women; and an unchanged age-specific death rate in men aged < 65 yr and a distinct decline in this rate in women < 65 yr old. There were no definite geographic gradients of the standardized mortality ratio and no apparent clustering of high ratios in prefectures with highly industrialized areas and large cities. These findings are incompatible with the recent remarkable increases in potentially hazardous agents in Japan's environment. Since bladder cancer is one of the few malignancies in which the role of environmental and occupational agents has been shown to be important, the results might indicate the relative importance of inherent host factors and unidentified environmental factors in bladder cancer. This suggestion is supported by the consistent proportion over the years of bladder cancer deaths among deaths from all urinary tract cancers. (47 refs)

78-3569 **Childhood Cancer in Sweden, 1958-1974. I. Incidence and Mortality.** (Eng) Ericsson, J. L. (Central Lab. Clinical Pathology, Univ. Hosp., Univ. Uppsala, Uppsala, Sweden); Karnstrom, L.; Mattsson, B. *Acta Paediatr Scand* 67(4): 425-432; 1978.

Data on the incidence and mortality of childhood cancer in Sweden are presented, based on the 3,797 cases of tumors and tumorlike conditions in children 0-14 yr old reported to the Swedish cancer registry during 1958-1974. During this period, the annual number of cancer cases, deaths, and deaths from cancer (per million) in boys was 142, 1,435, and 93, respectively; the corresponding numbers for girls were 124, 1,066, and 77. Almost half the cancers affected children < 5 yr of age. The lowest incidence occurred at age 7-8 yr, the highest during the first year of life. The most common diagnoses were leukemia and nervous system tumors, both of which constituted 58% of the total. The most common CNS tumors were astrocytomas and related forms, but neuroblastomas predominated in the peripheral nervous system. Teratoma was a comparatively common histopathological diagnosis. The most common leukemias were the acute blast cell and acute lymphatic types. Linear regression analysis of the data for 1958-1974 showed that the increase in incidence rates was significant for boys (2.3%/yr), much less so for girls (0.7%/yr). The major part of the increase in incidence rates was accounted for by nervous system tumors. If these tumors are excluded, the tendency toward an increased occurrence of childhood cancer is abolished. The increase in nervous system tumors was three times higher in boys than in girls. Mortality rates slightly decreased during this period, especially for girls. The decrease in mortality rates was most pronounced for Wilms' tumor and for leukemia in children 0-4 yr of age, a result of improved treatment methods. (13 refs)

78-3570 **Cancer Epidemics in the People's Republic of China.** (Eng) Miller, R. W. (Clinical Epidemiology Branch, Div. Cancer Cause and Prevention, A-521, Landow Building, NCI, NIH, Public Health Service, U.S. Dept. Health, Education and Welfare, Bethesda, MD, 20014). *J Natl Cancer Inst* 60(6): 1195-1203; 1978.

Information on cancer epidemiology gained during a trip by a U.S. Cancer Delegation to four cities and two rural areas of China is reported. Three of four epidemic types of cancer occur regionally and are apparently due to environmental exposures. High incidences of nasopharyngeal carcinoma (NPC) and hepatocellular carcinoma (HCC) occur in the South and esophageal carcinoma (ES) is found in some areas of the North. High incidences of trophoblastic neoplasia occur throughout China. Cases of NPC have also been found in domestic pigs in the same area endemic for human NPC, suggesting that the search for a cause of NPC in man may be narrowed to the small environment of the pig as contrasted with the diverse exposures of man. EC in man was found to be associated with cancers of chicken gullets. In one case of a mass relocation of the human population, the carcinogen causing gullet cancer appeared to have moved with the people to infect the chickens in the new area. HCC was found in a few cirrhotic ducks in an area where the neoplasm occurs excessively in man, but, unlike NPC in pigs and gullet cancer in chickens, no systematic survey of ducks for HCC has been made. Mass surveys have been undertaken in China for cancer detection. In one province, early detection resulting from these surveys has virtually eliminated death from cancer of the uterine cervix in textile workers. (31 refs)

78-3571 **An Outbreak of Pleural Mesothelioma and Chronic Fibrosing Pleurisy in the Village of Karain/Urgup in Anatolia.** (Eng) Baris, Y. I. (Dept. Chest Diseases, Hacettepe Univ. Sch. Medicine, Ankara, Turkey); Sahin, A. A.; Ozesmi, M.; Kerse, I.; Ozen, E.; Kolacan, B.; Altinors, M.; Goktepeli, A. *Thorax* 33(2): 181-192; 1978.

The cause of the 11 deaths from pleural mesothelioma and the 5 cases of fibrosing pleurisy that occurred among the 575 inhabitants of Karain, Turkey, in 1975-76 was investigated. In the previous 5 yr, there had been 25 cases of mesothelioma. There was no definite evidence of a genetic factor; the main occupation of those affected was agriculture. Examination of the physical and economic structure of the village and geological, biological, and chemical analyses of the food, rocks, soil, and water indicated the presence of a few asbestos fibers, 5-7 μm long, in the water. Asbestos fibers were also found in th pleural tissues of 2/5 patients examined. Pleural calcificatio was seen quite frequently, and it was typical of that resultin from asbestos exposure. Inhabitants of neighboring villag were free of mesothelioma. The asbestos fibers may b derived from the wearing down of asbestos-containing rock by the water supply. They are either ingested or inhaled afte the water evaporates and the fibers become airborne. (28 refs

78-3572 **Lung Cancer in Various Chronic Lung Disease** (Rus) Smirnov, E. A. (Dept. Pathoanatom Central District Hosp., Pushkin, Moscow Oblast, USSR *Vopr Onkol* 24(2): 93-95; 1978.

The association of lung cancer with other chronic respirator diseases was evaluated by autopsy examination of 104 patients with lung cancer. Lung cancer was combined wit chronic purulent bronchitis in 26.8% of the cases, wit chronic pneumonia in 12.5%, with fibrous-focal and cirrhoti tuberculosis in 17.3%, with diffuse pneumosclerosis i 11.5%, and with pulmonary emphysema in 9.6%. Centra lung cancer was predominantly associated with chronic purulent bronchitis and pulmonary emphysema (45.5%), peripheral cancer with tuberculosis and chronic pneumoni (56.2%). The patients with chronic purulent bronchitis usually developed poorly differentiated and glandular carcinomas, whereas squamous cell carcinomas were more common in patients with tuberculosis, chronic pneumonia, an diffuse pneumosclerosis. (12 refs)

78-3573 **Relation of Occupations to the Regional Differences of Lung Cancer Mortality in Fukuok Prefecture.** (Eng) Shigematsu, T. (Dept. Public Health Fukuoka Univ. Sch. Medicine, 34 Nanakuma, Nishi-ku Fukuoka 814, Japan); Yamasaki, M. *Jpn J Ind Health* 19(4) 182-188; 1978.

The relationship of occupation to geographical differences i lung cancer patterns was examined in Fukuoka Prefecture Japan. Data for 1971-1973 were taken from previous statistical analyses, cancer registry data, and census reports. In men the standardized mortality rate for lung cancer was 16% higher than that of all Japan; in women, the rate was less tha half that of the men, but 24% higher than the rate for al Japan. An evident clustering of cities and towns with hig death rates was observed in a district that, until 1965, ha been one of the largest coal-mining areas in Japan. Amon 20 cities and boroughs in the prefecture, there were significant correlations between lung cancer incidence and mortality for 1971-1973 and percentages of workers employed i mining and quarrying occupations in every census yr afte World War II. No other occupations showed consistent rela ions to lung cancer rates. The clustering suggests that coal-mining occupations may have a sizeable influence on lung cancer. (19 refs)

78-3574 **A Study of Diet and Breast Cancer.** (Eng) Miller, A. B. (NCIC Epidemiology Unit, Univ. Toronto, McMurrich Building, Toronto, Ontario M5S 1A8, Canada); Kelly, A.; Choi, N. W.; Matthews, V.; Morgan, R. W.; Munan, L.; Burch, J. D.; Feather, J.; Howe, G. R.; Jain, M. *Am J Epidemiol* 107(6): 499-509; 1978.

The effect of dietary factors on the development of breast cancer was studied in four areas of Canada. The study popu-ation, 100 patients with newly diagnosed breast cancer and an equal number of matched controls from each of the areas (total, 400 matched pairs), was administered personal and medical history questionnaires, a detailed dietary history questionnaire, a 24-hr recall for dietary information, and a 4-day diet record. In general, the mean nutrient intake was greater for breast cancer patients than for controls, and the mean nutrient intake for premenopausal breast cancer patients was greater than that for postmenopausal patients. In premenopausal breast cancer patients, total fat, saturated fat, and cholesterol showed risk ratios greater than unity; in postmenopausal patients, only total fat gave risk ratio estimates greater than unity. In all instances, there was little or no indication of a dose-response relationship. In premenopausal patients, when total fat was controlled for saturated fat and vice versa, the risk ratio for total fat became 2.0 and that for saturated fat became 0.4; the risk ratio for cholesterol was reduced when controlled for total fat and saturated fat. The findings on nutrient intake generally support the hypothesis that breast cancer in pre- and postmenopausal women is related to an increased dietary intake of fat. (39 refs)

78-3575 **An Epidemiologic Study of Breast Cancer.** (Eng) Choi, N. W. (Univ. Manitoba, Winnipeg, Manitoba, Canada); Howe, G. R.; Miller, A. B.; Matthews, V.; Morgan, R. W.; Munan, L.; Burch, J. D.; Feather, J.; Jain, M.; Kelly, A. *Am J Epidemiol* 107(6): 510-521; 1978.

An epidemiologic study of suspected risk factors in breast cancer was conducted in four areas of Canada. The study population, 100 patients with newly diagnosed breast cancer and an equal number of matched controls from each of the areas (total, 400 matched pairs), was administered medical and dietary questionnaires. There was evidence of an increased risk of breast cancer in postmenopausal women with younger age at menarche. The study confirmed that the increased risk of breast cancer in postmenopausal women with delay in age of natural menopause is most marked in those who develop breast cancer after the age of 70. However, it failed to confirm that late age at first pregnancy is a risk factor in either pre- or postmenopausal women. For all breast cancer respondents, irrespective of menopausal status, there was an increased frequency of pregnancies of 4 mo duration or less compared with controls; irregularity of periods occured more often in breast cancer patients who were still menstruating than in controls. The latter two findings suggest that women with breast cancer may be less fertile than their controls, tending to support the hypothesis that estrogenic stimulation in the absence of sufficient cyclic progesterone secretion may provide a setting favorable for the development of mammary carcinoma. There was no evidence that increased height or increased wt were risk factors in premenopausal women. Postmenopausal breast cancer patients were on the av taller than controls, but the difference was not significant. There was a significant difference between the wt of postmenopausal breast cancer patients and controls, with the former showing an increased wt both at the time of menopause and at 12 and 6 mo before the interview. This difference in wt was substantial for a group of 13 matched pairs aged 70 yr or more. (31 refs)

78-3576 **Risk Factors of Breast Cancer in Finland.** (Eng) Soini, I. (Detection Centre and Labs. Pirkanmaa Cancer Society, Univ. Tampere, Tampere, Finland). *Int J Epidemiol* 6(4): 365-373; 1977.

Reproductive, hormonal, and other characteristics associated with the risk of breast cancer were examined in a case-control study of 122 cases of breast cancer and 534 controls, all 41 to 60 yr old, in Finland. Finland represents an area of intermediate breast cancer incidence (40.1/100,000), a rate that is low compared with those in the other Nordic countries. Age at first marriage and at the birth of the first child, number of abortions, and parity adjusted for age at first birth were associated with breast cancer risk, whereas lactation was not. Breast cancer risk also showed a positive correlation with anamnestic angina pectoris but not with hypertension or diabetes. The results did not confirm the hypothesis that obesity influences the risk of breast cancer. (19 refs)

78-3577 **Estimates of Composite Risk Factors for Death from Breast Cancer.** (Eng) Davies, D. F. (Dept. Community Medicine, Coll. Medicine, Univ. Tennessee Center Health Sciences, 800 Madison Ave., Memphis, TN, 38163); Torng, A.; Morris, E. B.; Sebes, J. *J Natl Cancer Inst* 61(1): 41-48; 1978.

A method of ranking women according to their weighted composite risks of dying from breast cancer is described. The method uses the device of selecting presumed independent prognostic characteristics, standardizing reported relative risks by conversion to risk factors, and ranking the population of women in order of weighted composite risks. The following prognostic characteristics are employed in the method: family history of breast cancer, pregnancy history, history of benign breast disease, menstrual status, and history of annual breast examinations or recent self-examination of breasts. The proportion of the populations, risk ratios, and risk factors for each prognostic characteristic by mutually exclusive categories and by current 5-yr age groups are tabulated for women between the ages of 40 and 64 yr. For example, in one combination of 45- to 49-yr-old nulliparous women who had no family history of breast cancer, previous benign breast disease, surgical menopause at 40-44 yr of age, and no history of periodic breast examination, the composite risk factor (CRF) is 4.73. For women with these characteristics, the 10-yr risk is the product of the CRF and the av risk of dying from breast cancer in the next 10 yr, or 2,422/100,000. Women with composite risks as high or higher than 4.73 make up 3.1% of the population in the age group but will account for 20.2% of all the age group's breast cancer deaths during the succeeding 10 yr. Among 99.7% of women, the estimated CRF's range between 0.06 and 13.0. The validity and reliability of the method can be checked against data obtained in previous screening programs and in prospective studies that incorporate plans to study concordance of expected and observed data. (40 refs)

78-3578 **Horizontally Transmitted Lymphomas of Syrian Hamsters.** (Eng) Coggin, J. H. (Dept. Microbiology and Immunology, Univ. South Alabama, Mobile, AL, 36688); Thomas, K. V.; Huebner, R. *Fed Proc* 37(7): 2086-2088; 1978.

The occurrence of repeated epizootics in a hamster colony is reported, and attempts made to identify the agent responsible are summarized. The first epizootic was characterized by the development of lymphomas in half a colony of 2,900 male and female LSH, LCG, and LVG hamsters. Tumors were rare before 8 wk of age except among LSH hamsters (13% incidence). Most tumors were noted in 14- to 19-wk-old animals born at the facility or introduced as weanlings. When hamster >8 wk of age were housed in the diseased colony, tumors were less common and they appeared after 25 wk of age. Age matched and sibling hamsters kept 25 miles away remained free of disease. The diseased animals were killed, cleaning and sanitization methods were applied, and a new hamster colony was started. Despite continued sanitization, lymphomas began to appear within 7 mo, predominantly in the intestine. Younger animals introduced into the colony were more susceptible to tumor development than older animals. Tumor incidence in newborn often exceeded 60%/liter. The second colony was killed, the facility again sanitized, and a new colony started. Lymphomas developed in these animals within 4 mo. Other facilities receiving small numbers of hamsters from these infected colonies reported similar epizootics. The tumors were characterized as malignant lymphomas of B-cell origin that invade adjacent tissues and spread to distant sites; death usually resulted within 2-5 wk. A protamine sulfate extract of the tumor induced 50%-80% lymphomas in neonates infected ip. Electron microscopy of the extract did not reveal virus particles. Its infectious capacity could be destroyed by DNase. (3 refs)

78-3579 **High Incidence Area of Cattle Cancer with a Possible Interaction Between an Environmental Carcinogen and a Papilloma Virus.** (Eng) Jarrett, W. F. (Univ. Glasgow Veterinary Sch., Bearsden, Glasgow, England); McNeil, P. E.; Grimshaw, W. T.; Selman, I. E.; McIntyre, W. I. *Nature* 274(5668): 215-217; 1978.

Eighty cases of alimentary cancer in beef cattle from the upland areas of Scotland and northern England were studied. Cattle in these areas are substantially more prone to alimentary cancer than those in the immediately neighboring lowlands. Four types of neoplasms were found: squamous carcinoma and papilloma of the upper tract, and adenoma and adenocarcinoma of the intestine. Squamous carcinomas were found in the lateral dorsum of the tongue, the soft palate and oropharynx, the esophagus, the esophageal groove, and a specific area of the rumen. Papillomas were found in the same sites in 96% of the cattle with squamous carcinomas and in 144/366 of normal cattle from the same region. The number of papillomas per animal was markedly higher among those with cancer than among those without. Several cases showed incontrovertible evidence of the direct transformation of papillomas to carcinomas. Two-thirds of the cattle of known age were aged 12 yr or less. In all cases in which the farms of origin were traced (75%), they contained marginal or hill land infested with bracken. Thirteen percent of the papillomas contained virus that produced typical papillomas after inoculation into the esophagus, palate, or skin. This system provides a means for studying the way in which an oncogenic virus, an environmental chemical (in bracken), and a host cell genome interact to give rise to malignancy. (14 refs)

78-3580 **Epidemiology of Cancer Caused by Occupational Exposure to Harmful Chemicals in East Germany.** (Ger) Braunlich, A. (Zentralinstitut fur Arbeitsmedizin der DDR, Noldnerstrasse 40-42, DDR-1134 Berlin, E. Germany); Enderlein, G.; Haublein, H. G.; Konetzke, G.; Rebohle, E.; Schmidt, P.; Schulz, G.; Thiele, H.; Wallenstein, G. *Z Gesamte Hyg* 24(4): 237-247; 1978.

Epidemiological surveys of occupation-related morbidity and mortality in East Germany are reviewed. According to the National Cancer Registry, approx 40,000 persons die of malignancies each year and nearly 60,000 new cases are reported. Surveys of working populations, obtained by medical tests and routine examinations, revealed a connection between health hazards and harmful chemicals. Various respiratory cancers resulted from occupational exposure to asestos, arsenic, nickel, chromium, polycylic aromatic ompounds and other chemicals. (20 refs)

78-3581 **The Hazards of a Chemical Laboratory Environment--A Study of the Mortality in Two Cohorts of Swedish Chemists.** (Eng) Olin, G. R. (Safety and Health Dept., Royal Inst. Technology, S-100 44, Stockholm, Sweden). *Am Ind Hyg Assoc J* 39(7): 557-562; 1978.

The mortality of 857 chemists educated at two technology institutes in Sweden from 1930 to 1950 and followed until 1974 was studied. There were 93 deaths vs an expected (exp) number of 106, but a significant number of deaths were due to malignant tumors (34 vs 20.8 exp). The increase was accounted for partly by leukemias and malignant lymphomas (8 cases vs 2.6 exp): there were 3 cases of Hodgkin's disease (vs 0.5 exp), 3 lymphosarcomas, and 2 myelogenous leukemias. Prostatic cancer also contributed to the increase (4 cases vs 0.8 exp), and there were three cases of kidney cancer and one of bladder cancer. The frequencies of the other types of tumors did not diverge significantly from the exp number. However, in a separate analysis of the mortality pattern within 24 yr after graduation (in which the class of 1950 was excluded), an increase in brain tumors was found (3 cases vs 0.6 exp). The increased incidence of certain tumors might indicate that there is a relation between these types of tumors and exposure to organic chemicals. (18 refs)

78-3582 **Sunlight as a Risk Factor of Malignant Melanoma of the Skin.** (Eng) Teppo, L. (Finnish Cancer Registry, Liisankatu 21 B, SF-00170 Helsinki 17, Finland); Pakkanen, M.; Hakulinen, T. *Cancer* 41(5): 2018-2027; 1978.

A series of 2,511 malignant melanomas diagnosed in Finland from 1953 to 1975 was analyzed for possible relationships between sunlight and tumor induction. The age-adjusted incidence rate more than doubled for both sexes during the 20-yr study period, reaching $3.5/10^5$ in the early 1970's. The most common tumor sites were the trunk and lower limbs in men (48% and 17%, respectively) and the lower limbs and the trunk in women (56% and 28%, respectively). The age-adjusted incidence rates were higher in urban areas than in rural districts and higher in southern than in northern Finland. However, the north-south differences almost disappeared when the rates were adjusted for the urban/rural population ratio. Thus, the north-south gradient was attributable to urbanization and not to latitude alone. Tumors on the trunk in both sexes and those on the legs in women increased markedly with time; their age-specific rates increased sharply in middle age and then leveled off. Tumors on the head and neck in both sexes and those on the legs of men did not increase with time; the risk of these tumors was low in middle age but continuous throughout life. The increased melanoma risk with time is in accord with cohort analyses, which show that successive generations born between the late 1800's and 1930-1940 have been exposed increasingly to factors associated with melanoma development. The anatomical distribution of the melanomas in this series suggests that sunlight is not the only important risk factor. (21 refs)

78-3583 **Theoretical Aspects of Radiation Carcinogenesis: Cell Survival Dependent Dose-Rate Effects.** (Eng) Wells, J. (Central Electricity Generating Board, Berkeley Nuclear Lab., Berkeley, Gloucestershire, GL13 9PB, England). *J Institution Nuclear Engineers* 19(3): 89-97; 1978.

A cellular response function (CRF) for radiation carcinogenesis is presented and used in an illustrative study of cell survival-dependent dose rate effects, with particular reference to point source β-particle emitters. Parameters for a CRF for carcinogenesis are derived from human, animal, and in vitro data. The CRF is integrated to make predictions about the macroscopic response of organs and tissures to radiation. Previous mathematical methodology is considered in terms of a more general form of CRF that incorporates parameters that can be associated readily with all major variables. Such a function can be developed from established radiobiological data and can, in turn, be used as a planning criterion for experiments on cell transformation in vitro and tumor induction in animals. (19 refs)

78-3584 **Point of View--Misleading Reports on Japanese Incidence of Prostatic Cancer.** (Eng) Ravich, A. (1135-103rd St., Miami Beach, FL, 33154). *Urology* 11(5): 542; 1978.

Differences in the prostatic cancer incidence in Japan (2%) and the US (20%) are partially the result of the method of reporting: in the US, the number of cases is compared with the number of prostatectomies, but in Japan the number of cases is compared with the number of male patients in the urology clinic. There is small number of prostatectomies in Japan, the result of reluctance of patients to go to a urologist. It is suggested that prostatic cancer is a viral venereal disease involving the prostate and smegma. (3 refs)

See also:
*(Rev.): 78-3001, 78-3009, 78-3012, 78-3019, 78-3021, 78-3022, 78-3025, 78-3029, 78-3030, 78-3032, 78-3033, 78-3037, 78-3038.
*(Chem.): 78-3069, 78-3079, 78-3090, 78-3091, 78-3093, 78-3100, 78-3106, 78-3108, 78-3110.
*(Phys.): 78-3250, 78-3256, 78-3264.
*(Path.): 78-3481, 78-3491, 78-3493, 78-3500, 78-3505, 78-3506, 78-3528, 78-3534, 78-3546, 78-3553.

MISCELLANEOUS

78-3585 **Characterization of a Unique Cell Line (LAZ 221) from Human Acute Lymphocytic ("Null" Cell) Leukemia.** (Eng) Lazarus, H. (Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA, 02115); Barell, E. F.; Krishan, A.; Livingston, D. M.; Harris, K.; Schlossman, S. F.; Chess, L. *Cancer Res* 38(5): 1362-1367; 1978.

The establishment of a cell line, LAZ 221, from a 24-yr-old woman with acute lymphocytic leukemia of the null cell type is reported. After 3 mo, the cultures attained a doubling time of ≥4 days and had max viable cell densities of 4-5 x 10^6 cells/ml. Agar culture indicated that the line was free of *Mycoplasma* after 18 mo in culture. The cell line does not possess the Epstein-Barr virus nuclear antigen, and it has a karyotype of 45,XX,-9,-12,+t(9q12q). Both the cultured cells and the patient's uncultured blast cells shared the same phenotypic markers, and they both lacked T- and B-cell markers. They failed to form sheep RBC rosettes, they did not react with T-cell-specific antisera (TH_1-, HTL-), they did not form rosettes with RBC sensitized with antibody and complement, and they were surface immunoglobulin negative. However, they did possess a histocompatibility antigen (HLA-D)-related glycoprotein complex of 23,000-30,000 daltons, which was shown to have properties of an Ia-like antigen. This cell line is thus representative of about 75% of all human acute lymphocytic leukemias. (24 refs)

78-3586 **Growth Promoting and Inhibitory Activities of 3T3 and Other Cell Lines for Thiol-dependent Lymphoma Cells In Vitro.** (Eng) Tanapat, P. (Dept. Pathology, State Univ. New York, Downstate Medical Center, Brooklyn, NY, 11203); Gaetjens, E.; Broome, J. D. *Proc Soc Exp Biol Med* 157(3): 517-521; 1978.

The effects of various 3T3 cells (simian virus 40-transformed Swiss, spontaneously transformed BALB/c, Swiss, BALB/c) on the growth of the thiol-dependent lymphoma line L1210(V) were determined. All cells, regardless of whether they showed contact inhibition or were transformed, supported the growth of L1210(V) cells. However, these supporting cells do not condition the medium permanently. At high cell densities, growth promotion was diminished. These results were compared to other findings with macrophage cultures that had previously been shown to support the growth of mouse lymphoma cells. Macrophages produced approx the same degree of L1210(V) growth promotion as did the other lines, but as the cell density of the macrophages increased, lymphoma cell growth became increasingly more rapid (2.8 divisions at 2 x 10^6 cells). The mechanisms of this growth promotion were then studied further. Supernatants of macrophage cultures that had been able to sustain L1210(V growth were unable to do so. Once mixed cultures of macrophages and L1210(V) cells were established, however, lymphoma cell growth was supported. Supplements of fresh medium every 48 hr, to reduce the lymphoma cell density to its original size, resulted in no increase in cell number unless thiol was added. Diffusion chamber experiments indicated that macrophages and 3T3 cells, but not supernatants from macrophage cultures, supported lymphoma growth. These experiments indicate that growth promotion is mediated humorally. Studies with the L1210(A) thiol-independent subline indicated that the growth failure of L1210(V) in supernatant medium was distinct from the thiol dependency. Like L1210(V) cells, this subline failed to grow with 3T3 cells at high density or with the 3T3 culture supernatant. It is suggested that a nondialyzable inhibitor of lymphoma cell growth is produced by 3T3 cells. (9 refs)

78-3587 **Isolation of a Bone-resorptive Factor from Human Cancer Ascites Fluid.** (Eng) Nimberg, R. B. (Dept. Biochemistry, Boston Univ. Sch. Medicine, Boston, MA, 02118); Humphries, D. E.; Lloyd, W. S.; Badger, A. M.; Cooperband, S. R.; Wells, H.; Schmid, K. *Cancer Res* 38(7): 1983-1989; 1978.

A protein fraction that induces the resorption of bone explants in organ culture was isolated from the ascitic fluid of patients with advanced cancer metastatic to the peritoneal cavity who had not yet received any chemotherapy or radiotherapy. Partial purification was achieved by gel filtration, affinity chromatography, and ion-exchange chromatography. The isolated fraction, the components of which have an apparent mol wt of 60,000, was found to be heterogeneous by disk gel electrophoresis and to be composed primarily of proteins with relatively acidic electrophoretic properties. The specific bone-resorptive activity of this protein fraction was greatly increased over that of the unfractionated starting material, but the activity was completely destroyed by incubation with pronase and by heating. Immunoassay and extraction procedures showed that the bone-resorptive action was not due to the presence of parathyroid hormone, prostaglandin E_2, or vitamin D-like sterols. In parallel experiments with the supernatants of phytohemagglutinin-stimulated normal human peripheral WBC, a protein fraction with a mol wt of 60,000, which resembled the resorptive fraction isolated from cancer ascites fluid and which contained significant bone-resorptive activity, was also partially purified. If the bone-resorptive factor in tumor ascites fluid is involved in the production of tumor-related osteolytic lesions and tumor-related hypercalcemia, the purification and chemical ıd biological characterization of this protein could be a maır step toward understanding the mechanism by which tuıors destroy bone and alter calcium metabolism. (28 refs)

8-3588 **Production of a Corpus Luteum Angiogenic Factor Responsible for Proliferation of Capilaries and Neovascularization of the Corpus Luteum.** (Eng) ɔospodarowicz, D. (Cancer Res. Inst., Univ. California, San ⁼rancisco, CA, 94143); Thakral, K. K. *Proc Natl Acad Sci USA* 75(2): 847-851; 1978.

The abilities of ovarian corpus luteum and of follicles to inluce neovascularization were compared by implanting these issues in rabbit cornea, a transparent, avascular substratum n which the rates of new vessel formation and the parts played by the host and the ovarian implant can be easiliy studied. The corpus luteum was capable of inducing neovascularization but the follicles were not. This is in agreement with the in vivo situation, in which active capillary proliferation is observed only with corpus luteum, whereas follicles are an avascular structure. Corpus luteum crude extracts were potent stimulators of the initation of DNA synthesis in BALB/c 3T3 cells and in bovine vascular endothelial cells. Follicular extracts were inactive. This supports the hypothesis that corpora lutea can secrete an angiogenic factor, corpus luteum angiogenic factor (CLAF). Thus, although the initial stimulus for the neovascularization is probably the increased demand for oxygen of the luteal cells caused by their high metabolic activity, the factor involved in inducing the proliferation of capillaries is a mitogenic factor (CLAF) produced by the corpus luteum. (21 refs)

78-3589 **Density-dependent Mitosis in the Walker 256 Carcinoma and the Influence of Host Age on Growth.** (Eng) Bellamy, D. (Dept. Zoology, Univ. College, Cardiff CF1 1XL, Wales); Hinsull, S. M. *Eur J Cancer* 14(7): 747-751; 1978.

The Walker 256 carcinoma was tested for density dependence of cell division, and the effect of host age on growth regulation was examined. Tumor tissue (6-mg fragment) from 20-wk-old donor rats bearing a solid, sc Walker 256 carcinoma was inserted sc into the middorsal region of 20- or 120-wk old male rats of an inbred WAB substrain. Colchicine was administered ip 4 hr before the animals were killed (3-10 days following tumor implantation), and the tumor tissue was removed. There was no difference in tumor size between the two age groups until 6 days after transplantation. Thereafter, the tumor growth rate in the older group was lower than that in the 20-wk-old animals. Overall tumor cell proliferation in 120-wk-old animals was much lower than that observed in 20-wk-old hosts. The magnitude of this difference increased from 100% 3 days after transplantation to 300% at 6 days. Therefore, a significant difference in tumor cell proliferation between the two groups occurred before any difference in tumor wt became apparent. The mitotic rate declined with increasing cell density, and it was more dependent upon cell density in tumors of old hosts. The mitotic pattern of tumors of old hosts was closer to that observed in normal tissue, implying a better regulation. (21 refs)

78-3590 **Collagenolytic Activity and Spreading Mode of Human Stomach Cancer.** (Jpn) Itoh, K. (First Dept. Surgery, Okayama Univ., Medical Sch., Shikada-cho, Okayama 700, Japan). *Okayama Igaku Zasshi* 89(9/10): 1101-1117; 1977.

The degradation and resorption of collagen in malignant gastric tumors were investigated by the Gross and Lapiere method, which involves tissue incubation with an animal collagenolytic enzyme that acts at neutral pH and physiologic body temperature. Histological observations were performed simultaneously to study the relationship between tumor collagenolytic activity and morphology. The collagenolytic activity was significantly higher in gastric cancer tissue than noncancerous gastric tissue. In the cancers, the collagenolytic activity was elevated at the spreading front. Although not as high as in the cancer tissue, the collagenolytic activity of the surrounding tissues was higher than normal. Well-differentiated adenocarcinomas showed relatively high collagenolytic activity in their centers and spreading fronts. In poorly differentiated adenocarcinomas, the activity was low in the center, but it gradually increased in the spreading front. There was no difference in collagenolytic activity between the center and spreading fronts of massive, continuous-type lesions; in the nonmassive interspersed and nonmassive continuous types, however, there was a great difference in activity between the spreading front and the center of the lesion. Borrmann type I tumors showed the characteristic distribution of collagenase activity; ie, high in the lesion but low in the surrounding region. In contrast, in Borrmann type IV tumors it was elevated throughout the stomach. (37 refs)

78-3591 **Collagenase in Malignant Tumors. 2. Collagenolytic Activity and Its Relation to the Development of Human Stomach Cancer.** (Jpn) Mimura, T. (First Dept. Surgery, Okayama Univ., Medical Sch., Schikada-cho, Okayama 700, Japan). *Okayama Igaku Zasshi* 89(9/10): 1165-1176; 1977.

The collagenolytic activity of 38 human stomach cancers was measured by the culture methods of Gross and Lapiere, and histological observations were made simultaneously to examine the relationship between this activity and the morphological pattern of the cancer. Collagenolytic activity was significantly higher in gastric cancer tissues than in normal stomach tissues. The strongest activity was observed in the spreading front and the center of the gastric cancers. In the surrounding tissues, the activity was only slightly higher than normal. The more poorly differentiated the tumor, the higher the collagenolytic activity; and in poorly differentiated adenocarcinomas, the activity was stronger in medullary than in scirrhous tumors. Borrman Type IV cancer showed elevated collagenolytic activity throughout the lesion. In contrast, this activity was weak in 0- and 5-type cancers. In the superficial spreading type of early gastric cancer, the collagenolytic activity was associated with the horizontal direction of cancer development. (20 refs)

78-3592 **Isolation and Partial Purification of Cytochrome P-450 from Uninduced Rat Liver.** (Eng) Dialameh, G. H. (Dept. Biochemistry, Faculty Basic Medical Sciences, Univ. Tehran, Tehran, Iran). *Biochem Biophys Res Commun* 81(1): 19-23; 1978.

The isolation of cytochrome P-450 from uninduced male rat liver is reported. The specific level of cytochrome P-450 was 9.6 nanomoles/mg protein; it was stable in 20% glycerol, and it could be stored for 2 wk at -20 C without spectral changes. The cytochrome catalyzed the NADPH-dependent hydroxylation of benzo(a)pyrene when it was mixed with an NADPH-cytochrome c reductase preparation and purified microsomal lipid. (21 refs)

78-3593 **Dissociation of Plasminogen Activator from the Transformed Phenotype in a 5-Bromodeoxyuridine Dependent Mutant of Syrian Hamster Melanoma Cells.** (Eng) Rosenthal, S. L. (Div. Human Genetics, Children's Hosp. Medical Center, Boston, MA, 02115); Zucker, D.; Davidson, R. L. *J Cell Physiol* 95(3): 275-285; 1978.

The relationship between plasminogen activator levels and the expression of the transformed phenotype was studied in a 5-bromodeoxyuridine (BUdR)-dependent mutant of Syrian hamster melanoma cells. The BUdR-dependent cells resemble transformed cells when grown in the presence of BUdR, untransformed cells when grown in its absence. The BUdR-dependent cells released significant levels of plasminogen activator only when cultured in the absence of BUdR. In the presence of BUdR, the release of plasminogen activator by the dependent cells was suppressed, and the decreased level of plasminogen activator released in the presence of BUdR appeared to be due to decreased production of active enzyme. Growth tests indicated that when BUdR-dependent cells were attached to a substrate, BUdR was required to attain high densities. Furthermore, the cells were able to grow well in soft agar only in the presence of BUdR. These findings suggest that the production and release of high levels of plasminogen activator are not related to expression of the tran formed phenotype in the BUdR-dependent cells. Cells cu tured in medium containing dog serum as a plasminoge source exhibited morphological alteration, but only in th presence of BUdR. The morphological response of the cel to dog serum resembled that previously observed wit virus-transformed cells. In the BUdR-dependent cells, th morphological response to dog serum appeared to be correla ed with the release of plasminogen activator but separate from other transformed characteristics. (21 refs)

78-3594 **Genetic Transcription of Human Leukem Cells.** (Eng) Sawada, H. (Dept. Internal Med cine, Faculty Medicine, Kyoto Univ., Sakyo-ku, Kyoto 60 Japan); Tashima, M. *Acta Haematol Jpn* 40(6): 1117-113 1977.

Chromatins isolated from human leukemic WBC were ch racterized with respect to chemical composition, therm denaturation, template activity, transcription products DNA-RNA hybridization, and the amount of unique DN sequences transcribed. Chromatins from patients with acu lymphoblastic leukemia, acute myeloblastic leukemia, lymphosarcoma had higher thermal denaturation values an contained more nonhistone chromosomal protein tha chromatins from patients with chronic lymphocytic leukem (CLL) or normal subjects. The acute leukemia chromati also had higher template activity than CLL and normal lyn phocyte chromatins. The repetitive DNA transcripts fro CLL and normal lymphocyte chromatin templates were e sentially the same. In hybridization experiments with uniq ^{3}H-DNA, the RNA's from all leukemic WBC had essential the same saturation value (2.5%), indicating that the cel transcribed a similar amount of the unique genome. (28 ref

78-3595 **Stimulation of Thymidine Incorporation Mouse Liver and Biliary Tract Epithelium Lithocholate and Deoxycholate.** (Eng) Bagheri, S. A. (Liv Study Unit, Dept. Medicine, Univ. Chicago, Chicago, IL Bolt, M. G.; Boyer, J. L.; Palmer, R. H. *Gastroenterolo* 74(2, part 1): 188-192; 1978.

The effect of a single po dose of various bile salts on thym dine (TdR) incorporation in female white mouse (CF1) liv and biliary tract epithelium was investigated. The time cour of TdR incorporation after a single 1-mg/kg dose of sodiu lithocholate revealed a marked increase in incorporation in biliary tract DNA at 22-28 hr, but no increase in incorpor tion into liver DNA until 36 hr. For both tissues, the thres old dose for sodium lithocholate was approx 12.5 mg. T results for the same doses of other bile salts indicated th deoxycholate induced more incorporation into biliary tra and liver DNA than did lithocholate or cholate. Chenodeox

olate did not enhance incorporation in either tissue. Fol-
wing the administration of 1 mg/kg lithocholate, liver tis-
e showed areas of focal necrosis randomly distributed in
e parenchyma. Neither deoxycholate- nor cholate-treated
iimals had necrotic areas, but there was an increase in the
ımber of hepatocytes undergoing mitosis. Chenodeoxycho-
te, ursodeoxycholate, and hyodeoxycholate had no effect.
is suggested that these bile acid-induced changes could be
'importance in hepatic, biliary, or intestinal carcinogenesis.
2 refs)

8-3596 **Repair of Alkylated DNA in Chinese Hamster
Cells Measured by Loss of Enzyme-sensitive
ites in Isolated DNA.** (Eng) Shackleton, J. (Inst. Cancer
.es., Pollards Wood Res. Station, Nightingales Lane, Bucks,
'halfont St. Giles, England); Roberts, J. J. *Biochem Biophys
'es Commun* 81(4): 1168-1174; 1978.

procedure was developed for measuring the induction and
epair of alkylation-induced enzyme-sensitive damage in
)NA in Chinese hamster cells. Lesions induced in Chinese
amster cell DNA by dimethyl sulfate (DMS) were recog-
ized by an enzyme present in crude extracts of *Micrococcus
iteus*. These enzyme-sensitive sites on the alkylated DNA
'ere lost with a half-life of 2.4 hr during the first 6 hr after
eatment, and they disappeared completely by 24 hr. The
umber of the enzyme-sensitive sites in methylated DNA
10% of the estimated 100 methylation sites in a DNA
f 10^8 daltons), their rate of loss, and their heat lability
re consistent with the majority being 3-methyladenine
dducts of DNA. (21 refs)

8-3597 **Detection of Inapparent Nodule-transformed
Cells in the Mammary Gland Tissues of Virgin
'emale BALB/cfC3H Mice.** (Eng) DeOme, K. B. (Dept.
'oology, Univ. California, Berkeley, CA, 94720); Miyamoto,
1. J.; Osborn, R. C.; Guzman, R. C.; Lum, K. *Cancer Res*
8(7): 2103-2111; 1978.

napparent nodule-transformed cells were recovered from
orphologically normal mammary tissue from virgin female
ALB/cfC3H/Crgl (mouse mammary tumor-positive) mice
efore the appearance of hyperplastic alveolar nodules
HAN) or tumors by a cell-dissociation technique. Mammary
issues were dissociated by collagenase (0.1%), hyaluronidase
).1%), and pronase (1.25%). Aliquots of 10^5 viable cells in
.01 ml medium were injected into the gland-free mammary
at pads of 3-wk-old female syngeneic mice. After 10 wk, the
utgrowths were examined and classified as ductal, HAN,
ımorous, or combinations of these types. The presence of
IAN outgrowths indicated the presence of nodule-trans-
ormed cells in the donor mammary tissues. Nodule-trans-
ormed cells were recovered from 2-mo-old donors, and the
umber of HAN outgrowths increased with donor age. Overt
HAN and tumors did not appear in virgin female BALB/cfC3H/Crgl mice $<$8-9 mo of age. The data suggest that inapparent nodule-transformed cells were present long before the appearance of overt HAN or tumors and that the numbers of transformed cells increased with donor age. (25 refs)

78-3598 **Suppression by *Nocardia Rubra* Cell Wall Skeleton of Mammary DNA Synthesis, Plasma Prolactin Level, and Spontaneous Mammary Tumorigenesis in Mice.** (Eng) Nagasawa, H. (Pharmacology Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104, Japan); Yanai, R.; Azuma, I. *Cancer Res* 38(7): 2160-2162; 1978.

The effects of the cell wall skeleton (CWS) of *Nocardia rubra* on mammary gland DNA synthesis, plasma prolactin levels, and spontaneous mammary tumorigenesis in female SHN mice were investigated. The mice received sc injections of 100 μg CWS every 7 days between 2 and 12 mo of age. Whereas mammary tumors began to appear before 5 mo of age in control mice, they did not appear until 7 mo in CWS-treated mice. The incidence increased with age in both groups, but it was significantly lower in the CWS-treated mice than in controls throughout the experiment except at 9 and 12 mo of age. The onset age of mammary tumors was significantly higher in SWS-treated mice (8.9 ± 0.4 mo) than in controls (7.2 ± 0.2 mo). Treatment of mice with CWS also reduced normal mammary gland DNA synthesis and prolactin levels in the circulation; both of these parameters are primary factors in mammary tumorigenesis. The fact that there was no difference between CWS-treated mice and controls in terms of mammary tumor incidence at 12 mo of age infers that the limit of inhibitory activity of CWS on spontaneous mammary tumorigenesis in mice is largely controlled by genetic factors. (20 refs)

78-3599 **DNA Synthesis in Human Benign Breast Tumors 0, 15, and 30 Days After Transplantation to Athymic "Nude" Mice: Stimulation by Human Placental Lactogen (HPL) (Meeting Abstract).** (Eng) McManus, M. J. (Dept. Anatomy, Michigan State Univ., E. Lansing, MI, 48824); Patten, S. E.; Schwartz, J. E.; Pienkowski, M. M.; Welsch, C. W. *Proc Am Assoc Cancer Res* 19: 74; 1978. (no refs)

78-3600 **Stimulation of Mammary Epithelial Growth by Free Fatty Acids and Changes in this Response During Neoplastic Progression (Meeting Abstract).** (Eng) Hosick, H. L. (Dept. Zoology, Washington State Univ., Pullman, WA, 99164); Angello, J. C.; Anderson, M. E. *Proc Am Assoc Cancer Res* 19: 178; 1978. (no refs)

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ABBREVIATIONS

JOURNAL names are abbreviated according to the *List of Journals Indexed in Index Medicus, Abbreviation Listing*. If the journal is not listed in this, abbreviations are derived from the *International List of Periodical Title Word Abbreviations.*

LANGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

ABBREVIATIONS used in abstracts:

A	angstrom(s)
ACTH	adrenocorticotropic hormone
ADP	adenosine diphosphate
AMP	adenosine monophosphate
ATP	adenosine triphosphate
approx	approximately
av	average
BCG	bacillus Calmette-Guerin
bid	twice daily
C	degree(s) centigrade
cal	calorie(s)
kcal	kilocalorie(s)
cc	cubic centimeter(s)
Ci	curie(s)
mCi	millicurie(s)
μCi	microcurie(s)
cm	centimeter(s)
CNS	central nervous system
cpm	counts per minute
DNA	deoxyribonucleic acid
ED_{50}	median effective dose
EDTA	ethylenediamine tetraacetic acid
g	gram(s)
kg	kilogram(s)
mg	milligram(s)
μg	microgram(s)
Hb	hemoglobin
hr	hour(s)
ia	intra-arterial
id	intradermal
IgA	Immunoglobulin A
IgB	Immunoglobulin B
IgG	Immunoglobulin G
IgM	Immunoglobulin M
ILS	increased life span
im	intramuscular
ip	intraperitoneal
IU	International Unit(s)
iv	intravenous
Km	Michaelis constant
LD	lethal dose
LD_{50}	median lethal dose
M	molar
μM	micromolar
mOsm	milliosmolar
max	maximum
mEq	milliequivalent(s)
min	minute(s)
ml	milliliter(s)
μl	microliter(s)
mm	millimeter(s)
mo	month(s)
mol wt	molecular weight
N	normal concentration
NAD	nicotinamide adenine dinucleotide
NADH	reduced nicotinamide adenine dinucleotide
NADP	nicotinamide adenine dinucleotidephosphate
NADPH	reduced nicotinamide adenine dinucleotidephosphate
NCI	National Cancer Institute
NIH	National Institutes of Health
PAS	periodic acid-Schiff
po	orally
ppb	parts per billion
ppm	parts per million
qid	four times daily
qod	every other day
QO_2	oxygen quotient
R	roentgen
RBC	red blood cells (erythrocytes)
RNA	ribonucleic acid
rpm	revolutions per minute
sc	subcutaneous
sec	second(s)
SGOT	serum glutamic-oxaloacetic transaminase
SGPT	serum glutamic-pyruvic transaminase
soln	solution
TCD	tissue culture dose
TCD_{50}	median tissue culture dose
tid	three times daily
UV	ultraviolet
WBC	white blood cells (leukocytes)
wk	week(s)
wt	weight
X	times
yr	year(s)

8-3601 **Possibilities and Limitations of the Evaluation of Chemical Carcinogenesis.** (Ger) Schramm, T. Zentralinstitut fur Krebsforschung der AdW der DDR, Beran, E. Germany); Teichmann, B. *Sitzungsber Akad Wiss DDR* (11 N): 13-17; 1978.

'he carcinogenicity of some compounds varies from species o species, making extrapolation from animals to humans dificult. In addition, cocarcinogenic, synergistic, and protective actors usually exist in the human environment, making exrapolation even more difficult. An improved reporting sysem that links human cancer incidence with carcinogen expoure is needed. (8 refs.)

'8-3602 **Proper Perspectives in Extrapolation of Experimental Carcinogenesis Data to Humans.** (Eng) Craybill, H. F. (NCI, Bethesda, MD, 20014). *Food Technolo-y* 32(8): 62-64; 1978.

'he question whether data from animal studies are reliable redictions of carcinogenic events to humans is explored. All hemicals that have been shown to be carcinogenic to humans ave been ultimately verified as carcinogens in animals except enzene and arsenic (As). These chemicals are either noncarinogenic to experimental animals or there is no appropriate nimal model or, in the case of As, the proper compound emains to be tested. Several accepted predictive factors, inluding reproducibility of animal tests in various species and trains with multiple confirmations in different laboratories, lose-dependent increase of tumor incidence, experimental loses approximating human exposure, are listed. If animal lose-response data are augmented by significant biochemical, harmacokinetic data, the probability of translating events o humans becomes more meaningful. Also listed are some f the limiting factors in extrapolation, such as inappropriate oute of administration; improper test species and strains; ole of diet, nutrition, and diet contaminants; and contaminants in test chemicals. Animal data are presumptive evilence that should imply some element of concern as an inlicator test system. Epidemiological studies can provide lirect evidence, but these investigations may not reveal actual exposures and they may only provide observed effects long fter exposure. (8 refs.)

'8-3603 **Criteria for Selecting Chemical Compounds for ,Carcinogenicity Testing: An Essay.** (Eng) Aros, J. C. (Seamen's Memorial Res. Lab., U.S. Public Health Service Hosp., 210 State St., New Orleans, LA, 70118). *J Environ Pathol Toxicol* 1(4): 433-458; 1978.

Criteria for selecting suspected chemicals for carcinogenicity testing are described. The structural criteria are based on analogies with known types of chemical carcinogens. Operational criteria, which are complementary to structural criteria, are the set of biofunctional capabilities in organic compounds likely to bring about or lead to ultimately inheritable alterations in cellular synthetic templates. The 'guilt by association' criterion states that if a compound belongs to a chemical class that includes many potent and multitarget carcinogens, it should be assayed under more stringent conditions than the standard conditions of bioassay testing. The 'after the fact' criterion uses prior human epidemiological indications as the basis for selecting chemical agents for carcinogenicity testing. In determining the significance of bioassay data obtained by testing individual compounds, the possible complicating effects of other environmental carcinogens and noncarcinogens, viral agents, and factors such as nutrition, age, and stress should be considered. A flexible approach is needed for the establishment of standards and guidelines applicable to categories and types of chemical agents rather than individual compounds. (104 refs.)

78-3604 **Quantitative Theories of Oncogenesis.** (Eng) Whittemore, A. S. (Hunter Coll., City Univ. New York, New York, NY). *Adv Cancer Res* 27: 55-88; 1978.

Several quantitative theories of the origin of neoplasms are examined both qualitatively and quantitatively, by comparing their predictions with epidemiological and experimental observations. These theories, which relate the frequency and time of occurrence of tumors to the temporal and spatial distribution of oncogenic exposure, are the single-stage theory of Iverson and Arley, the multicell theory of Fisher and Hollomon, the multistage theory with negligible cell loss, the multistage theory with nonnegligible cell loss, and the multistage theory with proliferative advantage of intermediate cells (including variations by Armitage and Doll and by Fisher). Because these theories do not postulate alteration of tumor growth rates by carcinogenic exposure, none of them can account for the tumor regression that occurs after termination of promoter painting or the lack of tumors that accompanies short exposures to high doses of promoter. Therefore, the single stage or multistage theory with variation in transformed cell types was developed that generalizes many of the previous theories and, by including variable tumor growth that may be affected by exposure, provides a framework for examining initiation-promotion data. Most of these theories ignore the crucial distinction between benign and malignant tumors and none include the possibility of cell repair or the action of the host's immune system. However, despite their limitations, the single and multistage theories provide a flexible, broad, and biologically plausible framework in which to examine the gross behavior of oncogenesis data. (41 refs.)

78-3605 **Some Current Perspectives on Chemical Carcinogenesis in Humans and Experimental Animals: Presidential Address.** (Eng) Miller, E. C. (McArdle Lab. Cancer Res., Univ. Wisconsin Medical Center, Madison, WI, 53706). *Cancer Res* 38(6): 1479-1496; 1978.

Current concepts of chemical carcinogenesis in humans and experimental animals are reviewed. Topics discussed include: tumor induction as a multistage phenomenon; electrophilicity as a common property of ultimate carcinogens; examples of the metabolic activation and reactivity of chemical carcinogens; possible molecular mechanisms of chemical carcinogenesis; prevention of the promotion of initiated cells; and extrapolation of the basic knowledge of chemical carcinogenesis to the prevention of human cancer. Regarding the molecular mechanism of carcinogenesis, examples are cited to illustrate that metabolic activation is an essential step in the induction of neoplasia by most chemical carcinogens. The electrophilic ultimate carcinogens can react, probably more or less indiscriminately, with a number of nucleophilic sites in DNA, RNA, and proteins. Thus, the strong electrophilic nature of ultimate carcinogens is consistent with both genetic and epigenetic mechanisms of carcinogenesis or with mechanisms that include both genetic and epigenetic events. These mechanisms may or may not involve the expression of oncogenic viral information. Chemicals generally recognized as being carcinogens in humans include: 2-naphthylamine, benzidine (4,4′-diaminobiphenyl), 4-aminobiphenyl, 4-nitrobiphenyl, bis(chloromethyl) ether, bis(2-chloroethyl) sulfide, vinyl chloride, certain soots, tars, and oils, chromium compounds, nickel compounds, asbestos, cigarette smoke, betel nut or tobacco quids, N,N-bis(2-chloroethyl)-2-naphthylamine, and diethylstilbestrol. (235 refs.)

78-3606 **Fundamental Carcinogenic Processes and Their Implications for Low-Dose Risk Assessment (Letter to Editor).** (Eng) Crump, K. (Dept. Mathematics and Statistics, Louisiana Technocological Univ., Ruston, LA, 71272). *Cancer Res* 38(6): 1836-1839; 1978.

Differences in opinion concerning the validity and conservatism of the Mantel-Bryan approach to estimating the low-dose risk for carcinogens are discussed. It is emphasized that the arbitrariness and poor fit to date of the Mantel-Bryan probit model are conceded properties of the procedure and that the model was never claimed to provide a fit to the data. Rather, it was only claimed to provide an upper limit, under stated assumptions, to the true dose response at low dosage levels, with the plausible exception for one-hit models being indicated. (25 refs.)

78-3607 **Suggested Procedures for Reducing the Pathology Workload in a Carcinogen Bioassay Program, Part II: Incorporating Blind Pathology Techniques and Analysis for Animals with Tumors.** (Eng) Fears, T. R. (Field Studies and Statistics Program, NCI, NIH, Bethesda, MD); Douglas, J. F. *J Environ Pathol Toxicol* 1(3): 211-222; 1978.

The pathology workload in a carcinogen bioassay program can be reduced by procedures that incorporate blind pathology and recover the total tumor aspect. A procedure that incorporates the total tumor concept and results in pathology workload savings (PWS) of 17%-25% involves four steps: (1) examine microscopically (ExM) only common tumor sites in all of the control animal; (2) ExM all tissue sites in the treated animals; (3) in control animals, ExM all additional tissues for which a positive result is possible in at least one of the treatment groups; (4) ExM all remaining tissues of any control group for which a significant increase in total group tumors is still possible. In step 4, if any Fisher exact test for total tumors is positive and the increase cannot be attributed to an effect on one tissue or system, all tissues of the remaining controls are examined. Blind pathology can be incorporated by using either one of two procedures. One, which results in a PWS of 11%-33%, involves the following two steps: (1) ExM all tissue sites of all treatment animals; (2) examine in all controls and all treated animals those tissues for which a total of five or more animals in at least one treatment group had a tumor. In step 2, the slides are grouped by animals, and the groups are given to the pathologist in random order without being labeled as to treatment. The other procedure for incorporating blind pathology, which results in a PWS of 22%-58%, involves the following four steps: (1) identify treatment animals with a lump or bump at gross pathology; (2) ExM all tissues of animals in a random sample from the lump or bump section of each treatment group; (3) in the remaining treatment animals, examine all tissues in which a tumor was found in any of the sampled animals; (4) in all controls and all treated animals, examine those tissues for which a total of five or more animals of a treatment group had a tumor. In step 4, the slides are grouped by animals, and the groups are given to the pathologist in random order without being labeled as to treatment. (7 refs.)

78-3608 **Current Concepts on Mechanisms of Chemical Carcinogenesis.** (Eng) Weinstein, I. B. (Inst. Cancer Res., Columbia Univ., Coll. Physicians and Surgeons, Health Sciences Center Building, 701 W. 168th St., New York, NY, 10032). *Bull NY Acad Med* 54(4): 366-383; 1978.

Some of the mechanisms of chemical carcinogenesis are reviewed. There is a wide diversity of neoplastic agents, both natural and manmade, that cause cancer. Not all of these agents, however, are carcinogenic in themselves: they require metabolic activation. The metabolites generally act by modifying the nucleic acid composition, and if the defect is not excised, a mutation will occur that can be passed to progeny cells. The role of tumor promotors and cofactors in carcinogenesis is also reviewed. (39 refs.)

78-3609 **Mechanisms of Endonuclear Repair and Carcinogenesis.** (Ita) Santi, L. (Instituto Oncologia, Genova, Italy); Parodi, S. *Recent Prog Med (Roma)* 63(6): 571-591; 1977.

Studies of the carcinogenic and DNA-damaging effects of chemicals and radiation are reviewed. It is possible to predict the degree of correlation between the amount and type of DNA damage by a given compound and DNA repair and the carcinogenic potency of the compound. (33 refs.)

78-3610 **Occupational Cancer in Switzerland.** (Ger) Lamperth, E. (Tumbelenstrasse 58, 8330 Pfaffikon ZH, Switzerland). *Cancer Inf (Bern)* 12(2): 54-65; 1977.

Various occupational hazards associated with exposure to toxic chemicals in Switzerland are discussed, with emphasis on the problem of carcinogenesis. It is suggested that the risk of development of occupational cancer is significant in Switzerland. The control of occupational carcinogens in this country should be compulsory. (36 refs.)

78-3611 **Carcinogenic Hazards from Prenatal Exposure to Environmental Chemicals.** (Eng) Napalkov, N. P. (N. N. Petrov Res. Inst. Oncology, Leningrad, USSR). *In: Characterization and Treatment of Human Tumours 1977. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours Held in Budapest, 13-15 April, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 63-71; 1977.

A review of recent experimental data on transplacental carcinogenesis revealed the following: (1) a carcinogen (CG) administered during pregnancy may either kill the embryo or induce malformations and tumors in the offspring as a result of immediate action of the CG or its metabolites on fetal tissues; (2) reaction of the embryo to CG's depends on stage of embryonic development; tumor frequency is highest when exposure is during histogenesis; (3) in general, animals of a given species develop tumors of the same site and type following exposure to various CG's at different stages of embryogenesis; however, the teratogenic response of the embryo to CG's is determined by stage of embryogenesis rather than animal species or type of CG; (4) organ specificity of a CG does not invariably apply in transplacental carcinogenesis; (5) transplacentally induced congenital malformations and tumors represent independent consequences of the same cause and are not predetermined by the development of a congenital defect; (6) transplacental carcinogenesis may be due to endogenous synthesis of CG's in the mother; (7) transplancental exposure to CG's may heighten the susceptibility of an organism to the same or similar substance in postnatal life; (8) hormonal factors may influence the effect of transplacentally administered CG's significantly; (9) risk of tumor development depends on passage of a sufficient concentration of CG's or their active metabolites through the placental barrier and embryonal membranes; however, maternal organism is capable detoxifying transplacental CG's. (50 refs.)

78-3612 **Environmental Carcinogenesis: An Integrative Model.** (Eng) Trosko, J. E. (Dept. Human Development, Coll. Human Medicine, Michigan State Univ., E. Lansing, MI, 48824); Chang, C. C. *Q Rev Biol* 53(2): 115-141; 1978.

In this review, an integrative theory is proposed that attempts to organize the wealth of information on the phenomenon of environmental carcinogenesis. In this theory, environmental carcinogenesis is a process by which the genetic control of cell division and differentiation is altered by carcinogens, which include physical, chemical, and viral mutagens as well as chemical and viral gene modulators. Evidence seems to support the hypothesis that both mutations and epigenetic processes are components of carcinogenesis. The mutational basis of cancer is supported by the clonality of tumors; the mutagenicity of 90% of all tested carcinogens; the high mutation frequencies in humans having certain syndromes that predispose the individual to cancer; the correlation of in vitro DNA damage, mutagenesis, and transformation with in vivo carcinogenesis; age-related incidence of various hereditary tumors; and the correlation of photoreactivation of DNA damage and biological amelioration of UV-induced neoplasms. Since mutagens and gene modulators can be carcinogenic, carcinogens may affect the genes that control cell division. The proposed integrative theory is based on the mutation and epigenetic theories, the two-stage theory, and Coming's general theory of carcinogenesis. According to this theory, carcinogens affect regulatory genes that control a series of transforming genes. A general hypothesis is advanced that there is a common mechanism of somatic mutagenesis via error-prone repair of DNA damage linking carcinogenesis, teratogenesis, atherosclerosis, and aging. Concepts that provide a framework for evaluating the scientific, medical, and social implication of cancer are presented. (181 refs.)

78-3613 **Environmental Chemical Carcinogenesis.** (Eng) Stewart, B. W. (Sch. Pathology, Univ. New South Wales, Sydney, New South Wales, Australia); Sarfaty, G. A. *Med J Aust* 1(2): 92-95; 1978.

A carcinogen can be identified by large-population epidemiology, occupational epidemiology, chronic animal testing, short-term screening tests, and chemical analysis. Identification of a compound as a carcinogen in animals does not indicate that it is carcinogenic in humans, but is merely suggestive of the fact. Known human carcinogens should be regulated, but this is a social and economic problem as well as a biological and medical one. Certain personal habits still constitute the major established carcinogenic hazards for the general population. (14 refs.)

78-3614 **Air Pollution Research and Practice in East Germany.** (Ger) Dorre, W. H. (Lehrstuhl Allgemeine und Kommunale Hygiene des Hygiene-Instituts, Humboldt Universitat zu Berlin, Otto-Grotewohl-Strasse 1, DDR-108 Berlin, E. Germany); Grosser, P. J.; Hammje, K.; Knauer, A.; Theodor, H.; Wettig, K. *Z Gesamte Hyg* 24(1): 1-12; 1978.

Progress made during the last 15 yr in air pollution research in East Germany is reviewed. The medical aspects of air pollution are emphasized, and max permissible emission levels are tabulated for 143 industrial chemicals. (79 refs.)

78-3615 **Risks Caused by Low Levels of Pollution.** (Eng) Wilson, R. (Dept. Physics, Harvard Univ., Cambridge, MA, 02138). *Yale J Biol Med* 51(1): 37-51; 1978.

The theoretical and experimental evidence for the concept that pollutants in almost any concentration cause a small risk of death is reviewed. The hypothesis that cancer incidence is linear at low doses with applied dose of pollutant originated with radiation carcinogenesis. According to the linear theory, if the quantity of a carcinogen is constant and randomly distributed throughout the environment, the total number of cancers produced will be constant to within the square root of that number. Although there are various theoretical reasons for expecting to find a deviation from linearity, taking a linear dose-response relationship is recommended by most regulatory bodies. Data on cancers produced in humans by chemical carcinogens reveal few well-measured dose-response relationships, but there is a considerable amount of data on chemical cancers induced in animals. Many carcinogens such as vinyl chloride (VC) are indirect, and they only become carcinogenic as a result of the production of metabolites in the liver. The linearity of effect vs dose actually applies to the dose presented to the cell. The metabolic production of carcinogens in the liver might not be linear with the VC concentration. However, prudent public policy demands the assumption of linearity for exposure to the general public until further information is available. (50 refs.)

78-3616 **Complex Biological and Epidemiological Examinations to Recognize and Eliminate Occupational Health Risks Posed by Air Pollution.** (Ger) Haublein, H. G. (Zentralinstitut fur Arbeitsmedizin der DDR, Noldnerstrasse 40-42, DDR-1134 Berlin, E. Germany); Blau, F
Konetzke, G.; Schneider, W. D.; Thiele, H. *Z Gesamte H*
24(4): 248-252; 1978.

Respiratory tract diseases caused by air pollution accounte
for 11.49% of all occupational diseases in East Germany du
ing 1970-1976. The incidence of silicosis declined markedl
but that of asbestosis, including its pleural forms, remaine
constant. Increases in the incidence of bronchial cance
chronic unspecified lung diseases, and respiratory tract alle
gies are attributed to improved registration procedures. C
the total number of occupational diseases, 3.42% represente
chronic intoxication caused by the inhalation of chemica
such as carbon monoxide, halogenated hydrocarbons, ben
zene and its nitro and amino derivatives, and carbon disu
fide. Metals, metallic compounds, and pesticides are no long
er a leading cause of occupational intoxication. Th
frequency of intoxication due to inhalation of different type
of dust may be underrepresented, considering the large num
ber of exposed workers. The effectiveness of preventive indus
trial medicine programs and of the early registration of occu
pational diseases was reflected in an epidemiologic study o
$>$ 15,000 workers. A study covering 300,000 workers fron
1976 to 1978 is now in progress. (28 refs.)

78-3617 **Carcinogenesis Induced by Trace Contaminant
in Potable Water.** (Eng) Kraybill, H. F. (Di
of Cancer Cause and Prevention, NCI, Bethesda, MD). *Bu*
Ny Acad Med 54(4): 413-427; 1978.

The following are some recognized and suspect carcinogen
in US drinking water and their concentrations (in μg/lite
when given): aldrin (5.4), benzene (50), benzo(a)pyren
(0.0002-0.002), bis(2-chloroethyl)ether (0.42), lindane, ca
bon tetrachloride (2.0-3.0), chlordane (0.1), chlorofor
(0.1-311), 1,2-dibromoethane, dieldrin (8.0), dichlorod
phenyltrichloroethane (DDT), dichlorodiphenyldichlor
ethylene (DDE) (0.05), 1,4-dioxane (1.0), endrin (0.004), he
tachlor, trichloroethylene, and vinyl chloride (10.0). Of the
compounds, benzo(a)pyrene and vinyl chloride are the on
recognized carcinogens. Only volatile organic compoun
have been identified in US drinking water, and a much larg
component of nonvolatile substances remains to be identifi
or quantified. In city water in the Netherlands and German
concentration ranges of inorganic contaminants such as ars
nic, cadmium, chromium, and selenium are 1.0-8.1, 2.0-9.
4.5-10.0, and 3.1-6.0 μg/liter, respectively. Asbestifor
materials or asbestos particles have been found in many riv
systems at concentrations $>$ 10 μg/gallon. In a study of ca
cer mortality in 10 river basins in the US, nickel concentr
tions appeared to correspond with oral and intestinal canc
death rates, arsenic with cancer of the eye and larynx a
myeloid leukemia. Beryllium correlated with bone cancer a
with mortalities from breast and uterine cancer. Lead w
associated with leukemia and lymphoma and with kidne
stomach, intestinal, and ovarian cancer. The results of va
ous epidemiological studies throughout the US are reporte

The need for in vitro bioassays to augment in vivo procedures in determining carcinogenicity is stressed. (30 refs.)

78-3618 **Personal Habits.** (Eng) Wynder, E. L. (American Health Foundation, 1370 Ave. Americas, New York, NY, 10019). *Bull NY Acad Med* 54(4): 397-412; 1978.

A review of the association between various personal habits and cancer is presented. Among the relationships discussed are the following: tobacco smoking and lung cancer; alcohol intake and cancer of the mouth, esophagus, and larynx; iodine deficiency and cancer of the thyroid; atrophic gastritis and stomach cancer; and fat intake and colon and breast cancer. (8 refs.)

78-3619 **Tumor Promoters: Carcinogenesis Gets More Complicated.** (Eng) Marx, J. L. (No affiliation given.). *Science* 201(4355): 515-518; 1978.

Promotion is important in the development of several cancers in a variety of species, and many carcinogens may have the ability to both initiate and promote tumor formation. The initiation step is irreversible, but if exposure to promoter is stopped before the cells gain the ability to multiply in the absence of the promoter, tumor formation may be avoided. Among the chemicals with promoting activity are the phorbol esters; phenobarbital, which appears to promote liver tumors; saccharin and sodium cyclamate, which appear to promote bladder tumors; bile acids, which promote the development of colon cancer; and cigarette smoke. Most investigators think that initiation involves an alteration in the cell's DNA by the initiating agent. Tumor promoters appear to interfere with the normal development of cells. All promoters tested increased cellular ornithine decarboxylase activity, which may be a prerequisite for promotion. Proteases are also implicated in this respect. It is possible that promoters bind to a receptor for a naturally occurring material, namely, epidermal growth factor (EGF), which stimulates the division of epidermal cells. There may be several stages in the progression of initiated cells to benign and then to malignant tumors. Since the effects of promotion are reversible, it may be possible to use inhibitors to prevent cancers from developing. (no refs.)

78-3620 **Diethylstilbestrol: A Review of Its Toxicity and Use as a Growth Promotant in Food-producing Animals.** (Eng) McMartin, K. E. (Toxicology Center, Dept. Pharmacology, Univ. Iowa, Iowa City, IA, 52242); Kennedy, K. A.; Greenspan, P.; Alam, S. N.; Greiner, P.; Yam, J. *J Environ Pathol Toxicol* 1(3): 279-313; 1978.

The human and animal uses and effects of diethylstilbestrol (DES) are reviewed. In humans, DES has been used to prevent abortion, prevent implantation, replace estrogens, and treat prostatic cancer. However, it has induced neoplastic and nonneoplastic reproductive tract disorders in the female offspring of mothers treated during pregnancy and, possibly, endometrial carcinoma in women taking the compound. DES is also used a a growth promotor in livestock. It is estimated that DES residues in meat are at most 20% of the harmful effect dose. If the proper regulations are followed, there should be no danger to humans from this source. (162 refs.)

78-3621 **Food Policy and Health.** (Eng) Whitehead, R. G. (MRC Dunn Nutrition Unit, Cambridge, England). *J Royal Soc Arts* 76(5265): 552-563; 1978.

The role of proper nutrition in the maintenance of good health is discussed. One study indicated that an increase in carbohydrate intake to 55%-60% of the total energy intake will help prevent bowel cancer, that reducing fat intake from 40% to 30% of the total energy intake will help prevent breast and colon cancer, and that reducing salt consumption to 3 g/day will help prevent stomach cancer. (9 refs.)

78-3622 **The Chemistry and Toxicology of Hair Dyes.** (Eng) Burnett, C. M. (Clairol Res. Lab., Stamford, CT); Corbett, J. F. *In: Cutaneous Toxicity.* Drill, V. A.; Lazar, P., eds. (New York: Academic Press, Inc.): 277 pp.; 203-221; 1977.

Data on the toxicity and carcinogenicity of hair dye preparations are reviewed. Most of the current data are based on animal tests, which show no carcinogenic effects, and retrospective surveys, which show no evidence of increased cancer incidence that can be correlated with hair dye usage. Several in vitro screening tests have indicated that components of these dyes are mutagenic, but a correlation between this mutagenicity and human toxicity and carcinogenicity has yet to be proved. (47 refs.)

78-3623 **Beryllium: Carcinogenicity Studies (Letter to Editor).** (Eng) Wagoner, J. K. (Dept. Labor, Office Assistant Secretary, Occupational Safety and Health Adim., Washington, DC, 20210); Infante, P. F.; Mancuso, T. *Science* 201(4353): 298-303; 1978.

A previous article in which concern was expressed about fair play in the conduct of epidemiological studies and government regulations of beryllium (Be) is criticized. The authors, as government and independent researchers, accepted responsibility for the objective conduct and evaluation of these studies and they note that there is continued reluctance of industry and its consultants to accept the carcinogenicity of Be. According to several epidemiological studies, there is an increased risk of lung cancer mortality (LCM) among occupationally exposed subjects. One study by the National Institute for Occupational Safety and Health (NIOSH) found an excess of LCM in a subcohort of individuals entered in the US Beryllium Case Registry with a diagnosis of prior Be-related pneumonitis or bronchitis. The individuals had short-term exposure to Be. These studies are consistent with numerous animal bioassay studies demonstrating that Be is carcinogenic by several routes and in many species and, specifically, with animal bioassay results demonstrating lung cancer induction in 51% of exposed animals by a single dose of Be oxide. A Be exposure limit of 2 $\mu g/m^3$ set 30 yr ago by the government is still not being adhered to by industry, and in several operations levels have exceeded 1,000 $\mu g/m^3$. Industry consultants claim that the NIOSH study failed to exhibit a dose-response relation, as measured by duration of employment of LCM. However, individuals exposed to high concentrations over a short period could have a total lung Be burden as great or greater than that of individuals exposed over a long period to lower concentrations. this is supported by the fact that the body does not clear Be readily. In addition, the NIOSH study provides no data to refute a dose-response relation. (26 refs.)

78-3624 **Toxicological Effects of Cadmium.** (Eng) Lauwerys, R. (No affiliation given). *In: Criteria (Dose/Effect Relationships) for Cadmium. Report of a Working Group of Experts prepared for the Commission of the European Communites, Directorate-General for Social Affairs, Health and Safety Directorate.* (New York: Pergamon Press): 202 pp.: 67-116; 1978.

The potential hazards to humans resulting from short- and long-term exposure to cadmium are reviewed. Short-term ingestion primarily affects the gastrointestinal tract, and short-term inhalation primarily affects the lungs. Toxic effects on the cardiovascular system, testis, liver, kidney, and nervous system have also been demonstrated in animals. The two main target organs after long-term exposure to moderate Cd concentrations are the lungs and kidneys, but the bones and hematopoietic system can also be affected. In animals, effects on the cardiovascular system, liver, growth, and survival have been demonstrated. Cd induces cancer in animals, primarily at the site of injection. Some surveys among occupationally exposed subjects suggest that Cd may also be carcinogenic in humans, but the evidence is inconclusive. There is no indication that nonoccupational exposure to Cd constitutes a carcinogenic hazard. There are conflicting reports in the literature concerning the mutagenic action of Cd compounds. The effects of Cd on the reproductive processes (fertility, embryotoxicity, and perinatology) of animals are discussed. (no refs.)

78-3625 **An Assessment of Experimental Carcinogen-Detecting Systems with Special Reference to Inorganic Arsenicals.** (Eng) Wildenberg, J. (Dept. Epidemiology and Statistics, American Cancer Society, 777 Third Ave., New York, NY, 10017). *Environ Res* 16(1/3): 139-152; 1978.

Human, animal, and short-term carcinogenicity data for arsenical compounds are reviewed and assessed. Epidemiologic and clinical observations indicate a causal relation between inorganic arsenic and skin cancer, and they suggest that arsenicals may also be respiratory carcinogens. However, a carcinogenic role for inorganic arsenic has not been demonstrated in animal experiments, nor have positive findings with arsenicals been reported in short-term tests. Some limitations in the short- and long-term bioassay systems that are used to identify potential human carcinogens are discussed. It is recommended that mutation induction in *Salmonella* be adopted as a prescreen for certain classes of chemicals. Additional prescreen assays, independent of mutagenicity, should be developed and validated to supplement the bacterial results. (50 refs.)

78-3626 **Cancer in the Workplace--Percivall Pott to the Present.** (Eng) Cowles, S. R. (Occupational Medicine Branch, Naval Regional Medical Center, Bremerton, WA, 98314). *Milit Med* 143(6): 395-400; 1978.

Known and suspected carcinogens commonly found at Naval installations, particularly shipyards, are reviewed, and methods of controlling human exposure to these substances are outlined. The carcinogens include physical agents (cigarettes, asbestos), metals (Ni, As), and hydrocarbons (ethylenethiourea, 4,4′-methylenebis(2-chloroaniline), vinyl chloride, and polychlorinated biphenyls). (15 refs.)

78-3627 **The Significance of Vinyl Chloride as a Pollutant from the Viewpoint of Industrial Medicine, Experimental Oncology, and Industrial Toxicology.** (Ger) Konetzke, G. (Zentralinstitut fur Arbeitsmedizin der DDR, Noldnerstrasse 40-42, DDR-1134 Berlin, E. Germany); Bittersohl, G.; Grund, W.; Schramm, T.; Teichmann, B.; Zschunke, E. *Z Gesamte Hyg* 24(7): 501-508; 1978.

The carcinogenic effect of vinyl chloride (VC) and its polymers, recommendations for the protection of workers exposed to VC, and the industrial exposure of workers in East Germany to VC are reviewed. In rats, mice, hamsters, and rabbits, inhalation of 50-10,000 ppm VC monomer (VCM) induced tumors. Even low doses (50 ppm for 4 hr/day, 5x/wk, for 1 yr) produced hepatic angiosarcomas and nephroblastomas. Hemangioepitheliomas of the liver have been associated with exposure of humans to VCM. Brain, lung, blood, and lymph malignancies are also associated with VCM. However, no such cases have yet been found in East Germany. A list of clinical and x-ray studies for monitoring workers exposed to VC is presented. Several countries have decreased the standard levels of VC allowable in workplaces because of accumulating evidence that workers were being exposed to harmful levels. (41 refs.)

78-3628 **N-Nitrosomorpholine.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 263-280; 1978.

An evaluation of the carcinogenic effect of N-nitrosomorpholine (NMOR) is presented, NMOR was found in 3/11 batches of analytical grade dichloromethane at levels of 10-32 μg/liter and in 4/10 batches of analytical grade chloroform at levels of 2-376 μg/liter. Carcinogenicity studies have been conducted in mice, rats, hamsters, and zebra fish. The carcinogenicity of precursors, derivatives, and factors that modify carcinogenicity in animals have also been investigated. After po administration, NMOR produced benign and malignant tumors of the liver and lung in mice; the liver, kidney, and blood vessels in rats; and the liver in hamsters. After sc administration, of single and multiple doses, hamsters developed tumors of the digestive and respiratory tracts. After iv injection, rats developed liver tumors. When NMOR was added to their tank water, zebra fish developed liver tumors. A dose-response relationship was determined. NMOR was mutagenic in several bacterial strains, Chinese hamster V79 cells, in a host-mediated assay with mice, and in Drosophila. No human data are available. However, NMOR should be regarded for practical purposes as if it were carcinogenic to humans. (57 refs.)

78-3629 **N-Nitrososarcosine.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 327-335; 1978.

Studies on the carcinogenicity of N-nitrososarcosine (NSAR) are evaluated relative to a possible carcinogenic risk to humans. NSAR has been found in some meat samples. It produced squamous cell carcinomas of the nasal cavities in mice and esophageal carcinomas in rats after its po administration. After ip injection, it produced liver cell carcinomas in newborn mice. Rats exposed to NSAR ethyl ester developed esophageal carcinomas. NSAR (1 g/kg body wt) administered to mice by gavage was not mutagenic in the host-mediated assay. The general population may be exposed to low levels of NSAR, but no epidemiological data or case reports were available. There is sufficient evidence of a carcinogenic effect of NSAR in two experimental animal species, and therefore it should be regarded as if it were carcinogenic to humans. (24 refs.)

78-3630 **N′-Nitrosonornicotine.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 281-286; 1978.

Biological data relevant to the evaluation of a carcinogenic risk of N′-nitrosonornicotine (NNN) to humans are presented. NNN has been found in cigarette smoke and in various tobaccos. It has been shown to be carcinogenic in rats, mice, and Syrian golden hamsters. After po administration to 20 rats (total dose, 630 mg/animal), 12 surviving rats developed esophageal tumors (11 papillomas, 3 carcinomas) plus 1 pharyngeal papilloma and 3 carcinomas of the nasal cavity with brain invasion. Of 20 Syrian hamsters given sc injections of NNN (Total dose, 375 mg/animal) 12 developed single papillary tumors of the trachea within 83 wk, and 1 had an adenocarcinoma of the nasal cavity. Of 40 mice injected ip with 0.1 ml NNN once a wk for 41 wk, 25 died within 7 mo with no evidence of tumors, but 7/8 animals that died after 8 mo had multiple pulmonary adenomas. No human data are available; however, tobacco smokers are exposed to NNN along with other N-nitroso compounds. For practical purposes, NNN should be regarded as if it were carcinogenic to humans. (13 refs.)

78-3631 **N-Nitrosopyrrolidine.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 313-326; 1978.

Data on the carcinogenicity of N-nitrosopyrrolidine (NPYR), a compound formed during the heating of N-nitrosoproline, are presented. NPYR has been found in the vapor produced during the frying of bacon, tobacco smoke, waste water from chemical factories, grilled and fried bacon, dry premixed cures containing spices and sodium nitrite, animal feed, and cured lunchmeats, ham, and sausage. Of 40 mice given NPYR po (total dose, 0.015 g/mouse), 4/34 surviving animals had 51 lung adenomas. Of 25 BD rats given 5 or 10 mg/kg/day in the drinking water for 150 days and then double these doses, 23/25 animals developed hepatocellular carcinomas after an av induction time of 470 and 290 days, respectively (av total doses causing tumors in 50% of rats, 3.9 or 4.2 g/kg). In other rat species, hepatocellular carcinomas, papillary mesotheliomas of the tunica vaginalis, interstitial cell tumors, a cavernous hemangioma of the testes, cholangiocarcinomas, and olfactory carcinomas were observed. In Syrian golden hamsters, sc administration of NYPR induced lung tumors. A dose-response relationship was established in rats. NPYR produced reverse mutations in *Escherichia coli* and *Salmonella typhimurium* and 8-azaguanine-resistant mutants in Chinese hamster V79 cells. Although human data are available, there is sufficient evidence of a carcinogenic effect in rats for NPYR to be regarded as if it were carcinogenic to humans. (51 refs.)

78-3632 **N-Nitrosomethylethylamine.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 221-226; 1978.

The carcinogenicity of N-nitrosomethylethylamine (NMEA) was evaluated on the basis of current biological data. NMEA occurs in tobacco smoke condensate and in various meats and meat products. Of a group of 15 female BD rats given NMEA in the drinking water at doses at 1 or 2 mg/kg/day, 9 developed hepatocellular carcinomas and 1 a fibrosarcoma of the vagina. The av induction time was 500 to 360 days for the two dosage groups, respectively, and the av total doses to induce tumors in 50% of the animals was 0.42 and 0.75 g/kg. Human data are not available; however, reports suggest that tobacco smokers may be exposed to NMEA along with other N-nitroso compounds. The experimental evidence is sufficient for NMEA to be regarded as if it were carcinogenic to humans. (15 refs.)

78-3633 **N-Nitrosopiperidine.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 287-301; 1978.

An evaluation of the carcinogenic risk of N-nitrosopiperidine (NPIP) to humans is presented. NPIP has been found in tobacco smoke, wastewater from chemical factories, cheeses, smoked codfish, various processed and preserved meats, dry, premixed cures containing spices and sodium nitrite and animal feed. In mice, NPIP causes benign and malignant tumors of the liver, lung, forestomach, and esophagus. In rats, NPIP causes tumors of the liver, esophagus, and respiratory system. In addition, it produces tumors of the upper digestive tract, respiratory system, and liver in hamsters and hepatocellular carcinomas in monkeys. Single doses of NPIP are carcinogenic in mice and hamsters. No case reports or human epidemiological data are available. Since the general population may be exposed sporadically to low levels of NPIP and since the animal evidence is conclusive, NPIP should be regarded for practical purposes as if it were carcinogenic to humans. (59 refs.)

78-3634 **N-Nitrosoproline and N-Nitrosohydroxyproline.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland) IARC Monographs on the Evaluation of the Carcinogeni Risk of Chemicals to Humans. Vol. 17, 365 pp.; 303-311 1978.

An evaluation of the carcinogenic effect of N-nitrosoprolin (NPRO) and N-nitrosohydroxyproline (NHPRO) is presented. Chemical and physical data and the production, use, occurrence, and analyses of these compounds are reviewed NPRO has been found in uncooked cured meat products NHPRO was found in one meatloaf sample. When mice wer given 325 or 650 mg/mouse in the drinking water, the incidence of lung adenomas was not greater than that of controls When rats were given NPRO or NHPRO in drinking wate at a concentration of 0.015% for 75 wk, no significant differences in tumor incidence were observed between untreate and treated animals. No carcinogenic effects were observe in rats given NPRO or NHPRO intragastrically once a w for 4 wk (total dose: 19 or 38 g/kg body wt, respectively) No human data are available. Although the general population may be exposed to low levels of NPRO and NHPRO the data are insufficient for an evaluation of the carcinogenicity of NPRO or NHPRO. (51 refs.)

78-3635 **N-Nitrosomethylvinylamine.** (Eng) IAR Working Group (Lyon, France). *In: Som N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IAR Monographs on the Evaluation of the Carcinogenic Risk o Chemicals to Humans. Vol. 17, 365 pp.; 257-261; 1978.

The carcinogenic risk of N-nitrosomethylvinylamin (NMVA) was evaluated. NMVA is carcinogenic in rats, th only species tested. Of a group of 19 rats given 0.3 or 0. mg/kg/day in the drinking water, papillomas developed i 2 and nodular and widely distributed squamous cell carcinomas of the esophagus in 17. In addition, 4 had carcinomas of the tongue, 5 had carcinomas of the pharynx, an 8 had bile duct proliferation and hepatic cysts. Inhalatio and/or intratracheal administration of NMVA (single dos of 18 mg/kg, or 25 or 50 ppm in air 2x/wk for 30 min produced carcinomas of the nasal cavities. Although ther are no human data on the carcinogenic effect of NMVA, th experimental evidence indicates that for practical purpose it should be regarded as if it were carcinogenic to humans (11 refs.)

78-3636 **Toxicity of Inhalation Anaesthetic Agents** (Eng) Cohen, E. N. (Dept. Anesthesiology Stanford Univ. Medical Center, Stanford, CA, 94305). *Br Anaesth* 50(7): 665-675; 1978.

The relationship between anesthetic metabolism and toxicity the mechanisms of anesthetic toxicity, and specific organ toxicity of anesthetic agents are reviewed. Evidence for the direc induction of human cancer by anesthetic drugs is largely epidemiologic. Studies have indicated that there is an increased incidence of cancer (threefold increase in leukemia and lymphoma) in women exposed to waste gases; no similar increase has been observed for men. There is some evidence that nitrous oxide is metabolized. (82 refs.)

78-3637 **Animal Models: Diet and Large Bowel Cancer.** (Fre) Martin, M. S. (Laboratoire d' Immunologie, Faculte de Medecine, INSERM, U-45, boulevard Jeanne-d'Arc, F21033 Dijon, France). *Bull Cancer (Paris)* 65(1): 49-52; 1978.

Literature reports on the effect of various dietary factors on chemically induced cancer of the intestine in the rat are reviewed. The cancers were induced by direct intrarectal instillation of N-methyl-N'-nitro-N-nitrosoguanidine methylnitrosourea, or methylazoxymethanol or by feeding the rats 1,2-dimethylhydrazine or azoxymethane. The factors studied included fat level of diet, quality and quantity of fat, fecal excretion of neutral and acid steroids, intestinal microflora, and fiber content of the diet. Both animal and vegetable fats potentiated the effects of the carcinogens. Bacterial flora appeared to play a fundmental role in the metabolism of certain carcinogens. The bacterial enzyme converted noncarcinogens into carcinogens. A diet high in beef increases the number of intestinal bacterial flora and, thus, would indirectly contribute to increased levels of carcinogens. There did not appear to be any correlation between dietary fiber and carcinogenesis in the rat animal model. (13 refs.)

78-3638 **Phenacetin Abuse: A Review.** (Eng) Carro-Ciampi, G. (Laboratorio di Farmacologia, Istituto Superiore di Sanita, 00161 Rome, Italy). *Toxicology* 10(4): 311-339; 1978.

Excessive and prolonged consumption of phenacetin-containing analgesics is associated with one or more of the following: hemolytic anemia with methemoglobinemia and sulfhemoglobinemia, impaired renal function with renal papillary necrosis and pyelonephritis, and uroepithelial cancer of the renal pelvis and lower urinary tract. A working hypothesis might be that the three groups of conditions are successive stages of a progressive disease. (168 refs.)

78-3639 **Carcinogenic Activity of Antitumor Chemotherapeutic Agents.** (Ita) Cardinali, G. (Oncologia Clinica, Universita degli Studi, Centro Oncologico, Ancona, Italy); Scoponi, C.; Trivisonne, R. *Recent Prog Med (Roma)* 63(6): 649-668; 1977.

Studies of the carcinogenicity of various anticancer agents are reviewed. The anticancer drugs mechlorethamine, cyclophosphamide, isophosphamide, chlorambucil, melphalan, chloronaphazine, busulfan, urethane, nitrosourea, triethylenemelamine, triethylenetriphosphoramide, uracil mustard, dibromomannitol, dibromodulcitol, actinomycin D, adriamycin, daunomycin, mitomycin C, streptozotocin, 6-mercaptopurine, methotrexate, azathioprine, procarbazine, and dimethyltriazenoimidazole carboxamide have carcinogenic activity. (123 refs.)

78-3640 **Streptozotocin.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 337-349; 1978.

Carcinogenicity data for streptozotocin (STR), an antitumor drug, were evaluated to determine its risk to humans. STR is produced by the soil microorganism *Streptomyces achromogenes.* It is carcinogenic in mice, rats, Syrian golden hamsters, and Chinese hamsters following iv or ip injection. STR produces benign and malignant tumors of the liver and kidney and islet cell tumors of the pancreas. It is carcinogenic after administration in single doses. STR was shown to be mutagenic in *Salmonella typhimurium* and *Drosophila melanogaster* and in host-mediated assays in rats and mice. It has been used to treat pancreatic islet-cell tumors and malignant carcinoid tumors in humans. Side effects included nausea and vomiting, renal and hepatic toxicity, bone marrow damage, anemia, leukopenia, and thrombocytopenia. Although no epidemiological data were available, there is sufficient evidence of a carcinogenic effect of STR in several animal species, and it should be regarded as if it were carcinogenic to humans. (38 refs.)

78-3641 **The Mycotoxins and Human Health Hazards.** (Eng) Linsell, C. A. (International Agency Res. Cancer, 150, Cours Albert-Thomas, 69008 Lyon, France). *Ann Nutr Aliment* 31(4/5/6): 997-1004; 1977.

The role of mycotoxins in human disease is reviewed briefly. Among the various mycotoxins, aflatoxins have been shown to be the most potent hepatocarcinogens. This mycotoxin is present in human foodstuffs in areas of the world where liver cancer is commonly reported. It is suggested that the toxicity of mycotoxins may only be the tip of the iceberg and that more pathologies may develop with time. (31 refs.)

78-3642 **The Public Health Significance of Mycotoxins.** (Eng) Rutledge, A. E. (Microbiological Labs., Div. Agrimerica, Inc., 1829 Stanley St., Northbrook, IL, 60062). *Ann Nutr Aliment* 31(4/5/6): 1019-1030; 1978.

A review of literature data on the toxicity of the major mycotoxin-producing fungi is presented. Special attention is focused on contamination by aflatoxins, stachybotrys toxins, penicillium toxins, rubratoxins, ochratoxins, and various pulmonary toxins. Studies of aflatoxins indicate that the reticuloendothelial system is impaired seriously during aflatoxicosis. (30 refs.)

78-3643 **Carcinogens in Food.** (Fre) Griciute, L. (Centre International de Recherche sur le Cancer, 150, cour Albert-Thomas, F69372 Lyon Cedex 2, France). *Bull Cancer (Paris)* 65(1): 53-58; 1978.

A review of research linking human cancer to the intake of chemicals shows that a definite relationship has been established only for aflatoxin and primary liver cancer. However,a number of chemicals that are carcinogenic to animals, such as polycyclic aromatic hydrocarbons and N-nitroso compounds, are present in human food. Research has shown that the av intake of benzo(a)pyrene (BP) in East Germans is 24-85 mg over a life-span of 70 yr and that most of this BP is from bread, margarine, vegetable oil, and beans. Nitrosamines are converted in vivo from nitrates in food and from the nitrites used for curing meat. An attempt has been made to correlate the intake of N-nitroso compounds, aromatic hydrocarbons, and aflatoxin with the incidence of esophageal cancer in Iran. BP contamination of apple cider has been studied in Brittany, another area with a high incidence of esophageal cancer. (27 refs.)

78-3644 **A Review of the Literature of Rotenone 1,2,12,12a-Tetrahydro-8,9-dimethoxy-2(1-methylethenyl)-1-benzopyrano- [3,5-b]furo[2,3-h][1]benzopyran-6(6h)-one.** (Eng) Haley, T. J. (Dept. Health, Education and Welfare, Food and Drug Administration, Natl. Center Toxicological Res., Jefferson, AR, 72079). *J Environ Pathol Toxicol* 1(3): 315-337; 1978.

The chemistry, biotransformation, pharmacology, toxicology, and carcinogenicity of rotenone (1,2,12,12a-tetrahydro-8,9-dimethoxy-2-(1-methylethenyl)--1-benzopyrano- [3,5-b]furo[2,3-h][1]benzopyran-6(6h)-one are reviewed. Further studies with low doses of rotenone are necessary to determine the exact carcinogenic risk, and epidemiological studies with exposed workers are necessary to determine the risk to humans. (194 refs.)

78-3645 **Mechanisms of Ethionine Carcinogenesis.** (Eng) Sharma, O. K. (Dept. Microbiology, Univ. Colorado Medical Center, Denver, CO, 80262); Kuchino, Y.; Borek, E. *Adv Enzyme Regul* 16: 391-405; 1978.

Recent laboratory findings on the mechanisms of ethionine (Eth) carcinogenesis are reviewed. Acute exposure of rats to Eth induces a rapid decrease in hepatic ATP concentration followed by an inhibition of RNA and protein synthesis. There is a specific inhibition of ribosomal RNA synthesis. Cellular ATP is depleted because of an accumulation of S-adenosylethionine. These effects can be completely reversed by replenishing the cells with ATP precursors. In studies of chicks treated with Eth and adenine (Ade), induction of ovalbumin and conalbumin synthesis was not observed in the primitive oviduct of 5- to 6-wk-old chicks that had not received primary stimulation with estrogen. This suggests that Eth alone cannot cause cytodifferentiation and growth of the primitive oviduct, but that in tissues differentiated by the action of estradiol, it can induce specific protein synthesis. The elevation of progesterone levels upon administration of Eth and Ade may produce other hormonal imbalances and thus have implications in the mechanisms of carcinogenesis. Studies using transfer RNA (tRNA) isolated from the livers of rats injected with (ethyl-^{3}H)-Eth revealed that about 50% of the label was eluted with the lysine tRNA as the last peak. Rat liver tRNA-Lys has at least two isoreceptors; Eth preferentially alkylated tRNA$_2$-Lys. Whether the alkylation of tRNA by carcinogens plays a role in the onset of neoplasia is unknown. (76 refs.)

78-3646 **Pathological and Toxicological Data on Chlorinated Pesticides and Phenobarbital.** (Eng) Butler, W. H. (Dept. Histopathology, Saint George's Hosp. Medical Sch., Blackshaw Road, London SW17 0QT, England); Jones, G. *Ecotoxicology and Environmental Safety* 1(4): 503-509; 1978.

Data on the carcinogenicity of the organochlorine pesticides DDT (1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane), dieldrin, mirex, heptachlor, chlordane, and Aramite [sulfurous acid 2-(p-tert-butylphenoxy)-1-methylethyl 2-chloroethyl ester] in mice and rats are reported, along with current studies of phenobarbital. Aramite produced 2 hepatocellular carcinomas in a group of 21 FDRL Wistar rats receiving 1,580 ppm. Lower doses resulted in a low incidence of liver carcinomas and hyperplastic nodules in these rats, hyperplasia only in CFN rats, and neither hyperplasia nor neoplasia in Sprague-Dawley rats. However, these lower doses induced adenocarcinomas of the gallbladder and extrahepatic biliary ducts in dogs. Most of the chlorinated pesticides have been studied in mice, and on the basis of available data, it cannot be concluded that these compounds are carcinogenic for the mouse liver. When male C3H mice were fed 1,000 ppm PB, a 70%-80% incidence of hepatic nodules was seen at about 80 wk, but it was uncertain whether these lesions were carcinoma. Although the histological assessment is incomplete, hepatocellular carcinoma was not seen during a 2-yr study of feeding 1,000 ppm PB to male Fischer rats. Except possibly for Aramite, there are little reliable data as to the carcinogenicity of organochlorine pesticides or PB in other species. If this assessment of the available toxicological and pathological data is correct, it is not likely that these compounds represent a high carcinogenic risk for humans. (19 refs.)

78-3647 **On the Mechanism and Genetic Control of Mutagenesis Induced by Carcinogenic Mutagens.** (Eng) Radman, M. (Deparatement de Biologie Moleculaire, Universite Libre de Bruxelles, B-1640 Rhode St. Genese, Belgium); Villani, G.; Boiteux, S.; Defais, M.; Caillet-Fauquet, P.; Spadari, S. *In: Mechanisms of Carcinogenesis. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4 (Book B), 699 pp.; 903-922; 1977.

Experiments on the mechanisms of mutagenesis induced by carcinogensare reviewed. Studies using the radioactive phage πX174 and its single- and double-stranded DNA's indicated that the first pyrimidine dimer encountered by DNA polymerase blocked DNA replication in intact host cells. UV irradiation of the host cell induced an increased capacity to copy damaged DNA. This could be a result of the induction of new DNA polymerases, inhibition of the proofreading activity of constitutive DNA polymerases, or modification of the DNA. No evidence has been found for the induction of any new DNA polymerase. Beryllium, which has been shown to inhibit the proofreading $3' \to 5'$ exonuclease activity of the *Micrococcus luteus* DNA polymerase and to decrease the fidelity of avian myeloblastosis virus reverse transcriptase, caused infidelity of in vitro DNA synthesis by HeLa α, β, and γ DNA polymerases. UV irradiation of the template synergistically increased infidelity in the presence of Be^{++}. A synthetic protease inhibitor, tosylleucylchloromethyl ketone, inhibited UV-induced mutagenesis and prophage induction in *Escherichia coli* at concentrations that were not toxic for intact *E. coli* and that did not prevent induction at 42 C of a temperature-inducible prophage whose repressor is inactive at 42 C. It appears that the decisive inducing event is proteolytic cleavage of genetic repressors. It is proposed that three major factors are necessary for extensive mutagenesis: unrepaired DNA lesions, error-prone DNA synthesis (SOS induction), and cellular proliferation. (48 refs.)

78-3648 **Active Carcinogenesis and DNA Repair.** (Ita) Sbano, E. (Siena, Italy). *G Ital Dermatol* 113(1-2): 63-78; 1978.

Recent findings suggest correlations between defects in the repair of actinic DNA damage and actinic carcinogenesis. The mechanism of cancer induction by altered DNA repair is not known. (86 refs.)

78-3649 **DNA Repair: New Clues to Carcinogenesis.** (Eng) Marx, J. L. (No affiliation given). *Science* 200(4341): 518-521; 1978.

The strong correlation between the ability of an agent to produce mutations in DNA and its ability to cause cancer suggests that damage to DNA plays a causal role in carcinogenesis. Furthermore, persons with genetic diseases such as ataxia telangiectasia, which is characterized by defects in DNA repair, have an increased risk of dying from cancer. The repair of DNA lesions, especially those induced by UV radiation, is outlined. The induction of lesions in DNA by benzo(a)pyrene is also mentioned. (no refs.)

78-3650 **The Role of Enzymatic DNA Repair in Carcinogenesis and Cancer Chemotherapy.** (Ger) Harbers, E. (Abteilung fur Medizinische Molekularbiologie, Klinisch-Theoretischen Zentrum der Medizinischen Hochschule Lubeck, Ratzeburger Allee 160, D-2400 Lubeck 1, W. Germany). *Z Krebsforsch* 92(1): 1-9; 1978.

The mechanism of DNA repair and the effects of carcinogens and cytostatic drugs on this repair are reviewed. Excision repair of DNA is considered relatively error-free and postreplication repair is considered error-prone. A positive correlation between the binding of benzo(a)pyrene (BP) to DNA and a carcinogenic effect has been shown in animal studies and in human epithelial cell cultures. In the latter, the number of BP molecules bound per unit length of DNA varied 75-fold among cells from different patients. This observation might be used to determine persons at high risk for lung cancer. Cytostatic drugs are relatively weak carcinogens. Inhibitors of DNA repair (caffeine, 2-chlorethyl isocyanate) can increase the effect of alkylating cytostatics. However, tumor production by 4-nitroquinoline 1-oxide is decreased by caffeine, an apparently paradoxical effect. The different biological effects of carcinogens vs cytostatics may result from the ability of the former to cause DNA changes that are hard to repair. In contrast, cytostatics at comparable doses kill many cells and leave others with damage that can be repaired. If these differences could be increased, the late damage from cytostatics would be decreased. The result would be an increased use of these drugs in nonmalignant disease (eg, for immunosuppression). (38 refs.)

78-3651 **Experimental Tumor Metastasis: Characteristics and Organ Specificity.** (Eng) Nicolson, G. L. (Dept. Developmental and Cell Biology, Univ. California, Irvine, CA, 92717). *Bioscience* 28(7): 441-447; 1978.

The characteristics and organ specificity of experimental tumor metastasis are reviewed. Blood-borne metastasis appears to depend on the unique properties of the malignant cell as well as host properties. A successful experimental model of blood-borne metastasis was developed for the murine B16 melanoma. Malignant variant lines (B16-F1 to B16-F10) were selected from the original tumor population for their abilities to implant, invade, survive, and grow to form gross organ tumors after iv injection of B16 cells into C57/Bl mice.

In vivo selection of the B16-F system yielded unique tumor cell lines that were lung-specific trapping in the first capillary bed encountered. In addition, variant lines capable of colonizing different specific secondary sites were obtained using line B16-F1. B16-F1 was retargeted by sequentially selecting for tumor variants that colonize brain, ovary, liver, and other sites from primary sc tumor implants or experimentally administered blood-borne tumor cells. The heterogeneity of the tumor cell properties in the unselected population is probably responsible for the successful in vivo selection of organ-preferring lines. Cell-surface proteins and glycolipids and the adhesive, invasive, enzymatic, and immunological properties of metastatic tumor cells are surveyed using results obtained with the B16-F variants. Protection against experimental metastasis by immunization has been achieved in several animal tumor systems. However, in the B16-F variant system, the host immune response may actually aid in malignant cell arrest and survival. Other aspects of host immunity are reviewed. (54 refs.)

78-3652 **Primary Immunodeficiencies and Malignant Proliferation.** (Fre) Brouet, J. C. (Laboratoire d'Immunochimie et d'Immunopathologie, U. 108 INSERM, Hopital Saint-Louis, 2, place du Dr-Fournier, F 75475 Paris Cedex 10, France); Seligmann, M. *Bull Cancer (Paris)* 65(1): 89-94; 1978.

Reports in the literature reveal that the incidence of neoplasms in persons with primary immune deficiencies (PID) is 100-1,000 times greater than that in the normal population. The incidence of cancer is approx 10%-20% in patients with Wiskott-Aldrich syndrome, 10% in patients with ataxia telangiectasia, and 5%-10% in patients with variable deficiencies; the incidence is $<5\%$ for the other disease categories of PID. A genetic factor is probably involved, since an international cancer registry contains 14 PID families in which 2 or more children developed tumors; in 12/14 families, the tumors were identical. Malignant lymphomas account for two-thirds of all the tumors in patients with PID. Since neoantigens have been demonstrated on only a small number of human tumors, the immunological surveillance theory, which states that abnormal cells are eliminated by the surveillance of immune cells, does not appear feasible. Also, in animals, immunodepression induced by antilymphocyte serum does not increase the incidence of spontaneous tumors. The more likely explanation appears to be antigenic stimulation of a faulty immune system that lacks regulatory controls. (24 refs.)

78-3653 **Transfer of Specific Immunity with RNA.** (Eng) Sell, S. (Dept. Pathology, Univ. California, San Diego, CA); Mendelsohn, J. *Arch Pathol Lab Med* 102(5): 217-222; 1978.

A review of the current status of studies on immune-RNA (iRNA) is presented. iRNA refers to phenol extracts of lymphocytes from immunized animals that have the capacity to transfer a variety of immune products or activities to recipient animals. iRNA from lymphoid cells of immunized or tumor-bearing animals has been found to consistently convert lymphoid cells from nonimmunized humans or animals to killer cells. Positive results have been obtained with syngeneic, allogeneic, and xenogeneic iRNA. Furthermore, iRNA extracted from tumor-immunized guinea pig or sheep lymphoid cells can equip normal human or mouse lymphocytes to kill tumor cells in vitro. It appears that T cells serve both as the source of the iRNA and as the recipient cells. Phase 1 and 2 clinical trials in humans have yet to produce a beneficial effect. More extensive trials are not warranted until more is known about what iRNA is, where it comes from, and what it does. (80 refs.)

78-3654 **Dual Mechanism of Surveillance of Malignant Cells by the Host.** (Ita) Alexander, P. (Div. of Tumor Immunology, Chester Beatty Res. Inst., Belmont, Sutton, Surrey, England). *Recent Prog Med (Roma)* 63(6): 641-648; 1977.

Immune surveillance over malignant cells is reviewed. The significant increase in tumor frequency and distant metastases in immune deficiency states indicates the important role of specific immunity in the control of malignant processes. (7 refs.)

78-3655 **The Radiobiological Significance of the Studies with ^{224}Ra and Thorotrast.** (Eng) Mole, R. H. (Medical Res. Council Radiobiology Unit, Harwell, Didcot, Oxfordshire, OX11 ORD, England). *Health Phys* 35(1): 167-174; 1978.

The results of three large national (Denmark, Portugal, and Germany) surveys of populations injected with Thorotrast (TT, thorium dioxide) are reviewed. In these countries the use of TT stopped 20 yr ago. All three surveys showed a similar excess of liver tumors (3%-8% of the affected population) and leukemia (1%). The pooled observations suggest that other malignant diseases arising in bone marrow (0.8 expected vs 5 observed) and lymphoid tissue (2 expected vs 10 observed) were also increased. Male lung cancer was in excess in Denmark and Portugal, and the combined excess of 8 cases/1,200 men is statistically significant. However, there was no evidence of such an excess in Germany. The risk coefficients on the linear hypothesis for leukemia, lung cancer, and osteogenic sarcoma from TT and other irradiations are compared. The high-risk coefficient for liver tumor induction and the pathology of the TT-bearing liver itself indicate that the convention of averaging dose over a whole organ may be misleading. Local deposits of TT provide sufficient local dose to produce severe local necrosis in this nominally radiation-resistant organ, regeneration in successive cycles could then lead to carcinogenesis. The contribution of localized irradiation by TT deposits to induction of malignant disease in bone marrow and lymph nodes is uncertain. This has to be allowed for if tumor induction in TT subjects is to be understood in terms of translocated daughters of thorium-232, including radium-224. (37 refs.)

78-3656 **Cancer and Trauma.** (Ger) Thomas, C. (Pathologisches Institut, Albertstrasse 19, D-7800 Freiburg, W. Germany); Moehrs, D.; Joachim, H. *Med Klin* 73(36): 1229-1237; 1978.

Literature concerning the theory that trauma leads to cancer is reviewed. In the older literature (1895-1906), it was estimated that about 20% of all tumors were due to by trauma. Increasing knowledge of carcinogenesis biostatistical methods has led to stricter criteria for assigning a causal role to trauma and a lower estimate of trauma-related tumors. The number of sarcomas that developed at the site of old World War I wounds was estimated to be 1/200,000 in 1949. A survey was made of 340 published cases of a connection between trauma and tumor development. The latency time between tumor and trauma varied from 2 wk to 64 yr. The time decreased as the av age of the patient increased. The peak incidence of these 340 tumors was at age 40-60, about 10 yr earlier than that for the general population. When tumors of early adulthood (sarcomas and testicular tumors) were removed from consideration, the peak age was the same as that in the general population. Study of the frequency curves indicated an association between irritating traumas and carcinomas and between blunt traumas and sarcomas. In contrast to the earlier literature, few mammary tumors were reported to be associated with trauma. Instead, it appears that trauma causes patients to find mammary tumors. Repeated trauma may lead to tumors through the action of promoting factors that are released when regeneration occurs. (42 refs.)

78-3657 **Biological Effects of Radiation and Dose-limiting Systems.** (Ita) Commissione Internazionale Per Le Protezioni Radiologiche (International Commission for Radiation Protection, Rimini, Italy). *Radiol Med (Torino)* 64(Suppl 1): 6-26; 1978.

Methods of estimating the risk of cancer development in various organs and tissues due to radiation, particularly low-LET (linear energy transfer) radiation, received at varying rates are reviewed. By calculating dose-response relationships, safe max levels of exposure for humans can be estimated, but these approximations must not be regarded as absolutes. The reproductive organs, hematopoietic system, bones, lungs, and thyroid glands are particularly susceptible to radiation-induced cancer. In Japanese atom bomb survivors, the incidence of leukemia peaked shortly after the time of exposure and finally returned to prebombing levels 25 yr later. It is estimated that the risk of developing leukemia or lung tumors becomes significant at a dose of 2 x 10^{-3} Sievert^{-1} (1 Sievert = 100 rem). Because the amount of radioactivity in the atmosphere varies, it is difficult to estimate the radiation risk for lung tumors in subjects exposed occupationally to radioactive material. There is a higher risk when the radioactive material is inhaled and uniformly distributed throughout the lung. Extensive proposals for limiting radiation doses received therapeutically, occupationally, and by the population at large are outlined. (no refs.)

78-3658 **Radiotherapy for Benign Dermatoses: Indications and Side Effects.** (Ger) Goldschmidt, H. (Suite 620, 1 Bala Cynwyd Plaza, Bala Cynwyd, PA, 19004). *Hautarzt* 29(5): 283-291; 1978.

The use of radiotherapy for benign dermatoses has decreased markedly during the past decades, and the remaining current indications are reviewed. The side effects of ionizing radiation are presented in detail, with special emphasis on radiodermatitis, skin cancer, the mutagenic and genetic effects of x-rays, and cancer of the internal organs. Modern theories of radiocarcinogenesis and risk coefficients of various organ systems are surveyed with the use of thyroid cancer as an example. Guidelines for the radiotherapy of benign dermatoses are proposed. (45 refs)

78-3659 **Hazards of Radiation Exposure for Pregnant Women.** (Eng) Swartz, H. M. (Radiation Biology and Biophysics Section, Dept. Radiology, Medical Coll. Wisconsin, Box 186, Milwaukee, WI, 53226); Reichling, B. A. *JAMA* 239(18): 1907-1908; 1978.

The risk of fetal genetic damage and congenital anomalies and cancer in the children of women irradiated during pregnancy is discussed. Radiation during the first trimester of pregnancy should be avoided, since a large dose (5-15 rads) to the pelvis increases the risk of a congenital anomalies from 1% to 3%. Low doses (0.2 rad) of radiation can increase the risk of a child developing cancer, but this risk is offset by the low risk of cancer (1/1,500) in the first 15 yr of life. Radiation to the gonads should be minimized to prevent mutations that could be passed to progeny. (5 refs.)

78-3660 **The Sensitivity of the Human Breast to Cancer Induction by Ionizing Radiation.** (Eng) Mole, R. H. (MRC Radiobiology Unit, Harwell, Oxon OX11 ORD, England). *Br J Radiol* 51(606): 401-405; 1978.

Evidence that ionizing radiation causes breast cancer in humans is reviewed, and the application of this evidence to practical problems is considered. Several studies have shown an increase in the incidence of breast cancer in women who have undergone multiple fluoroscopy for pulmonary tuberculosis or radiotherapy for nonmalignant diseases of the breast and in survivors of the bombing of Hiroshima and Nagasaki. These studies suggest that the max sensitivity to breast cancer induction by radiation is in women between the ages of 10 and 20 yr, and that this sensitivity may be correlated with hormonally regulated tissue activity. The minimum latent period before induced breast cancer was seen was 10-15 yr for fluoroscopy and radiotherapy and perhaps shorter in the Japanese bomb survivors; induced cases continued to appear, however, for up to 40 yr after exposure. The use of mammography and other diagnostic radiology procedures and occupational exposure and radiological protection are discussed. No recommendations are made about the use of radiotherapy in breast disease or about mammography as a screening procedure to detect early breast cancer. (17 refs.)

78-3661 Experience with Breast Biopsies for Mammographic Findings. (Eng) Cheek, J. H. (Dept. Surgery, Baylor Univ. Medical Center, Dallas, TX). *Breast* 4(1): 4-9; 1978.

Recent experience with breast biopsies performed because of mammographic findings is discussed. During a 7-yr study period, 3,833 new breast examinations were performed. There were 237 cancers among 253 patients biopsied for suspicious clinical and mammographic findings, 60 cancers among 81 patients biopsied for clinical reasons despite negative mammograms, 13 cancers in 13 patients with physical examinations and mammograms indicative of benign lesions, 21 cancers in 21 patients with suspicious clinical findings and no mammograms, and 7 cancers in 7 patients aged < 32 yr with clinical findings indicative of benign lesions and no mammograms. There was no significant difference in the numbers of patients above and below age 50 operated on for mammographic findings, although more cancers were found in patients aged > 50 yr. A majority of the lesions were infiltrating duct cell carcinomas or intraductal carcinomas. Indications for biopsy and mammography and mammographic indications for biopsy are listed. The findings suggest that if there are suspicious clinical and mammographic findings, the lesion is likely to be a carcinoma. (31 refs.)

78-3662 Mammography in Younger Women: The Concern, the Controversy, and the Confusion. (Eng) Frankl, G. (Kaiser-Permanent Medical Center, Los Angeles, CA). *Breast* 4(1): 14-15; 1978.

The concern, controversy, and confusion related to the role of mammography in women <50 yr old are discussed. The concern in younger women is about the hazard of radiation However, the minimal dose of 0.3 rad should not dissuad doctors from performing a potentially life-saving examination. Breast cancer is an important entity in women aged 35-50 yr. One-third of the 708 breast cancers found among 20,214 patients examined by xeromammography at Kaiser-Permanente Foundation Hospital (Los Angeles) occurred in women <50 yr old. Moreover, 31% of all the occult cancers among this population were in younger women. With current equipment and techniques, which allow penetration of young, dense breast tissue, the detection rate for younger women has improved. Recommended indications for performing mammography are given. (no refs.)

78-3663 Adipose Tissue, a Neglected Factor in Aetiology of Breast Cancer? (Eng) Beer, A. E. (Dept. Cell Biology, Univ. Texas Health Science Center at Dallas, 5323 Harry Hines Blvd., Dallas, TX, 75235); Billingham, R. E. *Lancet* 2(8084): 296; 1978.

The adipose tissue in the breast may contribute significantly to the hypersusceptibility of this organ to cancer development by prolonging the duration of exposure of mammary epithelium to lipid-soluble endogenous and environmental carcinogens via a reservoir or depot effect. (8 refs.)

78-3664 Cancer in Old Age. (Eng) Lew, E. A. (No affiliation given). *Ca* 28(1): 2-6; 1978.

In the last decade, new information concerning mortality patterns in the aged population of the US has become available. The 1969-1971 US cancer incidence rates (from the Third National Cancer Survey) and death rates (from the National Center for Health Statistics) for whites aged 75-99 in selected areas are presented. The data are for cancer at all sites and for cancer of the digestive system, respiratory system, prostate, and breast. The incidence and death rates for all forms of cancer tend to increase from age 65 to the late 80's, after which they decline. Elderly patients are frequently reported as dying from intercurrent disease, especially when the symptoms of cancer are silent and metastasis is not apparent. Heart and cerebrovascular diseases are often given preference over cancer as causes of death when both are present. The elderly may constitute a biologically select group that may be relatively insusceptible to cancer, at least insofar as a high proportion of cancer-prone individuals might be expected to have died by age 85. (no refs.)

78-3665 Epidemiology of Gastrointestinal Tumours: A Review. (Eng) McConnell, R. B. (Gastroenterology Unit, Broadgreen Hosp., Thomas Drive, Liverpool L14 3LB, England). *J R Soc Med* 71(4): 278-281; 1978.

The epidemiology of gastrointestinal tumors is reviewed. For different tumors there exists not only a variation between countries, but also a variation within countries, as illustrated by the high occurrence of colon cancer in industrialized countries, except Japan, and the finding that gastric cancer is more common in the lower socioeconomic strata. Incidences tend to change with time, as shown by the dramatic fall in gastric cancer incidence in the US and the less-dramatic fall in Canada. Various epidemiological factors in cancer of the esophagus are outlined. (16 refs.)

78-3666 **The Aetiology of Cancer: Clues from Spontaneous Recovery.** (Eng) Merkin, L. (Dept. Medicine, Roosevelt Hosp., New York, NY). *Med Hypotheses* 4(2): 136-140; 1978.

A review of the literature indicates that several patients, some with highly invasive tumors, survive > 15 yr after standard treatment with no evidence of disease. It is suggested that persons with a long-term disease-free survival be questioned extensively in an attempt to discover some factor, perhaps unique to all, that could have been involved in the origin of the cancer and its cure. (8 refs.)

78-3667 **Genetics of Gastrointestinal Polyposis.** (Eng) Bussey, H. J. (Pathology Dept., Saint Mark's Hosp., City Road, London CE1V 2PS, England); Veale, A. M.; Morson, B. C. *Gastroenterology* 74(6): 1325-1330; 1978.

The types of gastrointestinal polyposis with a possible or proved genetic etiology are (1) the hamartomatous lesions of Peutz-Jeghers syndrome and multiple juvenile polyposis and (2) the neoplastic tumors of familial polyposis coli and multiple adenomas. The mode of inheritance, associated lesions, malignancy potential, and possible interrelationships among these conditions are discussed. The degree of associated malignancy is slight in the Peutz-Jeghers syndrome and juvenile polyposis, but it is definite and strong in familial polyposis coli and multiple adenomas. The knowledge that conditions predisposing to gastrointestinal cancer can be inherited should enable their diagnosis at an early stage. The extent to which cancer prevention can be effective has been shown in the case of familial polyposis. (50 refs.)

78-3668 **Cancer Death Risk in Hospitalized Mental Patients (Letter to Editor).** (Eng) Fox, B. H. (NCI, Bethesda, MD, 20014). *Science* 201(4360): 966-968; 1978.

The statistical methods used in previous studies of the risk of cancer death among hospitalized mental patients are analyzed. In these studies, the mental patients were found to have both lower and higher cancer death rates than those of the general population. Most of these findings can be explained on the basis of whether proportionate or absolute mortality rates were used. Most studies using the latter, more accurate, method have found that the overall cancer mortality in mental patients is greater than that in the general population. (14 refs.)

78-3669 **Genetics and Human Cancers: Methods of Study.** (Fre) Feingold, J. (Groupe de Recherches de Genetique epidemiologique, INSERM, U. 155, Chateau de Longchamp, Bois de Boulogne, F76016 Paris, France). *Bull Cancer (Paris)* 65(1): 73-77; 1978.

The following method of determining the hereditary nature of the common cancers are reviewed: (1) study of twins; (2) familial studies involving statistics on mortality or comparisons of certain groups with cancer and matched controls; (3) interracial studies; (4) studies of the association between genetic markers and cancer incidence. Certain problems arise with the last method, such as whether the presence of a certain gene is simply a predisposing factor rather than an absolute condition for development of a cancer, whether the existence of the gene marker and the cancer in a subpopulation is coincidental or causal, etc. A familial concentration of certain cancers may be due to exposure to a common environment or to the presence of heritable genetic factors. (27 refs.)

78-3670 **Current Assessment of "Case Clustering" of Lymphomas and Leukemias.** (Eng) Smith, P. G. (DHSS Cancer Epidemiology and Clinical Trails Unit, Univ. Oxfordk, 9 Keble Road, Oxford, England). *Cancer* 42(2, Suppl): 1026-1034; 1978.

Data on case clusters of lymphomas and leukemias are reviewed and problems associated with epidemiologic studies of clustering designed to detect a viral etiology are discussed. Many reported case clusters of leukemia and lymphoma are impossible to evaluate statistically, but controlled studies of space-time clustering involving patients with leukemia and Hodgkin's disease (HD) have not yielded convincingly positive findings. Clustering would not be expected, however, if the period between infection and onset were long. A study of childhood leukemia, assuming a possibly long latent period, did not produce evidence of case-to-case transmission. Studies of HD are conflicting: strong evidence of clustering of cases in schools was found in one US study, but a similar British study had negative results. Further studies are necessary to determine whether case-to-case transmission can take place. There is indirect evidence that suggests that HD might arise as a rare response to a common infection. In such circumstances, case clustering of HD would not necessarily be expected to occur. Space-time clustering of Burkitt's lymphoma (BL) has been a distinctive feature of the disease, but it has not been seen in all areas where BL is endemic. In a recent study, patients involved in such clusters were, on the av, older than other patients, which supports the notion that the clustering is a real effect and does not represent an artifact of case ascertainment. Current evidence suggests that most cases of "African" BL arise as a result of an interaction between Epstein-Barr virus infection and chronic severe malaria. If so, it is likely that it is the malaria that precipitates the onset of BL and that clustering of cases is likely to be related with malarial infection. (63 refs.)

78-3671 **Multiple Etiologies of Human Acute Leukemia?** (Eng) Killmann, S. A. (Div. Hematology, Dept. Medicine A, Rigshospitalet, 9, Blegdamsvej, DK-2100 Copenhagen, Denmark). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 431-435; 1977.

That human acute leukemias have a multiple etiology is suggested in part by geographical differences in chromosome aberrations (trisomy 8) in acute myeloid leukemia (AML) patients. Additional evidence is derived from studies of histocompatibility antigens in black children with AML or acute lymphoblastic anemia (ALL) and studies of WBC group 5 antigens in AML and ALL patients and their families. The monoclonal origin of chronic myeloid leukemia is discussed together with the reported antineoplastic effects of certain virus infections. (14 refs.)

78-3672 **The Biosynthesis of Oncovirus Proteins.** (Eng) Eisenman, R. N. (Fred Hutchinson Cancer Res. Center, Seattle, WA, 98104); Vogt, V. M. *Biochim Biophys Acta* 473(3/4): 187-239; 1978.

The biosynthesis of oncovirus proteins is reviewed with particular emphasis on the biosynthetic steps involved in the regulation of virus growth and how synthesis and modification are related to function. The patterns of oncovirus protein biosynthesis are essentially similar for avian and mammalian viruses, and in each case, the four major internal structural proteins are synthesized from a common *gag* gene precursor. The products of the *pol*, *env*, and *src* genes are also discussed. (385 refs.)

78-3673 **Comparative Epidemiology of Virus-Induced Leukemias.** (Eng) Jarrett, O. (Dept. Veterinary Pathology, Univ. Glasgow, Glasgow, Scotland). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 11-17; 1978.

The epidemiology of virus-induced leukemias in domestic poultry, cats, and cattle is discussed in relation to its relevance to the study of leukemia and related diseases in other animals, including humans, for which evidence of a viral etiology is still equivocal. Avian leukosis virus (ALV) is common in domestic poultry and is maintained by congenital transmission from persistently infected and viremic hens, through their eggs, to their chicks. Contact transmission of ALV is less important for the maintenance of the virus cycle. Contact and congenital transmission of feline leukemia virus (FeLV) also occurs in cats. The development of FeLV-related disease appears to be related to the degree of contact between infected and uninfected animals and, hence, to the dose of virus. A low dose effectively immunizes the recipient without causing disease, whereas a high dose leads to overt disease. Leukemia in cattle appears to occur in two forms: an enzootic form in which adult multicentric disease is typical and a sporadic form which adolescent thymic or calf multicentric disease is found. A viral etiology of leukemia has been proved in all three species. However, in both cats and cattle, virus-negative cases of leukemia are found, and study of the epidemiology of these leukemias will be needed to determine how they relate to the natural history of FeLV and bovine leukemia virus and how closely they resemble human leukemia. (28 refs.)

78-3674 **Carcinogenesis due to the Type C Oncornavirus. Biological, Immunological and Genetic Aspects.** (Ita) Chieco-Bianchi, L. (Istituto di Anatomia Patologica, Padova, Italy); Collavo, D.; Colombatti, A.; Biasi, G. *Recent Prog Med (Roma)* 63(6): 592-621; 1977.

There is no direct evidence of oncornavirus being the cause of neoplasm formation in humans. However, C-type oncornavirus was isolated from human leukemia and sarcoma cells in culture. (115 refs.)

78-3675 **Viruses and Mammary Neoplasia in Mice and Men.** (Eng) Bentvelzen, P. (Radiobiological Inst. TNO, Rijswijk, Netherlands). *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 7-19; 1978.

The role of mammary tumor viruses MTV) in murine breast cancer is reviewed. Particular attention is focused on the properties of murine MTV, their transmission, genetic factors controlling their expression, epigenetic factors involved in their expression, the immunological reactivity of mice to MTV, and the vaccination of mice against mammary tumor development. The viral etiology of murine mammary tumors but not that of human breast tumors, has been proved. MTV sequences have been found in the cellular RNA of human breast cancers, but their role in the human disease is conjectural. If the MTV-related viral genome present in human cellular DNA should be oncogenic to humans, its major influence would be in premenopausal breast cancer cases. (89 refs.)

78-3676 **Common Progenitors in the Evolution of Mammalian Retroviruses: Implications in the Search for RNA Tumor Virus Expression in Man.** (Eng) Aaronson, S. A. (Lab. RNA Tumor Viruses, NCI, Bethesda, MD, 20014); Barbacid, M.; Hino, S.; Tronick, S. R.; Krakower, J. M. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 127-133; 1978.

Information on the distribution and transmission of endogenous C- and D-type mammalian retroviruses is summarized. The evolution of C-type viruses from a common ancestor has been supported by radioimmunoassays of the translational product of the C-type viral genome. Of the known products, structural proteins p30, p15 (p12 of the baboon/RD114 virus group), and p10 and reverse transcriptase contained interspecies antigenic determinants shared by all mammalian C-type viruses isolated to date. Competition radioimmunoassays have also established that endogenous D-type viruses, encompassing isolates of both New and Old World primates, are antigenically and hence genetically related. The application of radioimmunoassays for detecting retrovirus expression in humans is discussed. (26 refs.)

78-2677 **Cell Transformation in Culture and Cutaneous Carcinogenesis.** (Ita) Andreassi, L. (Sienai, Italy). *G Ital Dermatol* 113(1-2): 29-50; 1978.

Human epithelial cell cultures are suitable for studying the neoplastic transformation of cells by papilloma virus and chemical carcinogens and the subcellular mechanisms of transformation. (169 refs.)

78-3678 **Skin Tissue Culture and Verruca and Molluscum Contagiosum Virus.** (Ita) Spagnoli, U. (Siena, Italy); Baiocchi, R. *G Ital Dermatol* 113(1-2): 79-86; 1978.

Human epidermal cell cultures are suitable for the study of cell transformation due to papilloma virus and molluscum contagiosum virus. These viruses are unable to form complete virus particles in the absence of keratinization. (34 refs.)

78-3679 **Fibronectins--Adhesive Glycoproteins of Cell Surface and Blood.** (Eng) Yamada, K. M. (Lab. Molecular Biology, NCI, Bethesda, MD, 20014); Olden, K. *Nature* 275(5677): 179-184; 1978.

The structure, location, and probable functions of fibronectins (FN's), a class of adhesive, high-mol-wt glycoproteins present on the surfaces of cells, in connective tissue matrices, and in extracellular fluids, are reviewed. Cell-surface FN constitutes 1%-3% of the total cellular protein of embryonic or adult fibroblasts cultured in vitro. Most of the FN is present in extracellular fibrils and aggregates. Plasma FN is present in human plasma at 0.3 mg/ml and at a lower concentration in serum. The tumorigenicity of cell lines generally correlates with decreases in cell-surface FN. However, recent studies indicate that decreases in this protein may correlate more with the propensity of the cells to metastasize. Decreases in cell-associated FN after malignant transformation are 5- to 7-fold in chick fibroblasts and > 10-fold in human fibroblasts and glial cells. FN's are thought to be involved in a wide variety of cellular activities, including cell-cell aggregation, cell-substratum adhesiveness, reversion of the transformed phenotype, increased cell motility, reticuloendothelial system clearance of colloids, and specific binding to macromolecules. It is not clear if all of these biological activities are shared by both the plasma and cell-surface forms of FN. In transformed chick fibroblasts and hamster cells, major factors in the loss of FN after transformation appear to be reduced biosynthesis and increased degradation. In human transformants, however, decreased capacity to retain the protein on the cell surface may be more important than decreased synthesis. (175 refs.)

78-3680 **Mutagenic Effect of Aflatoxin B_1.** (Eng) El-Zawahri, M. (Dept. Genetics, Assiut Univ., Assiut, Egypt); Moubasher, A.; Morad, M.; El-Kady, I. *Ann Nutr Aliment* 31(4/5/6/): 859-866; 1977.

The mutagenic effect of aflatoxin B_1 on human peripheral blood lymphocytes was investigated. Aflatoxin B_1 induced a high rate of nonrandom chromosome breaks and interchanges. A highly significant number of breaks were selectively induced on chromosomes 2, 11, 19, and 20. The high breakage rate was accompanied by an increase in sister chromatid exchanges. (14 refs.)

78-3681 **Morphological Observations of Aflatoxin-induced Liposis in Rat Hepatocytes.** (Eng) Kendall, M. W. (Dept. Anatomy, Sch. Medical Sciences, 1 Manville Medical Sciences Building, Univ. Nevada, Reno, NV, 89557). *Chemosphere* 7(8): 625-632; 1978.

Light and electron microscope studies were made of hepatocytes from male weanling Fischer rats fed aflatoxin B_1 (AFB) in order to determine the cell organelle alterations of AFB-induced liver toxicity, particularly the accumulation of lipid. Rats were fed Purina rat chow with or without the addition of 4.0 ppm AFB. Animals were killed and their livers prepared for histological study at the end of every 3 mo for 15 mo. AFB-treated animals were also sacrificed on day 1. At the end of 15 mo, 12/20 rats fed AFB had hepatocellular carcinomas. No fatty metamorphosis, lipid accumulation, or tumors were observed in livers from control rats over the 15 mo period. Lipid was observed in the periportal hepatocytes in 18/20 AFB-fed rats sacrificed at 3 mo, although it probably occurred much earlier. Electron microscope study of these hepatocytes revealed myelin figures and lipid vacuoles with numerous vesicles of smooth endoplasmic reticulum (SER). The lipid may have originated in the SER vesicles. The accumulation of lipid in the hepatocytes did not seem to increase with duration of treatment. Centrilobular hepatocytes were not affected by AFB. It is concluded that the cytoplasmic lipid accumulations in the periportal hepatocytes probably reflect nucleolar biochemical alterations that eventually lead to hepatoma. (17 refs.)

78-3682 **Epidemiological Features of the Mycotoxicoses.** (Eng) Akkmeteli, M. A. (Div. Noncommunicable Diseases, World Health Organization, Geneva, Switzerland). *Ann Nutr Aliment* 31(4/5/6): 957-976; 1978.

Several features associated with the outbreak of mycotoxicoses in man and animals are presented. The possible role of mycotoxins in endemic nephropathy in some East European countries and liver disease in Mongolia is discussed. It is suggested that these two diseases, as well as stachybotryotoxicosis and alimentary toxic aleukia, have common epidemiological features, but in the former two, these features are spread out over a long period of time. The toxic and carcinogenic metabolites of fungi are believed to play important roles in these diseases. (40 refs.)

78-3683 **Transmission of Aflatoxin B_1 into the Tissues of Growing Pigs.** (Eng) Jacobson, W. C. (Bureau Veterinary Medicine, Food and Drug Admin., Beltsville, MD, 20705); Harmeyer, W. C.; Jackson, J. E.; Armbrecht, B.; Wiseman, H. G. *Bull Environ Contam Toxicol* 19(2): 156-161; 1978.

The levels of aflatoxin B_1 (AFB) and aflatoxin M_1 (AFM) in the tissues of pigs fed diets containing 100, 200, or 400 μg AFB/kg feed were determined. Appreciable amounts of AFB were found in tissue samples of animals fed all three diets; measureable amounts of AFM were found in all specimens except two (both muscle). Liver tissue had no evidence of pathologic damage. Mathematical analysis indicated a linear relationship between the logs of AFB intake and the amounts of residues in the tissues. (10 refs.)

78-3684 **Inhibition of Mitochondrial DNA Synthesis by Aflatoxin B_1 and Dimethylnitrosamine.** (Eng) Friedman, M. A. (Allied Chemical Corporation, P.O. Box 1021R, Morristown, NJ, 07960); Bailey, W.; Van Tuyle, G. C. *Res Commun Chem Pathol Pharmacol* 21(2): 281-294; 1978.

The acute and subchronic effects of aflatoxin B_1 (AFB) and dimethylnitrosamine (DMN) on mouse liver mitochondrial DNA synthesis were determined using male C3H/He mice. In single acute injection experiments, mice received 20 mg/kg of the test carcinogen ip and were killed 16 hr later. In the subchronic experiments, mice received a weekly injection of 0.16 mg DMN or 0.23 mg AFB. During the week of sacrifice, these mice received an extra dose of AFB 2 days after their regular injections and they were killed 18 hr later. At 10, 20, 30, and 40 min after a single injection of AFB, labeled thymidine (TdR) uptake by mitochondrial DNA was inhibited to 49%, 46%, 42%, and 46% of the control uptake, respectively. This inhibition appeared to represent a general inhibition of DNA synthesis together with loss of replicative intermediates. After a single injection of DMN, TdR uptake was inhibited to 24%, 54%, 59%, and 51% of control, respectively. As with AFB-treated mice, incorporation into high-mol-wt mitochondrial DNA was markedly inhibited, the effect on low-mol-wt mitochondrial DNA synthesis was less marked. After 5 wk of AFB treatment, TdR uptake was inhibited to 45%, 56%, 48%, and 51% of controls at incubation times of 10, 20, 30, and 40 min, respectively. With DMN, the respective inhibition was 68%, 72%, 72%, and 70% of controls. After 13 wk of AFB treatment, the AFB diet, inhibition of mitochondrial DNA synthesis at incubation times of 10, 20, 30, and 40 min was 51%, 46%, 41%, and 47% of controls. The synthesis of high-mol-wt DNA was decreased to the same extent as gross TdR uptake. The respective figures for DMN were 46%, 50%, 37%, and 46% of controls. However, in DMN-treated mice, there appeared to be a uniform inhibition of TdR incorporation that appeared to be greater in magnitude than that suggested by gross TdR incorporation. (12 refs.)

78-3685 **Trout Hepatic Enzyme Activation of Aflatoxin B_1 in a Mutagen Assay System and the Inhibitory Effect of PCBs.** (Eng) Stott, W. T. (Dept. Food Science and Technology, Oregon State Univ., Corvallis, OR, 97331); Sinnhuber, R. O. *Bull Environ Contam Toxicol* 19(1): 35-41; 1978.

Optimal conditions for use of the Ames assay with rainbow trout (*Salmo gairdneri*) liver postmitochondrial fraction (PMF) were defined using aflatoxin B_1 (AFB) as the test compound. In addition, the effect of several polychlorinated biphenyl (PCB) compounds on the response of the assay to AFB was investigated. The experiments clearly indicated the ability of fish liver drug-metabolizing enzymes to function in the Ames mutagen assay. Optimal mutagen assay conditions were obtained with 6.4 mg PMF protein and 160 nanograms AFB/incubation mixture and a metabolic incubation temperature of 25 C. A significant increase in the number of revertants was observed when metabolic incubations were performed at 25 C for 9 hr. When the fish were pretreated ip with 500 mg/kg of Aroclor 1242, Aroclor 1254, or Aroclor 1260, there was a significant reduction in the assay response vs the control response. This result was in contrast to the results of assays using rat PMF. The mutagen assay response with fish PMF decreased with increasing chlorination of the PCB's, which may reflect the ability of PCB's as inducers of these enzyme systems in rainbow trout. This finding appears to substantiate recent findings in rainbow trout feeding trials of Aroclor 1254 and AFB in which PCB-treated fish had a lower AFB-induced incidence of cancer. (30 refs.)

78-3686 **Aflatoxicosis in Swine.** (Eng) Hayes, A. W. (Dept. Pharmacology and Toxicology, Univ. Mississippi Medical Center, Jackson, MS, 39216); King, R. E.; Unger, P. D.; Phillips, T. D.; Hatkin, J.; Bowen, J. H. *J Am Vet Med Assoc* 172(11): 1295-1297; 1978.

Necropsy studies of several pigs from a herd in which 30/250 had died within 1 mo indicated that they had aflatoxicosis. Histiopathologic findings were characteristic of experimentally induced aflatoxicosis, and aflatoxin B_1 (AFB_1) was found in the serum. AFB_1 and AFB_2 were found in the feed corn. Combine harvesting, which cracks the kernels, plus a prolonged drying time probably contributed to the AFB production. (15 refs.)

78-3687 **Aflatoxicol: Major Aflatoxin B_1 Metabolite in Rat Plasma.** (Eng) Wong, Z. A. (Dept. Environmental Toxicology, Coll. Agricultural and Environmental Sciences, Univ. California, Davis, CA, 95616); Hsieh, D. P. *Science* 200(4339): 325-327; 1978.

Aflatoxicol (AFL), a carcinogenic metabolite of aflatoxin B_1 (AFB), was identified as the major aflatoxin metabolite in the plasma of Sprague-Dawley rats, a susceptible species, that had been dosed po (3.0 mg/kg) or iv (1.0 mg/kg) with ^{14}C-AFB. AFL, however, was not detected in the plasma of similarly dosed mice and monkeys, which are resistant to AFB-induced carcinogenesis. The formation of AFL both in vitro and in vivo may be an indicator of species sensitivity to AFB-induced carcinogenesis and may be useful in the prediction of human susceptibility. (26 refs.)

78-3688 **The Effects of Asbestos-Cement Dust Inhalation on Baboons.** (Eng) Goldstein, B. (Natl. Res. Inst. Occupational Diseases, South African Medical Res. Council, P.O. Box 4788, Johannesburg, S. Africa); Webster, I.; Rendall, R. E.; Skikne, M. I. *Environ Res* 16(1/3): 216-225; 1978.

Twelve male baboons (*Papio ursinus*) were exposed for 266-444 days to dust collected from an extraction system in the hard-sheet cutting section of an asbestos-cement factory. The sheets were composed of 12% asbestos (1.2% crocidolite and 10.8% chrysotile) and 88% cement. The exposure sessions were 7 hr/day, 5 days/wk. The mean fiber count of the dust was 166 fibers/cm^3 and the respirable dust concentration was 17.7 mg/m^3. The length of the fibers ranged from 5 to 100 μm, and 80% of the cement particles were < 10 μm. By 266 days, the animals developed lung fibrosis that was morphologically similar to asbestosis. In all cases, asbestos fibers and dust particles were noted in the fibrotic areas. There were also occasional ferrunginous bodies. Two animals, exposed for 266 and 441 days, respectively, also developed atypical metaplasia of the bronchiolar epithelium, and a bronchiolo-alveolar carcinoma could not be excluded. (5 refs.)

78-3689 **Hemolytic Action of Asbestos.** (Fre) Vaille, C. (69, boulevard de Beausejour, 75016 Paris, France); Souchard, M. *Ann Pharm Fr* 36(1/2): 23-32; 1978.

In vitro, Canadian, Corsican, and Rhodesian chrysotile asbestos caused hemolysis in human and rat blood. The South African amosite and crocidolite forms of asbestos did not produce this effect, and the degree of hemolysis due to Finnish anthophyllite was only one-third that due to chrysotile asbestos. In vivo, hemolysis can be measured by the amount of bilirubin in the bile after iv injection of various types of asbestos. Iv injection of the three chrysotiles, crocidolite, and amosite into Wistar CF rats caused hemolysis, but anthophyllite did not. Inhalation exposure of the rats to chrysotile aerosols did not produce a hemolytic effect. (20 refs.)

78-3690 **Malignant Mesothelioma and Gas-Mask Assemblers.** (Eng) Jeffreys, D. B. (Guy's Hosp., London SE1 9RT, England); Vale, J. A. *Br Med J* 2(6137): 607; 1978.

The case histories of two employees of gas mask assembly plants who developed malignant mesothelioma are presented. In each case, the employees had worked on masks with filters containing asbestos. The first was an 83-yr-old woman who developed mesothelioma 63 yr after exposure during the first world war and the second was a 62-yr-old man who developed the disease 37 yr after exposure during the second world war. (5 refs.)

78-3691 **Familial Mesothelioma after Intense Asbestos Exposure at Home.** (Eng) Li, F. P. (Clinical Studies Section, Clinical Epidemiology Branch, NCI, 35 Binney St., Boston, MA, 02115); Lokich, J.; Lapey, J.; Neptune, W. B.; Wilkins, E. W. *JAMA* 240(5): 467; 1978.

Pleural mesotheliomas occurred in the wife and daughter of an asbestos handler, indicating that gross asbestos contamination of the home may be a hazard. The father, who had worked as a pipe insulator in a shipyard from 1940 to 1965, was diagnosed at age 60 with pulmonary asbestosis. He died at age 71 due to metastatic adenocarcinoma in the upper left lobe of the lung. He also had been a heavy cigarette smoker for several decades. The mother and daughter, who experienced no occupational exposure to asbestos, developed right pleural effusions at ages 50 and 34, respectively. Thoractomy/thoracentesis and pleural biopsy specimens revealed the mesotheliomas. The daughter's tumor had a biphasic or mixed pattern of epithelial-type cells with papillary formations, glandlike spaces, and spindle cell metaplasia. The mother's tumor was similar in appearance but with more extensive fibrosis. Two younger daughters were found to be free of cancer. During World War II and several years thereafter, the father had experienced intensive exposure to asbestos insulation dusts, which covered his work clothes. He brought these asbestos-covered clothes into the home. As no other source of asbestos exposure was identified, asbestos pollution of the home is considered a cause of cancer in the mother and daughter. Contributing factors may include a genetic susceptibility to cancer in this family and the early age of exposure in the daughter. (3 refs.)

78-3692 **Asbestos Exposure and Cancer of the Larynx.** (Fre) Bianchi, C. (Institut d'anatomie et histologie pathologique, l'Universite de Trieste, Trieste, Italy); Di Bonito, L.; Castelli, M.; Brollo, A. *Pathologica* 70(1005/1006): 403-408; 1978.

Exposure to asbestos and use of tobacco (in cigarettes) were compared in 60 patients (58 men, 2 women) who underwent laryngectomy (LG) for epidermoid carcinoma of the larynx and in 60 sex- and age-matched controls who were hospitalized for noncancerous diseases. The LG patients ranged in age from 30 to 89 yr, but most were aged 40-69 yr. Some patients had laryngeal surgery as long ago as 22 yr, but most had been operated after 1970. Fifty-eight of the LG patients were smokers vs 44 controls. The difference between the two groups in av daily number and total number of cigarettes smoked was significant ($p < 0.02$); but not the difference in duration of smoking habit. There was also no significant difference ($p < 0.1$) in the number of LG patients and controls exposed to asbestos. Four LG patients and three controls had a history of heavy exposure; otherwise, the degree of exposure could not be determined. There was a history of exposure to other respiratory irritants (gas, smoke from soldering operations and combustion) that frequently coincided with asbestos exposure in 40 LG patients and 35 controls. A combination of exposure to asbestos and smoking habit was more frequent ($0.05 < p < 0.07$) in the LG group. (19 refs.)

78-3693 **Hazards of Inorganic Arsenic (Letter to Editor).** (Eng) Nurse, D. S. (71 Mayston St., E. Hawthorne, Victoria 3123, Australia). *Med J Aust* 1(2): 102; 1978.

A 45-yr-old man developed an adenocarcinoma of the kidney 17 yr after cessation of 5-yr of inorganic arsenic treatment for psoriasis. One year prior to diagnosis of the renal tumor, a basal cell carcinoma was removed from his neck. It is suggested that the arsenic played a significant role in the development of the renal carcinoma. (1 ref.)

78-3694 **Occupational and Environmental Risks in and Around a Smelter in Northern Sweden. II. Chromosomal Aberrations in Workers Exposed to Arsenic.** (Eng) Nordenson, I. (Dept. Medical Genetics, Univ. Umea, S-901 85 Umea, Sweden); Beckman, G.; Beckman, L.; Nordstrom, S. *Hereditas* 88(1): 47-50; 1978.

The frequency of chromatid aberrations was investigated in smelter employees with high, medium, and low exposure to arsenic, newly employed persons (most of whom lived near the smelter), and controls. A significantly increased frequency in aberrations was found in all employees compared with controls; however, the correlation between frequency of aberrations and As exposure was rather poor, probably because of individual susceptibility and a proposed cocarcinogenic action of As with cigarette smoking. As may inhibit DNA repair of aberrations caused by other agents. (9 refs.)

78-3695 **Urothelial Hyperplasia of the Urinary Bladder of the Rat Induced by Mechanical Perforation and Phenacetin Treatment.** (Eng) Johansson, S. (Dept. Pathology II, Sahlgren's Hosp., 413 45 Goteborg, Sweden). *Acta Pathol Microbiol Scand (A)* 86(4): 333-335; 1978.

The combined effects of mechanical trauma and phenacetin treatment on the urinary bladder were studied in male Sprague-Dawley rats. Twelve rats were subjected to mechanical perforation of the bladder (with a 23-gauge needle) and dietary phenacetin (0.535%). Four rats received phenacetin only, four perforation only. Rats from each group were sacrificed at 1, 2, and 3 wk after treatment, and the bladder and kidneys were examined histologically. Eleven of the twelve rats given the combined treatment developed urothelial hyperplasia. In two of the rats, the entire bladder mucosa was involved. All of the rats subjected to perforation alone or phenacetin treatment alone had a normal bladder mucosa. The renal pelvis and papillae of all rats appeared normal. It was not possible to determine whether the urothelial hyperplasia was reactive or preneoplastic. (8 refs.)

78-3696 **Mutagenic Effect of Orally Given AF-2 on Embryonic Cells in Pregnant Syrian Hamsters.** (Eng) Inui, N. (Dept. Experimental Pathology, Cancer Inst., Japanese Foundation Cancer Res., Toshima-ku, Tokyo 170, Japan); Nishi, Y.; Taketomi, M. *Mutat Res* 57(1): 69-75; 1978.

The mutagenic effect of 2-(2-furyl)-3-(5-nitro-2-furyl)acrylamide (AF-2) was studied in hamster embryo cells. Syrian golden hamsters were given 2, 5, 10, 50, or 100 mg/kg AF-2 by gastric intubation on the 11th or 12th day of pregnancy, and their fetuses were excised 24 hr later. Cells were isolated from the embryos and cultured in normal medium. Chromosome preparations were made within 24 hr after the start of primary culture. Marked chromosomal abnormalities were observed in cells from embryos of animals treated with >20 mg/kg AF-2. Samples of surviving cells were also cultured in normal medium for 48 hr and then selected in medium containing 8-azaguanine (8AG) or 6-thioguanine (6TG). AF-2 treatment caused a marked dose-dependent induction of 8AG- or 6TG-resistant mutations; mutant colonies were even obtained after a single treatment with 2 mg/kg AF-2. It is concluded that this mammalian system is sensitive and useful for detecting environmental mutagens. (19 refs.)

78-3697 **Mutagenicity of Acrylonitrile.** (Eng) De Meester, C. (Lab. Analytical Chemistry, Univ. Louvain, Sch. Pharmacy, U.C.L.-73.69, B. 1200 Brussels, Belgium); Poncelet, F.; Roberfroid, M.; Mercier, M. *Toxicology* 11(1): 19-27; 1978.

Incubation of several *Salmonella typhimurium* strains in an atmosphere of 0.2% gaseous acrylonitrile increased the number of cells that underwent a reversion to histidine prototrophy only in the presence of a fortified postmitochondrial (S_9) liver fraction. The mutagenic effect was particularly marked with strains TA1530, TA1535, and TA1950 and was much weaker with strains TA100, TA98, and TA1978. The results of bacterial fluctuation tests confirmed the absence of a direct mutagenic effect of AN and the particular sensitivity of strain TA1530 can the presence of the S_9 mix. The mutagenicity of AN in *S. typhimurium* is this microsome-mediated and is optimal with strains sensitive to base-substitution mutagens. Studies of the effect of the composition and origin of the S_9 mix showed that 300 μl S_9/ml mix and a mouse origin produced the highest numbers of revertants per plate. Liver fractions from Arochlor 1254- or 3-methylcholanthrene-treated rats produced the greatest mutagenic effects, followed by those from butadiene- and styrene-treated rats. (18 refs.)

78-3698 **Trapping and Determination of Labile Compounds in the Gas Phase of Cigarette Smoke.** (Eng) Zeldes, S. G. (Analytical Chemistry Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Horton, A. D. *Anal Chem* 50(6): 779-782; 1978.

Adsorption-gas chromatographic techniques that allow the trapping and recovery of all the isoprene and acrolein and 80% of the acetaldehyde in the gas phase of cigarette smoke are presented. Optimum conditions for the adsorption and desorption of the gas phase and the effects of aging on the trapped gases were also determined. (9 refs.)

78-3699 **Induction of 6-Thioguanine Resistance in Human Cells Treated with N-Acetoxy-2-acetylaminofluorene.** (Eng) Huang, S. L. (Lab. Environmental Mutagenesis, Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC, 27709); Lieberman, M. W. *Mutat Res* 57(3): 349-358; 1978.

Induction of 6-thioguanine resistance was studied in human fibroblasts treated with the direct-acting chemical carcinogen N-acetoxy-2-acetylaminofluorene (NA-AAF). At low

NA-AAF concentrations (2.5 to 7.5 μM), induction of resistant clones was linear and followed one-hit kinetics. At 10 μM, some higher order function appeared to be involved. Only 2.5 to 3.0 of every 10,000 NA-AAF molecules bound at the hypoxanthine-guanine phosphoribosyltransferase (HGPRT) locus resulted in detectable mutations. Fifty mutant colonies were evaluated for growth in 6-thioguanine-containing medium. About 12% of the clones had low ($<8\%$ of wild-type) HGPRT activity and showed poor ability to grow in hypoxanthine plus azaserine (HAs) medium. Most of the other clones had 25% to 80% as much HGPRT activity as wild-type cells and grew in HAs medium. There was no correlation between the NA-AAF concentration used to induce the mutations and the enzymatic activity or growth characteristics of the cells. The data suggest that most bound acetylaminofluorene molecules either produce no change in the primary DNA sequence or result in changes that are phenotypically undetectable. (17 refs.)

78-3700 **Induction of Sister-Chromatid Exchanges in Mammalian Cells by Low Concentrations of Mutagenic Carcinogens that Require Metabolic Activation, as well as by Those that Do Not (Meeting Abstract).** (Eng) Wolff, S. (Lab. Radiobiology and Dept. Anatomy, Univ. California, San Francisco, CA); Takehisa, S. *Mutat Res* 53(2): 284-285; 1978. (no refs.)

78-3701 **Dietary Lipid-dependent Activation of the Carcinogen N-2-Fluorenylacetamide in Rats as Monitored by *Salmonella typhimurium*.** (Eng) Castro, C. E. (Dept. Food and Nutrition, Texas Tech Univ., Lubbock, TX, 79409); Felkner, I. C.; Yang, S. P. *Cancer Res* 38(9): 2836-2841; 1978.

Salmonella typhimurium TA1538 was used to quantify the ability of tissue homogenate supernatants to activate the carcinogen N-2-fluorenylacetamide (FAA) and the proximate carcinogen N-hydroxy-2-fluorenylacetamide (N-OH-FAA). The supernatants were obtained from male Sprague-Dawley rats fed a basal diet supplemented with 5% or 20% beef tallow (bt) or corn oil (co) for 6 mo and given ip injections of FAA (10 mg/100 g) or of the control vehicle. During the feeding period, animals were given seven injections, the first at the end of week 2 and the remaining at weekly intervals during weeks 7-12. After only 21 days of feeding and one injection of FAA or control vehicle, elevated activity of the liver microsome-free supernatant (S-150) enzyme that converts N-OH-FAA to mutagen was dependent upon the kind and level of dietary fat. Enzyme activity from FAA-treated rats fed 5% co was three times higher than it was in FAA-treated animals fed 5% bt, whereas in FAA-treated animals fed 20% co, enzyme activity was two times higher than it was in rats fed 20% bt. After 6 mo, the mutagen-producing potential of S-150 was increased, regardless of the diet. However, FAA injections plus 20% co or bt promoted the greatest enzyme activity, suggesting a synergistic effect of high fat and FAA. The ability of S-150 to activate potentially mutagenic compounds was 22-fold higher in FAA-treated rats fed 20% co than in FAA-treated animals fed 20% bt. Between 21 days and 6 mo, hepatic enzyme activity increased 2-fold for rats fed 5% bt or co, 5-fold for rats fed 20% bt, and 52-fold for animals fed 20% co. Lung and kidney S-150 activities were assayed with the bacterial system after 6 mo. Dietary fat did not affect the lung activity, and although kidney enzyme activity was increased from one- to threefold, it was less responsive to diet than liver. After 21 days and one FAA injection, rat had a greater concentration of urinary N-OH-FAA glucuronide when fed co than when fed bt. After 6 mo, the urine of rats fed 20% co and given seven injections of FAA exhibited the greatest amount of urinary N-OH-FAA. (40 refs)

78-3702 **Effect of Sodium Selenite and Methyl Methanesulfonate or *N*-Hydroxy-2-acetylaminofluorene Co-exposure on Sister-Chromatid Exchange Production in Human Whole Blood Cultures.** (Eng) Ray, J. H. (Medical Genetic Center, Univ. Texas Graduate Sch. Biomedical Sciences, P.O. Box 20334, Astrodome Station, Houston, TX, 77025); Altenburg, L. C.; Jacobs, M. M. *Mutat Res* 57(3): 359-368; 1978.

The effect of sodium selenite (Na_2SeO_3), alone and combined with methyl methanesulfonate (MMS) or N-hydroxy-2-acetylaminofluorene (N-OH-AAF), on the frequency of sister chromatid exchanges (SCE's) in human whole blood cultures was studied. Mitotic figures were reduced or eliminated by exposure to 3.95×10^{-6} or 7.90×10^{-6} M Na_2SeO_3 for 96 hr, but exposure to 1.19×10^{-5} M for 19 hr did not alter the mitotic index. The higher Na_2SeO_3 concentrations resulted in overall SCE frequencies of 20.71 and 21.51, respectively; these frequencies were significantly elevated compared with background frequencies (6-7 SCE's/cell). Approx 30 SCE's/cell were found in cells exposed to 1×10^{-4} M MMS for the final 19 hr of culture. In cultures exposed to Na_2SeO_3 plus MMS, decreasing MMS:Na_2SeO_3 ratios resulted in SCE frequencies that were 25%-30% below the sum of the SCE frequencies produced by the individual compounds. Exposure of lymphocytes to 2.7×10^{-5} N-OH-AAF resulted in 13.61 SCEs/cell, and exposure to Na_2SeO_3 plus N-OH-AAF resulted in SCE frequencies produced by the individual compounds. (39 refs)

78-3703 **Bladder Carcinogenesis Using Bilharzia-infested Swiss Albino Mice.** (Eng) El-Aaser, A. A. (Dept. Cancer Biology, Cancer Inst., Cairo Univ., Cairo, Egypt); Hassanein, S. M.; El-Bolkainy, M. N.; Omar, S.; El-Sebai, I.; El-Merzabani, M. M. *Eur J Cancer* 14(6): 645-648; 1978.

The role of liver injury and mechanical irritation of the bladder mucosa in the production of bladder cancer was studied in female Swiss albino mice. Liver injury was induced by infecting the mice with *Schistosoma mansoni* (bilharzia, B) or by feeding them 2-naphthylamine (2-NA, 1%) or 2-acetylaminofluorene (2-AAF, 0.06%) in their diet. One group of mice was given indole (1%) in the diet. Bladder irritation was induced by inserting glass beads into the lumen of the bladder to mimic the passage of ova. Liver injury, bladder irritation, and indole treatment were given alone or in various combinations. Mice with liver injury induced by B and/or the hepatocarcinogens did not develop bladder tumors. Furthermore, mice receiving indole alone or with B infection did not show any change in the bladder mucosa. However, the insertion of glass beads into the bladder induced carcinomas in 8/30 mice by 70 wk. This tumor induction was enhanced by 2-AAF (10/25 mice), but not by B infection (5/20), 2-NA (5/18), or indole (6/22). All urinary bladders containing the beads showed evidence of bacterial infection (*Escherichia coli* and gram-positive cocci) and epithelial hyperplasia and metaplasia. (17 refs)

78-3704 **Sequential Alkaline Phosphatase Changes in Hyperplastic and Neoplastic Urothelium of Mice Fed 2-Acetylaminofluorene.** (Eng) Highman, B. (Div. Pathology Services, Natl. Center Toxicological Res., Jefferson, AR, 72079); Frith, C. H.; Littlefield, N. A. *Environ Pathol Toxicol* 1(3): 223-231; 1978.

Histologic and enzymatic changes in the urinary bladders of 430 BALB/c mice fed a stock diet containing 0, 100, or 500 ppm of 2-acetylaminofluorene (2-AAF) were studied. After 12 wk of receiving 500 ppm 2-AAF, nearly all mice showed bladder epithelial hyperplasia with an epithelial decrease and a stromal increase in alkaline phosphatase (AP). There were frequently many cytoplasmic vacuoles, with eosinophilic inclusions often staining like AP, in the superficial polypoid and upper intermediate epithelial cell layers. After 2-AAF withdrawal at 12 wk, these changes decreased, but 1/4 female mice and 2/4 male mice sacrificed at 51 wk had papillary transitional cell carcinomas. Carcinomas were found in all mice receiving 500 ppm 2-AAF continuously for 51 wk or longer. Mice on 100 ppm 2-AAF developed similar changes more slowly. At 57 wk, epithelial changes were moderate or were marked in nearly all males,and they decreased only slightly after the withdrawal of 2-AAF at 54-57 wk. In females, cytoplasmic vacuolation and other changes were milder,and they virtually disappeared at 67 wk (13 wk after withdrawal of 2-AAF). At 57-103 wk,no females on 100 ppm 2-AAF developed carcinomas, whereas nearly all males, even after the withdrawal of 2-AAF at 54-57 wk, developed papillary carcinomas. There appeared to be some correlation between the severity and persistence of cytologic damage, evidenced by vacuoles with inclusions, in a given treatment group and the later development of carcinomas in that group. Invasive tumors were found only after prolonged administration of 500 ppm 2-AAF, whereas papillary tumors were generally found in mice given 500 ppm 2-AAF for only the first 12 wk or in those given 100 ppm 2-AAF continuously or for a limited period. Invasive tumors generally lacked or showed relatively little AP and showed no vacuoles or inclusions, whereas papillary carcinomas often contained abundant AP, suggesting a possible correlation between the degree of invasiveness or malignancy and the degree of enzymatic and morphologic dedifferentiation. (6 refs)

78-3705 **Distribution of α-Fetoprotein- and Albumin-containing Cells in the Livers of Fischer Rats Fed Four Cycles of N-2-Fluorenylacetamide.** (Eng) Sell, S. (Dept. Pathology, Univ. California at San Diego, La Jolla, CA, 92093). *Cancer Res* 38(9): 3107-3113; 1978.

The immunofluorescent localization of α-fetoprotein (AFP)- and albumin-containing cells was determined in the livers of Fischer rats fed 0.05% N-2-acetylfluorenamide for four 2-wk-on, 1-wk-off cycles. After this exposure, multiple changes in the liver included > 1,000 neoplastic nodules/liver, as well as extensive production of oval cells and focal zones of atypical hepatocellular hyperplasia. Approx 1% of the oval cells contained AFP and about half of the zones of atypical hyperplasia included cells that contained AFP, but none of the neoplastic nodules or normal hepatocytes had any AFP-containing cells. Since up to 60% of the hepatocellular carcinomas developing from this regimen will predictably produce AFP, it is tentatively concluded that hepatocellular carcinoma may arise not only from premalignant neoplastic nodules but also from oval cells or the atypical differentiation of hepatocytes. (32 refs)

78-3706 **Disturbances in the Lipid Regulation of Hepatic Microsomal Enzyme Activity During Chemical Carcinogenesis.** (Rus) Burlakova, E. B. (Inst. Chemical Physics, Moscow, USSR); Dzhaliabova, M. I.; Kobliakov, V. A.; Koliada, A. Iu.; Molochkina, E. M. *Dokl Akad Nauk SSSR* 241(2): 477-480; 1978.

To evaluate the effect of chemical carcinogenesis on the regulation of cellular metabolism, random-bred mice were fed a diet containing 2-acetylaminofluorene (2-AAF) or diethylnitrosamine (DENA). Rats treated with 2-AAF were sacrificed after 4-9 wk (first liver tumors appeared during week 12) and rats treated with DENA were sacrificed after 5-27 wk) (first tumors appeared during week 26). The intensity of the free-radical oxidation in the microsomal lipids was measured by the antioxidative activity (AOA) of the lipids. In control rats, the increase in AOA was correlated with a decrease in phosphatidylcholine (PC) and an increase in phosphatidylethanolamine (PEA), but in rats exposed to the carcinogens, the increase in AOA was associated with an increase in PC and a decrease in PEA. (13 refs)

78-3707 **Cotton Rat Anomaly.** (Eng) McGregor, D. B. (Inveresk Res. International, Inveresk Gate, Edinburgh, Scotland). *Nature* 274(5666): 21; 1978.

2-Acetylaminofluorene (AAF) is more efficiently activated to a mutagen detectable by *Salmonella typhimurium* TA1538 if the postmitochondrial supernatant (S-9) is derived from cotton rat liver rather than Wistar rat liver. However, carcinogenicity tests show that AAF may be noncarcinogenic in cotton rats, but it is a potent carcinogen in rats. Although induced liver enzymes may give different results, mutation frequencies obtained with induced liver S-9 are concluded to be less important to quantitative correlations between mutagenesis and carcinogenesis than results with normal liver S-9. (3 refs)

78-3708 **Effects of Chronic Feeding of 2-Acetylaminofluorene on Nuclear Populations in Rat Liver.** (Eng) Irving, C. C. (Cancer Res. Lab., Veterans Admin. Hosp., Memphis, TN, 38104); Roszell, J. A.; Fredi, J. L. *Adv Enzyme Regul* 16: 365-377; 1978.

The effects of chronic 2-acetylaminofluorene (2-AAF) feeding on the liver nuclei of male and female Sprague-Dawley rats were investigated. Male rats were fed 0.04% 2-AAF in the diet and killed at weekly intervals for 6 wk. Liver nuclei were separated into two fractions: one sedimenting through 2.3 M sucrose (class N-I) and another sedimenting through 1.6 M but not 2.3 M sucrose (N-II). During 2-AAF administration, the percentage of DNA found in N-I nuclei decreased from the control value of 86% to 68% by 2 wk and to 1.4% by 3 wk; it then fluctuated between 10% and 40% at 4-6 wk. Corresponding increases were noted in the amounts of DNA recovered in N-II nuclei. These changes occurred in the absence of any appreciable change in net liver DNA content. The ploidy of the nuclei in the fractions was determined by examining nuclei pelleting in 2.3 M sucrose (H nuclei) and nuclei sedimenting through 1.6 and 1.8 M sucrose and banding at the 1.8-2.3 M interface (L nuclei). In normal adult rat liver, the H and L fractions appeared equivalent to the N-I and N-II nuclei, respectively, but in younger animals, the L fraction predominated. The percentage of L nuclei decreased as the animals grew. Both H and L nuclei contained diploid, tetraploid, and octaploid nuclei, but the degree of ploidy was greater in the H fraction. As the percentage of L nuclei decreased with age, the degree of polyploidy in the H fraction increased, suggesting that the shift of nuclei from the L to the H fraction was associated with hepatocyte maturation. Male and female rats were fed 2-AAF and killed at weekly intervals for 12 wk. By 6 wk, the amount of nuclei in the H and L fractions had changed from 75% and 25%, respectively, to 3% and 97%, respectively. The degree of polyploidy in the livers of male rats decreased during the initial 6 wk from 40%-50% to 20%. Alterations in the nuclear populations in the livers of female rats (which are resistant to 2-AAF-induced cancer) were minimal. (25 refs)

78-3709 **Changes in Monoamine Oxidase, Adenylate Cyclase, and Adenosine Triphosphatase Activity in Rat Liver Mitochondria During Hepatocarcinogenesis.** (Rus) Gurkalo, V. K. (N. N. Petrov Res. Inst. Oncology, Leningrad, USSR); Tret'iakov, A. V. *Vopr Med Khim* 24(4): 495-499; 1978.

Changes in liver mitochondria monoamine oxidase (MAO), adenylate cyclase (AC), and ATPase activity were studied during 2-acetylaminofluorene (AAF)- or diethylnitrosamine (DENA)-induced hepatocarcinogenesis in random-bred albino rats. Three histological stages of hepatocarcinogenesis were identified: Stage 1 was characterized by structural rearrangements in the liver tissue (proliferation of embryonal precursors of biliary cells after exposure to AAF and proliferation of atypical hepatocytes after exposure to DENA), Stage 2 by the development of benign tumors, and Stage 3 by the development of cholangiocarcinomas (after AAF) or hepatocellular carcinomas (after DENA). During AAF-induced carcinogenesis, there was trace ATPase activity during Stage I, but during Stage 2, ATPase activity was 50% of the normal value; MAO activity increased markedly from Stage 1 to Stage 3. During DENA-induced carcinogenesis, AC activity remained unchanged during Stage 1, doubled in Stage 2, and showed a progressive decrease (up to 40%-50% of the normal value) during Stage 3. (10 refs)

78-3710 **Quantitative Kinetics of Development of N-2-Fluorenylacetamide-induced, Altered (Hyperplastic) Hepatocellular Foci Resistant to Iron Accumulation and of Their Reversion or Persistence Following Removal of Carcinogen.** (Eng) Williams, G. M. (Naylor Dana Inst. Disease Prevention, American Health Foundation, 1 Dana Road, Valhalla, NY, 10595); Watanabe, K. *J Natl Cancer Inst* 61(1): 113-121; 1978.

The kinetics of development of N-2-fluorenylacetamide (FAA)-induced, iron-free altered (hyperplastic) hepatocellular foci in inbred F344 rats was studied. Iron deposition was induced by feeding the animals a diet (diet 1) containing 0.8% 8-hydroxyquinoline (HQ) and 2.19% ferrous gluconate (FG) for 3 mo. The rats were then fed a diet 1 supplemented with 0.02% FAA for 13 wk, after which they were returned to diet 1. Groups of rats were killed at weekly intervals during the 13-wk carcinogen-feeding period and at 4, 12, and 24 wk after discontinuation of FAA. The absence of iron storage by altered cells was a sensitive marker that permitted the identification of extremely small lesions within 3 wk of carcinogen treatment. These lesions grew in size and number until 13 wk, by which time all lesions corresponded to one of the types of recognized, altered (hyperplastic) foci. They also displayed abnormal glycogen storage. After FAA was discontinued, the foci disappeared by a reversion back to iron storage while temporarily retaining the nuclear abnormalities of altered cells. More than 95% of altered foci underwent reversion by 6 mo following removal of FAA. More advanced lesions developed in the lobes that displayed the greatest incidence and persistence of foci. The behavior of foci at the stage delineated in this study corresponds closely to previously made generalizations for precancerous lesions. (32 refs)

78-3711 **Influence of DDT on the Hepatotoxicity of 2-Acetylaminofluorene in Wistar Rats.** (Pol) Syrowatka, T. (Zaklad Toksykologii Sanitarnej, Panstwowy Zaklad Higieny, ul. Chocimska 24, 00-971 Warsaw, Poland); Jurek, A.; Nazarewicz, T. *Rocz Panstw Zakl Hig* 29(1): 57-64; 1978.

The effect of the simultaneous administration of 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT) and 2-acetylaminofluorene (2-AAF) on the intensity of neoplastic changes in the liver of Wistar rats was determined. In a 9-mo experiment in which animals were given 2-AAF and/or DDT by gastric intubation, no neoplastic changes were noted in the liver. Two groups of animals were then fed 2-AAF (300 mg/kg diet or 2-AAF + DDT (50 mg/kg diet) for 3 mo and then maintained on a standard diet for 10 mo. Histological examination of the liver, kidneys, spleen, and lungs showed neoplastic changes in both groups. However, a much higher mortality was observed among the animals receiving 2-AAF only. The intensity of the neoplastic changes was slightly higher in all the 2-AAF-treated rats, and animals in this group had metastases to various organs. These findings indicate that DDT, at the dose administered, does not augment 2-AAF-induced carcinogenesis, but rather appears to reduce it. (14 refs)

78-3712 **Sacrococcygeal Teratoma in a Neonate. Association with Maternal Use of Acetazolamide.** (Eng) Worsham, G. F. (Dept. Lab. Medicine, Natl. Naval Medical Center, Bethesda, MD, 20014); Beckman, E. N.; Mitchell, E. H. *JAMA* 240(3): 251-252; 1978.

The occurrence of a sacrococcygeal teratoma in an infant born to a mother treated with acetazolamide (AAA) is reported. The mother (22-yr-old) was admitted at 27 wk gestation with spontaneous rupture of the membranes. She had been treated with AAA (750 mg/day) since the age of 13 for glaucoma. AAA therapy was discontinued at 19 wk gestation. The infant had a large tumor in the sacrococcygeal area that was a multilobulated, irregular mass of solid elements: tissue resembling brain, opalescent gray-white cartilaginous tissue, loops of small bowel, mineralized foci, and clotted blood. Histologically, the tumor was composed of tissue from all three germ layers, with a preponderance of neuroectodermal derivatives. Several congenital abnormalities, but not teratomas, have been associated with the use of AAA in gestating experimental animals. This is the third known case of infant morbidity associated with this drug. (5 refs)

78-3713 **In Vivo Somatic Mutation Systems in the Mouse (Meeting Abstract).** (Eng) Russell, L. B. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830). *Mutat Res* 53(2): 255; 1978. (no refs)

78-3714 **Mutagenicity Screening of Feed Additives in Microbial Systems (Meeting Abstract).** (Eng) Shirasu, Y. (Inst. Environmental Toxicology, Kodaira, Tokyo 187, Japan); Moriya, M.; Kato, K. *Mutat Res* 53(2): 261; 1978. (no refs)

78-3715 **Diphenylhydantoin as an Inducer for the Salmonella Mutation Assay.** (Jpn) Shimizu, H. (Dept. Public Health, Jikei Univ. Sch. Medicine, Tokyo 105, Japan); Takemura, N.; Hayashi, K. *Jpn J Ind Health* 19(4): 202-203; 1977.

The effectiveness of diphenylhydantoin (DPH) as an inducer of Sprague-Dawley rat liver microsome drug-metabolizing enzymes for the Salmonella mutation assay was compared with that of polychlorinated biphenyl (PCB), phenobarbital (PB), and 3-methylcholanthrene (3-MC). The order of effectivenss of PCB $>$ PB, DPH $>$ 3-MC. (7 refs)

78-3716 **Is Phenobarbital Carcinogenic? A Follow-up of 8078 Epileptics.** (Eng) Clemmesen, J. (Danish Cancer Registry, Natl. Anti-Cancer League, Strandboulevard 49, Copenhagen 2100, Denmark); Hjalgrim-Jensen, S. *Ecotoxicology and Environmental Safety* 1(4): 457-470; 1978.

A total of 4,268 men and 3,810 women admitted to a Danish epilepsy center from 1933 to 1962 were followed to December 1972 or to their deaths by population files and a nationwide cancer registry to determine if treatment with the anticonvulsant phenobarbital (PB) increases the risk of cancer. PB was given in individual doses of 100-300 mg, mainly in the evening. Phenylhydantoin was given in daytime doses of 100-400 mg, and primidone was administered in doses of 0.5-1.5 g. Separate tables of data were constructed for patients observed $<$ 10 yr and $>$ 10 yr. Besides the expected excess of brain tumors, 11 cases of liver cancer occurred among patients observed $>$ 10 yr vs an expectant number of 2.1. However, eight of these patients had received thorium dioxide for angiography. One of the eight also developed leukemia. There was no excess of leukemia or lymphoma or cancer at any other sites. (37 refs)

78-3717 **Isolation and Structure Studies of Mutagenic Principles in Amino Acid Pyrolysates.** (Eng) Kosuge, T. (Shizuoka Coll. Pharmacy, Shizuoka, Japan); Tsuji, K.; Wakabayashi, K.; Okamoto, T.; Shudo, K.; Iitaka, Y.; Itai, A.; Sugimura, T.; Kawachi, T.; Nagao, M.; Seino, Y. *Chem Pharm Bull (Tokyo)* 26(2): 611-619; 1978.

The mutagenicity of the pyrolysis products of various amino acids was investigated using *Salmonella typhimurium* strains TA98 and TA100. The basic fractions from pyrolysate of DL-tryptophan, L-ornithine, L-glutamic acid, L-serine, L-lysine, creatine, and creatinine were mutagenic toward TA98. Two active agents in the tryptophan pyrolysate were 3-amino-1,4-dimethyl-5H-pyrido-[4,3-b]indole and 3-amino-1-methyl-5H-pyrido[4,3-b]indole. 2-Amino-5-phenylpyridine, benzo[f]quinoline, and phenanthridine were among the mutagenic antimicrobial tar constituents. (11 refs)

78-3718 **Mutagenic and Recombinogenic Effects of the Antitumor Antibiotic Anthramycin.** (Eng) Hannan, M. A. (Coll. Pharmacy, Univ. Kentucky, Lexington, KY, 40506); Hurley, L. H.; Gairola, C. *Cancer Res* 38(9): 2795-2799; 1978.

Anthramycin (AM), a pyrrolo(1,4)benzodiazepine antibiotic with potent antitumor activity, was tested for its effects on histidine reversion in *Salmonella typhimurium* and on forward and reverse mutations, mitotic crossing-over, and gene conversion in *Saccharomyces cerevisiae.* AM was not mutagenic in *S. typhimurium*; it was antimutagenic in three *S. cerevisiae* strains and mutagenic in one. However, AM induced mitotic crossing-over, mitotic gene conversion, and, possibly, other chromosomal alterations in a diploid strain of *S. cerevisiae.* These studies emphasize the need for a battery of test systems, including eukaryotic organisms, to detect the genetic activity of certain antitumor drugs. The importance of considering data that distinguish between highly and weakly mutagenic cancer chemotherapeutic agents is also discussed. (23 refs)

78-3719 **Potential Carcinogens Tested by Induction and Mutagenesis of Prophage λ in *Escherichia coli* K12.** (Eng) Moreau, P. (Section de Radiobiologie, Laboratoire d'Enzymologie, C.N.R.S., 91190 Gif-sur-Yvette, France); Devoret, R. *In: Human Risk Assessment. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4 (Book C), 583 pp., 1451-1472; 1977.

A test for possible carcinogens based upon lysogenic induction, ie, the induction of prophage λ in *Escherichia coli*, is described. The test is designed so that mutagenesis of prophage λ can be scored independently of lysogenic induction. Thus, it can be determined whether a chemical induces mutagenesis, lysogenic induction, or both. It is felt that a chemical that induces both is a potential carcinogen. Treated λ lysogens in the latent period are plated onto GT-amp plates with 2.5-3 ml of soft agar and with 0.2 ml of indicator bacteria GY4015 at about 2 x 10^9 cells/ml. The plates are incubated overnight at 37 C; ampicillin is added to the GT medium because this antibiotic prevents the noninduced bacteria from multiplying and releasing phage during the overnight incubation. However, ampicillin does not inhibit the induced lysogens from forming infective centers. The efficiency of lysogenic induction is calculated as the ratio of the number of induced infective centers to the number of original viable lysogens. Three variations of this simple, efficient, and inexpensive test are described: a qualitative spot test, a quantitative test on plates, and a quantitative test in liquid medium. The test is as sensitive as mutagenicity tests, and a good correlation was found between the carcinogenic activity of chemicals and their inducing activity. (56 refs)

78-3720 **Comparative Mutagenicity and Toxic Effects of 2,5,2′,5′,-Tetrachlorobiphenyl and Its Metabolites in Bacterial and Mammalian Test Systems.** (Eng) Hsia, M. T. (Dept. Entomology, Univ. Wisconsin, Madison, WI, 53706); Lin, F. S.; Allen, J. R. *Res Commun Chem Pathol Pharmacol* 21(3): 485-486; 1978.

The possible toxicological properties of 2,5,2′,5′-tetrachlorobiphenyl (TCB), 4-hydroxy-2,5,2′,5′-tetrachlorobiphenyl (TCB-OH), and 2,5,2′,5′-tetrachlorobiphenyl-3,4-oxide (TCB oxide) were evaluated in three bioassay systems. Incubation of rat hepatocyte plasma membranes with 50 μg/ml TCB-OH decreased the activity of Mg^{2+}-ATPase by 90%, whereas approx half of the enzyme activity remained after TCB or TCB oxide treatment. All three compounds were found inactive in the *Salmonella typhimurium*/microsome mutagenicity assay. Only TCB-OH possessed potent cytotoxic activity against all four *S. typhimurium* strains tested. It affected the viability of strain TA1537 by as much as 40% even at concentrations as low as 1 μg/ml. These data suggest the potential toxicological significance of PCB phenolic metabolites and of metabolic activation by the hepatic microsomal mixed-function oxidases. (24 refs)

78-3721 **Mutagenic Activity of Chemicals Identified in Drinking Water (Meeting Abstract).** (Eng) Simmon, V. F. (Stanford Res. Inst., Menlo Park, CA); Kauhanen, K.; Mortelmans, K.; Tardiff, R. *Mutat Res* 53(2): 262; 1978. (no refs)

78-3722 **Role of Metabolic Activation in Azide Mutagenesis (Meeting Abstract).** (Eng) Nilan, R. A. (Program Genetics and Dept. Agronomy and Soils, Washington State Univ., Pullman, WA, 99164); Kleinhofs, A. *Mutat Res* 53(2): 242; 1978. (no refs)

78-3723 **Efficiency of Short-Term Tests for Evaluating Mutagenicity of Environmental Chemicals.**

(Rus) de Serres, F. J. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC). *Genetika* 14(6): 935-946; 1978.

The effectiveness of the short-term *Salmonella typhimurium* assay for estimating the mutagenicity and, in view of the proposed correlation between mutagenicity and carcinogenicity, the carcinogenicity of various chemicals was evaluated. Of 175 known carcinogens tested, 157 showed mutagenic activity, and of 108 noncarcinogens, only 14 were mutagens. Since the assay may yield false-positive results, tests should also be made in other systems, including eukaryotic cells. (19 refs)

78-3724 **Purification and Characterization of an Endonuclease from *Micrococcus luteus* That Acts on Depurinated and Carcinogen-modified DNA.** (Eng) Hecht, R. (Institut fur Biochemie, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, D-6900 Heidelberg 1, W. Germany); Thielmann, H. W. *Eur J Biochem* 89(2): 607-618; 1978.

An endonuclease (EN) from *Micrococcus luteus* that is capable of recognizing DNA damage induced by methyl methanesulfonate and heat/acid treatment was purified 500-fold and characterized in order to discover which types of DNA damage are handled by which repair enzymes. The purification, achieved by chromatography on phosphocellulose + gel filtration on Sephadex G-50, produced an EN (peak A, or EN-A) that was separate from pyrimidine dimer-specific UV EN (peak B). The mol wt of EN-A was 30,000 daltons. EN-A was not absolutely dependent on divalent cations, but 5-30 mM Mg^{2+} produced a fivefold stimulation of activity. KCl concentrations < 100 mM were optimal, but those > 100 mM inhibited EN-A activity. The enzyme had no effect on native DNA, but it caused single-strand breaks in DNA containing apurinic/apyrimidinic sites produced by heating at an acidic pH. Carcinogen-modified DNA was also susceptible to the action of this enzyme. EN-A activity was detected by making use of the difference in mobility between supercoiled closed-circular DNA of *Pseudomonas* phage PM2 and its nicked form in agarose gel electrophoresis. Even depurinated or carcinogen-modified supercoiled PM2 DNA migrated faster than the respective nicked forms. EN-A introduced approx twice as many breaks into carcinogen-modified PM2 DNA as the number of apurinic sites present. It is concluded that EN-A either recognizes both apurinic sites and DNA bases carrying carcinogenic residues or, more likely, that it contains DNA glycosidase activity in addition to EN activity. Upon exposure to UV-irradiated PM2 DNA, EN-A did not act on pyrimidine dimers but it did act on apyrimidinic sites and on minor photoproducts that may be 5,6-saturated pyrimidine residues. (52 refs)

78-3725 **Comparative Studies of 14 Mutagenic or Carcinogenic Substances in Seven Mutagenicity Test Systems (Point Mutation Tests, Cytogenetic Test and the Dominant Lethal Test) (Meeting Abstract).** (Eng) Muller, D. (Res. Dept., Pharmaceuticals Div., Ciba-Geigy Ltd., Basel, Switzerland); Arni, P.; Fritz, H.; Langauer, M.; Strasser, F. F. *Mutat Res* 53(2): 235; 1978. (no refs)

78-3726 **Crankcase Oils: Are They a Major Mutagenic Burden in the Aquatic Environment?** (Eng) Payne, J. F. (Fisheries and Marine Service, Newfoundland Biological Station, St. John's, A1C 1A1 Newfoundland, Canada); Martins, I.; Rahimtula, A. *Science* 200(4339): 329-330; 1978.

Fractions from used crankcase oil enriched in polyaromatic hydrocarbons (PAH) were mutagenic in *Salmonella typhimurium* strain TA98 when activated by rat or trout liver extracts. The mutagenic activity was not due to benzopyrene or benzanthracene. Fractions from various crude and refined petroleums were nonmutagenic. The results establish that fish can produce mutagenic metabolites from PAH and that used crankcase oils, which are released into the terrestrial and aquatic environment in considerable quantities, may represent a considerable mutagenic threat. (18 refs)

78-3727 **Activation of Carcinogens and Mutagens by Rat Colon Mucosa.** (Eng) Fang, W. F. (Dept. Biochemistry and Molecular Biology, Univ. Texas Medical Sch. at Houston, P.O. Box 20708, Houston, TX, 77025); Strobel, H. W. *Cancer Res* 38(9): 2939-2944; 1978.

Colon mucosal cells can catalyze the activation of precarcinogens to mutagenic metabolites without the intermediacy of intestinal bacteria as, shown in a mutagenesis assay system composed of *Salmonella typhimurium* strain TA100 and the 9,000 x *g* supernatant fraction of rat colon mucosal cells. Pretreatment of rats with β-naphthoflavone increased the activation of 2-aminoanthracene (AA) 10- to 20-fold and the activation of benzo(a)pyrene (BP) 4-fold. Pretreatment of rats with Aroclor 1254 doubled the activation of AA over control levels but had no effect on the activation of BP. The activation of AA and BP by liver was induced significantly by pretreatment with both β-naphthoflavone and Aroclor 1254. Phenobarbital/hydrocortisone pretreatment did not increase the activation by the colon system of any precarcinogen tested, but it did increase the activation of AA, cyclophosphamide, and isophosphamide by the liver system. The activation of precarcinogens in the bacterial test system was directly correlated with the activities of the pretreated colon and liver preparations toward several drug and polycyclic hydrocarbon substrates assayed in vitro. (39 refs)

78-3728 **Metabolism and DNA Binding of Polycyclic Aromatic Hydrocarbons by Human Diploid Fi-**

broblasts. (Eng) Baird, W. M. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA, 19104); Diamond, L. *Int J Cancer* 22(2): 189-195; 1978.

The metabolism of benzo(a)pyrene (BP), 3-methylcholanthrene (3-MC), and 7,12-dimethylbenz(a)anthracene (DMBA) was measured in mid- and late-passage cultures (population-doubling levels of 20-39 and > 55, respectively) of WI-38 human diploid fibroblasts. Cultures re-fed with 30 ml median containing 0.1 nanomole/ml ^{3}H-hydrocarbon for 144 hr metabolized the hydrocarbons to water- and organic solvent-soluble derivatives. Seven-day-old cultures of late-passage cells metabolized similar amounts of BP and 3-MC as mid-passage cells, but only about one-third as much DMBA per cell. The DNA adducts formed from BP were examined by chromatography of enzyme-degraded DNA samples on Sephadex LH20 columns. On columns eluted with methanol-water gradients, the ^{3}H-BP-deoxyribonucleoside adducts eluted in the same volume as the product peak isolated from DNA that had been reacted with 7,8-dihydroxy-9,10-epoxy-7,8,9,10-tetrahydro-BP. On columns eluted with borate-containing methanol-water gradients, 62% of the cellular products eluted in the same volume as the product peak isolated from DNA that had been reacted with (±)-7α,8β-dihydroxy-9β,10β-epoxy-7,8,9,10-tetrahydro-BP (anti-isomer) and 35% eluted in the same volume as the product peak isolated from DNA that had been reacted with (±)-7α,8β-dihydroxy-9α,10α-epoxy-7,8,9,10-tetrahydro-BP (syn- isomer). The fact that the adducts eluted as two peaks on borate-containing gradients suggests that two isomeric BP-diol epoxides are involved in the binding of BP to DNA in WI-38 cells. (36 refs)

78-3729 **Malignant Transformation of a Mouse Epithelial Cell Line with Polycyclic Aromatic Hydrocarbons.** (Eng) Mondal, S. (Los Angeles County-Univ. Southern California, Comprehensive Cancer Care Center, 1303 N. Mission Road, Los Angeles, CA, 90033); Sala, M. *Nature* 274(5669): 370-372; 1978.

A new epithelial cell line, MC 3T3 [developed from wild mouse (*Mus musculus castaneous*) embryos] that can be transformed by polycyclic aromatic hydrocarbons is described. The cells are polygonal, with a large nucleus and prominent nucleolus, and they grow in compact colonies before they reach confluence. The cells were fed with medium containing 3-methylcholanthrene (3-MC) at 5 or 2.5 μg/ml, benzo(a)pyrene (BP) at 2.5 μg/ml, or 7,12-dimethylbenz(a)anthracene (DMBA) at 1 μg/ml. With each passage, piled-up areas of more refractile cells of various sizes increased in size and number in all the carcinogen-treated dishes. Within 3 to 4 wk, all nude mice injected with 3-MC-, BP-, and DMBA-treated cells developed progressively growing local tumors at the site of injection. The tumors were histologically identified as poorly differentiated carcinomas. (14 refs)

78-3730 **Comparison of the Effects of Beeswax:Trioctanoin and Trioctanoin Vehicles on 3-Methylcholanthrene, Benzo(a)pyrene, and 7,12-Dimethylbenz(a)anthracene Subcutaneous Carcinogenesis in Three Strains of Mice and One Hybrid.** (Eng) Whitmire, C. E. (Toxicology Branch, Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NCI, Landow Building., Room 3C25, NIH, Bethesda, MD, 20014); Lopez, A. *J Natl Cancer Inst* 61(4): 1107-1111; 1978.

The relative carcinogenic effects of 3-methylcholanthrene (3-MC), benzo(a)pyrene (BP), and 7,12-dimethylbenz(a)anthracene (DMBA) suspended in trioctanoin and in a 1:1 beeswax:trioctanoin (B:T) vehicle were compared. Sc tumor induction with 37.5, 150, or 600 μg of each compound/0.1 ml vehicle was studied in C3H/Anf Cum, C57BL/6 Cum, DBA/2J, and (C57BL x C3H/Anf)F$_1$ (BC3F$_1$/Cum) mice. Median tumor dose levels were significantly lower when the three carcinogens were suspended in trioctanoin. When B:T was used as a vehicle, the three carcinogens differed in their abilities to be absorbed or solubilized from the vehicle by the three strains of mice and the hybrid. In C3H/Anf mice, BP in B:T failed to produce tumors. In BC3F$_1$ mice, no tumors were produced by 3-MC, BP, or DMBA in B:T. In C57BL/6 mice, no tumors were produced with DMBA or 3-MC in B:T. In DBA/2 mice, no tumors were produced by BP or 3-MC in B:T. These results indicate that the interpretation of tumor induction results obtained with B:T may be related to the conditions of bioassay rather than to the carcinogenic potential of a compound. (12 refs)

78-3731 **Effect of Prostaglandins on Skin Tumorigenesis.** (Eng) Lupulescu, A. (Wayne State Univ., Medical Res. Building, 550 E. Canfield Ave., Detroit, MI, 48201). *Experientia* 34(6): 785-788; 1978.

The effect of prostaglandins E$_2$ (PGE$_2$) and F$_2\alpha$ (PGF$_2\alpha$) on skin tumors induced by 3-methylcholanthrene (3-MC) was studied in male albino Swiss mice. Mice were treated with 2 ml of a 0.4% acetone soln of 3-MC and/or 10 μg PGE$_2$ or 10 μg PGF$_2\alpha$ im 3 x/wk for 2 mo. Multiple and large squamous cell carcinomas occurred in 90% of the 3-MC and PGE$_2$- or PGF$_2\alpha$-treated mice at the site of 3-MC treatment. No tumors were observed in mice treated with 3-MC or PG only. There was a marked increase (15- to 20-fold) in the incorporation of ^{3}H-thymidine followed by ^{3}H-uridine, ^{3}H-proline and ^{3}H-leucine, in homogenized material from tumors of mice treated with 3-MC + PG compared with the incorporation found in mice treated with 3-MC or PG only or in the control mice. Only epidermal cell hyperplasia with papillary projections protruding in the dermis were visible in 3-MC-treated mice. The results indicate that PGF$_2\alpha$ and PGE$_2$ markedly stimulate DNA, RNA, protein synthesis in hyperplastic epidermal cells following 3-MC application. This may explain the enhancement of cutaneous carcinogenesis by PG's and their shortening of the tumor latency period.

hus, PG's may act as cocarcinogens and have important nplications in cutaneous carcinogenesis. (9 refs)

8-3732 **Accelerated Growth of Syngeneic Tumors in Mice Treated with Methylcholanthrene.** (Eng) ill, P. H. (Cancer Biology Program, NCI Frederick Cancer es. Center, Frederick, MD, 21501); Jessup, J. M.; Kripke, 1. L. *Int J Cancer* 21(4): 511-515; 1978.

he occurrence of selective immunosuppression following reatment with methylcholanthrene (MC) was studied in 3H-, AKR, C57BL, and BALB/c mice. MC was given as single sc injection (1 or 0.05 mg), and the mice were examied at various times thereafter for immunologic reactivity nd resistance to syngeneic tumor challenge. MC treatment ad no effect on the ability of C3H- mice to reject H-2-comatible skin allografts and H-2 incompatible tumor allografts. 1C also had no consistent effect on the induction of delayed ypersensitivity to dinitrochlorobenzene, as measured by ootpad challenge in BALB/c mice. Based on tumor growth, ate of tumor regression, and, in many cases, final tumor ncidence, four different syngeneic tumors showed some evilence of preferential growth in MC-treated as opposed to ehicle (trioctanoin)-treated mice. The preferential growth of hese tumors in MC-treated mice was not associated with the resence of primary tumors in the test animals. The results ndicate that the immune response to exogenous antigens loes not always correlate with the ability of the host to deal vith syngeneic tumors. They further suggest that there may e a surveillance system against chemically and physically nduced tumors that is distinct from the immune responses o exogenous antigens. (17 refs)

8-3733 **Enhancement of Carcinogenesis by Prostaglandins in Male Albino Swiss Mice.** (Eng) Lupules- u, A. (323 Medical Res. Building, 550 E. Canfield Ave., etroit, MI, 48201). *J Natl Cancer Inst* 61(1): 97-106; 1978.

he effect of prostaglandins E_2 (PGE_2) and $F_2\alpha$ ($PGF_2\alpha$) on he development of 3-methylcholanthrene (3-MC)-induced kin tumors was studied in male albino Swiss mice. Mice were reated topically with 2 ml of a 0.4% acetone soln of 3-MC x/wk for 2 mo and/or 10 -5m-1g PGE_2 $PGF_2\alpha$ every other ay during the same 2 mo. Multiple and large squamous cell arcinomas occurred in 90% of the 3-MC + PGE_2- or PGF α-treated mice at the site of 3-MC treatment. No tumors vere observed in mice treated with 3-MC or PG only. Radi- isotope studies showed a marked increase (15-to 20-fold) in he incorporation of ^{3}H-thymidine, followed by ^{3}H-uridine, H-proline, and ^{3}H-leucine, in the neoplastic cells of mice reated with 3-MC + PG compared with the incorporation een in mice treated with 3-MC alone, PG alone, or in control nice. Electron microscopy showed the predominance of neo- lastic squamous cells with large nuclei, an increased nu- lear:cytoplasmic ratio, dark and light cells with distinct ultrastructural features, increased polysome and dense granule populations, altered mitochondria, and discontinuities of the basement membrane in mice treated with 3-MC and $PGF_2\alpha$/ or PGE_2. Only hyperplastic and dysplastic cellular changes were seen in the epidermis of 3-MC treated mice. The results indicate that $PGF_2\alpha$ and PGE_2 markedly stimulate DNA, RNA, and protein synthesis in hyperplastic epidermal cells following 3-MC application, which may explain their enhancement of cutaneous carcinogenesis. (14 refs)

78-3734 **Lack of Initiating Activity in Mutagens Which Are Not Carcinogenic.** (Eng) Coombs, M. M. (Dept. Chemistry, Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2A 3PX, England); Bhatt, T. S. *Br J Cancer* 38(1): 148-150; 1978.

Two noncarcinogenic but mutagenic compounds, 15,16-dihydrocyclopenta[a]phenanthren-17-one (I) and 1,2,3,4-tetrahydrochrysen-1-one (IV), two carcinogenic and mutagenic compounds, 15,16-dihydro-11-methylcyclopenta[a]phenanthren-17-one (III) and 1,2,3,4-tetrahydro-11-methylchrysen-1-one (V), and a noncarcinogen-nonmutagen 15,16-dihydro-3-methylcyclopenta[a]phenanthren-17-one (II), were examined for tumor production in mice using croton oil as a promoting agent. All compounds were applied in four equal doses of 100 μg in 40 μl of toluene on 4 consecutive days to the shaved back of albino TO mice. Starting 1 wk after the last application, 20 animals receiving each compound were promoted twice weekly with 10 μl of a 1% volume/volume soln of croton oil in toluene until the end of the experiment. Twenty mice receiving each compound were treated with the toluene vehicle only. Controls were treated with toluene, croton oil, or III only. Neither crotol oil nor toluene by themselves were carcinogenic. Only the known carcinogens (III) and (V) produced skin tumors; promotion increased the tumor incidence and decreased the latent period. All tumors in mice receiving III alone were carcinomas, but those in animals receiving III or V plus croton oil or toluene were both carcinomas and papillomas. Compounds I and IV are capable of metabolic activation to active species that bind to DNA in vitro and cause mutations in bacteria, but in animal cells, some factors must operate to prevent these active metabolites from damaging the genetic material. (7 refs)

78-3735 **Non-phorbol Mouse Skin Tumor Promoters Do Not Mimic Phorbol Myristate Acetate in Its Effects on Chick Embryo Fibroblasts.** (Eng) Driedger, P. E. (Dept. Pharmacology, Harvard Medical Sch., 250 Longwood Ave., Boston, MA, 02115); Blumberg, P. M. *Int J Cancer* 22(1): 63-69; 1978.

Several compounds that promote mouse skin tumors but are chemically unrelated to the phorbol esters were tested for their abilities to cause loss of large external transformation-sensitive (LETS) protein from chick embryo fibroblasts

(CEF) and to stimulate deoxyglucose (DG) transport in the CEF. After 3 days' exposure of the CEF to max nontoxic levels of the seven test agents, only oleic acid (140 μM) decreased LETS protein > 20% (actual value, 50%), as measured by lactoperoxidase-catalyzed surface radioiodiniation. In contrast, phenol (1 mM) increased LETS protein levels 70%. Only anthralin, lauric acid, and limonene increased DG transport. Iodoacetic acid and cantharidin were inactive in both assays. None of the compounds affected CEF LETS levels or DG transport to the extent that the phorbol esters do. Palmitic and stearic acids, which are structurally similar to oleic acid but do not promote mouse skin tumors, were also inactive in both assays. It is suggested that the phorbol ester promoters and the non-phorbol promoters may have different mechanisms of action. (30 refs)

78-3736 **Cocarcinogens of the Diterpene Ester Type from *Croton flavens* L. and Esophageal Cancer in Curacao.** (Eng) Weber, J. (Deutsches Krebsforschungszentrum, Institut fur Biochemie, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany); Hecker, E. *Experientia* 34(6): 679-682; 1978.

Croton flavens L., a plant widely used in Curacao for a variety of purposes (it is chewed, used to make tea, used topically to relieve oral inflammation, etc), was investigated for possible irritants and cocarcinogens. Three highly irritant and tumor-promoting Croton factors (F_1-F_3) and corresponding three cryptic Croton factors (F_1'-F_3') were isolated from the roots of the plant and characterized as novel esters of 16-hydroxy- and 4-deoxy-16-hydroxyphorbol, respectively. The results suggest that tumor promoters of the phorbol ester type, ingested through the widespread and frequent use of *C. flavens*, may be causally related to the well known high rate of esophageal cancer on Curacao. (21 refs)

78-3737 **Tumor Promoters Inhibit Morphological Differentiation in Cultured Mouse Neuroblastoma Cells.** (Eng) Ishii, D. N. (Dept. Pharmacology, Coll. Physicians and Surgeons Columbia Univ., New York, NY, 10032); Fibach, E.; Yamasaki, H.; Weinstein, I. B. *Science* 200(4341): 556-559; 1978.

The effects of 12-O-tetradecanoyl-phorbol-13-acetate (TPA) and other tumor promotors on the morphologic differentiation of murine neuroblastoma cultures were determined. Cells cultured in the presence of 1.7 x 10^{-7} M TPA [100 nanograms (ng)/ml] during serum deprivation had a 64% inhibition of neurite fiber outgrowth. With a concentration of 5 x 10^{-7} M, the response was inhibited by 75%. TPA (100 ng/1 ml) also blocked neurite outgrowth induced by prostaglandin E_1, papaverine, 5-bromo-2′-deoxyuridine, and hemin. Other macrocyclic plant diterpene tumor promotors, such as phorbol-12,13-didecanoate (PDD) and ingenol dibenzoate, were also effective inhibitors of neurite formation; mezerein, a less potent promotor than TPA, was also a fairly activ neurite inhibitor. Other diterpenes such as phorbol and 4-α PDD, which are not active as skin tumor promotors, hac no effect on neurite formation. The inhibition of neurite outgrowth by TPA was reversible, as indicated by an increas in outgrowth after a 2-hr incubation in the absence of serum (34 refs)

78-3738 **Effects of Retinoic Acid and Juvenile Hormon on the Induction of Ornithine Decarboxylas Activity by 12-O-Tetradecanoylphorbol-13-acetate.** (Eng Kensler, T. W. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI, 53706); Verma, A. K.; Boutwell, R. K. Mueller, G. C. *Cancer Res* 38(9): 2896-2899; 1978.

When 10 mM 12-O-tetradecanoylphorbol-13-acetate (TPA was added to phytohemagglutinin-treated bovine lymphocyt cultures, ornithine decarboxylase activity was increased 1 times over that in cultures treated with the lectin alone. Thi synergistic action of TPA could be antagonized by simultaneous addition of the acyclic sesquiterpene, insect juvenile hormone III. Retinoic acid (vitamin A acid), an inhibitor of th tumor-promoting action of TPA in mice, was also an effectiv antagonist but required administration to lectin-activatec lymphocytes 1 hr prior to TPA. These data suggest that metabolic activation of retinoic acid is required in order for th compound to exert its antagonistic action. Comparison of th response in the lymphocytes and mouse skin suggests that th lymphocytes provide an excellent system for studying th molecular processes through which phorbol esters and retinoids influence the growth and differentiation of both norma and premalignant cells. (22 refs)

78-3739 **Rapid Stimulation of Phospholipid Metabolisn in Bovine Lymphocytes by Tumor-promoting Phorbol Esters.** (Eng) Wertz, P. W. (McArdle Lab. Cance Res., Univ. Wisconsin, Madison, WI, 53706); Mueller, G. C *Cancer Res* 38(9): 2900-2904; 1978.

The effects of 12-O-tetradecanoylphorbol-13-acetate (TPA), a highly active tumor-promoting agent and lymphocyt comitogen, on phospholipid (PL) metabolism in bovine lymphocytes were studied. TPA accelerated the incorporation o [methyl-^{3}H]choline chloride ([^{3}H-Me]choline) into the PL's of the lymphocytes. Chromatographic analysis of the PL's from lymphocytes exposed to 10^{-8} M TPA for various intervals in the presence of [^{3}H-Me]choline showed that there was a lag period of about 20 min, after which the rate of labeling of phosphatidylcholine, lysophosphatidylcholine, and sphingomyelin were all enhanced. Structure-activity studie showed thathe ability of phorbol diesters to accelerate [^{3}H-Me]choline incorporation was correlated with their potency as lymphocyte comitogens and as tumor promoters. The response to TPA was insensitive to the inhibition o

RNA and protein synthesis by actinomycin D and cycloheximide, respectively, and to the inhibition of membrane movement by cytochalasin B. Retinoic acid, an antagonist of the tumor-promoting and comitogenic actions of TPA, also blocked the acceleration of [^{3}H-Me]choline incorporation into PL's by TPA. The bovine lymphocyte system may be useful for studies of the molecular mechanisms of action of tumor-promoting phorbol esters and their retinoid antagonists. (18 refs)

78-3740 **Stimulation of Prostaglandin Synthesis by Tumor-promoting Phorbol-12,13-diesters in Canine Kidney (MDCK) Cells. Cycloheximide Inhibits the Stimulated Prostaglandin Synthesis, Deacylation of Lipids, and Morphological Changes.** (Eng) Ohuchi, K. (Dept. Biochemistry, Faculty Pharmaceutical Sciences, Tohoku Univ., Sendai, Japan); Levine, L. *J Biol Chem* 253(13): 4783-4790; 1978.

Normal dog kidney cells (MDCK cells: 2×10^5 cells/dish) were incubated with 12-O-tetradecanolyphorbol-13-acetate [TPA: 0.1-10 nanograms (ng)/ml] phorbol-12,13-didecanoate (PDD: 1 ng/ml), or 4α-phorbol-12,13-didecanoate (4α-PDD: 100 ng/ml) for 60 min in 4 ml of non-serum-supplemented medium. The cells were then washed with minimal essential medium and incubated with 4 ml of serum-supplemented medium for various periods of time. TPA and PDD, but not 4α-PDD, stimulated the deacylation of cellular lipids, prostaglandin (PG) biosynthesis, and morphological changes in the MDCK cells. The levels of PGE2 and PGF2 increased with time of incubation. Stimulation of PG production was a slow process, and at low concentrations of promoters the reaction(s) was not complete even after 24 hr. There was a pronounced lag time, the length of which depended on promoter concentration. Cells treated with TPA and PDD had long, thin processes and they appeared longer and thinner than normal cells. They seemed to have much less contact with neighboring cells, and giant cells with multiple processes were seen occasionally. These morphological changes also required at least 24 hr for expression. Cycloheximide (0.005-1.0 μg/ml) inhibited the stimulated PG biosynthesis, the changes in morphology, and the increased lipid deacylation, but hydrocortisone (1.0 μg/ml) did not. Indomethacin (0.5 μg/ml) inhibited the PG biosynthesis completely and some of the lipid deacylation, but it did not affect the morphological changes. (31 refs)

78-3741 **Inflammatory, Tumor Initiating and Promoting Activities of Polycyclic Aromatic Hydrocarbons and Diterpene Esters in Mouse Skin as Compared with Their Prostaglandin Releasing Potency In Vitro.** (Eng) Brune, K. (Dept. Pharmacology, Biozentrum der Universitat, CH-4056 Basel, Switzerland); Kalin, H.; Schmidt, R.; Hecker, E. *Cancer Lett* 4(6): 333-342; 1978.

Release of prostaglandin E_2 (PGE_2) from cultured peritoneal macrophages of NMRI mice by skin irritant tumor initiators and promoters was investigated. 7,12-Dimethylbenz(a)anthracene (DMBA) caused slight irritation on the mouse ear, but even relatively high doses (10^{-6})did not stimulate PGE_2 release to any measurable extent within 4 hr after administration. Apparently, there is no correlation between the irritating and initiating activity in mouse skin and PGE_2 release from macrophages. On the other hand, promoters of the diterpene ester type, eg, 12-O-tetradecanoylphorbol-13-acetate (TPA), were strong irritants on the mouse ear and, even at low doses (10^{-7} and 10^{-8} M), they stimulated PGE_2 release from macrophages dramatically within 1 hr after administration. Moreover, a good correlation was established between irritating and promoting activity in mouse skin and PGE_2 release from macrophages for a series of tigliane-, ingenane- and daphnane-type diterpene derivatives. The results suggest that the rapid and specific release of PGE_2 from macrophages by the diterpene esters may also take place in the epidermal cells of the target tissue. This event could initiate both skin irritation and cell proliferation. (21 refs)

78-3742 **Effects of Multiple Phorbol Myristate Acetate Treatments on Cyclic Nucleotide Levels in Mouse Epidermis.** (Eng) Garte, S. J. (Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY, 10016); Belman, S. *Biochem Biophys Res Commun* 84(2): 489-494; 1978.

The effects of repeated applications of phorbol myristate acetate (PMA) on cyclic nucleotide levels and ratios in mouse epidermis were studied with the use of 6- to 8-wk old female Swiss Ha/ICR mice. A tumor-initiating dose of 25 μg 7,12-dimethylbenz(a)anthracene was applied to the shaved skin of the back of each mouse. One week later, the first dose of 10 μg PMA was applied. PMA treatments were repeated 3x/wk, and animals received 1, 2, 10, or 20 applications. Animals were sacrificed 2, 36, 72, or 120 hr after the last PMA application. Filtered homogenates of epidermis or tumors from the treated skin area were assayed for cyclic AMP (cAMP), cyclic 3′,5′-guanosine monophosphate (cGMP), and DNA. There were slight but significant ($p < 0.05$) decreases in cAMP and dramatic (5- to 10-fold) increases in cGMP after multiple PMA treatments. Increases were greater at 120 hr after the last treatment than at 2 hr. After two more treatments, the cyclic nucleotide levels and the ratios of AMP and GMP to DNA in the isolated solid tumors were very close to those found in the epidermis. (15 refs)

78-3743 **Effects of 7,8-Benzoflavone on Skin Tumor-initiating Activities of Various 7- and 12-Substituted Derivatives of 7,12-Dimethylbenz(a)anthracene in Mice.** (Eng) DiGiovanni, J. (Dept. Pharmacology, Univ. Washington Sch. Medicine, Seattle, WA, 98105);

Slaga, T. J.; Viaje, A.; Berry, D. L.; Harvey, R. G.; Juchau, M. R. *J Natl Cancer Inst* 61(1): 135-140; 1978.

The skin tumor-initiating activities of various 7- and 12-substituted derivatives of 7,12-dimethylbenz(a)anthracene (DMBA) were investigated in female outbred Charles River CD-1 mice. 7-Formyl-12-methylbenz(a)anthracene (7-CHO-12-MBA), at a dose 74-fold greater than that of DMBA [740 nanomoles (nmol)/mouse], was as effective tumor initiator. 7,8-Benzoflavone (7,8-BF) inhibited the tumor-initiating activity of 7-CHO-12-MBA by 51%. 12-Formyl -7- methylbenz(a)anthracene and 7,12-diformylbenz(a)anthracene, applied at initiating doses of 740 and 704 nmol/mouse, respectively, were much less active than 7-CHO-12-MBA. 7-Bromomethyl-12-methylbenz(a)anthracene (7-BRME-12-MBA) was a more effective tumor initiator than 7-bromomethylbenz(a)anthracene (7-BRMEBA), but both were less active than DMBA. 7,8-BF inhibited the tumor-initiating activity of 7-BRME-12-MBA and 7-BRMEBA by 34% and 54%, respectively. 12-Bromomethyl-7-methylbenz(a)anthracene (12-BRME-7-MBA) was as active an initiator as 7-BRME-12-MBA. 7,8-BF inhibited tumor initiation with 12-BRME-7-MBA by 29%. Three naturally occurring flavones and the cytochrome P_{450} inhibitor 1-benzylimidazole were investigated after topical application (100 μg) for their effects on tumor initiation with DMBA. Quercetin inhibited tumor initiation with DMBA by 22%, but myricetin and 4′,5,7-trihydroxyflavonone enhanced tumor initiation by 54% and 29%, respectively. 1-Benzylimidazole had no effect. The abilities of the flavones and 1-benzylimidazole to inhibit aryl hydrocarbon hydroxylase (AHH) in vitro did not correlate with their effects on tumor initiation with DMBA. This suggests that AHH is not the only enzyme system involved in DMBA tumorigenesis and that 7,8-BF may produce inhibition by affecting some other system. (39 refs)

78-3744 **Quantitative Histological Changes During Early Experimental Carcinogenesis in the Hamster Cheek Pouch.** (Eng) Eveson, J. W. (Dept. Oral Medicine and Pathology, Glasgow Dental Hosp., Glasgow, Scotland); MacDonald, D. G. *Br J Dermatol* 98(6): 639-644; 1978.

Fifteen male Syrian golden hamsters had their cheek pouches painted with 9,10-dimethyl-1,2-benzanthracene (DMBA: 0.25% soln in acetone) 3x/wk for a max of 6 wk. Groups of five animals were sacrificed after 3 wk of painting, 6 wk of painting, and 3 wk after the last application. Early sequential changes in the cheek pouch epithelium were quantified. At 3 wk, the epithelium was in the inflammatory phase and was twice the normal thickness, the stratum corneum was three times the normal thickness. The increased size of the cellular compartments was due to increases in the size of the individual progenitor (P) and maturation (M) cells, their numbers actually being reduced. At 6 wk, the epithelium and stratum corneum were thicker, but the inflammatory reaction was less severe. The increased thickness of the cellular compartments was due to a recovery in the number of M cells and an increase in the number and size of the P cells. At 9 wk, overall epithelial thickness was the same, but the size of the keratinized compartment decreased and that of the cellular compartments increased. This suggests that much of the early increase in stratum corneum thickness was due to hyperplastic rather than neoplastic action of the carcinogen. The increased thickness of the cellular layers appeared due to a progressive increase in the size and number of P cells; ie, P cell hyperplasia and hypertrophy. (13 refs)

78-3745 **Protection Against 7,12-Dimethylbenz(a)anthracene-induced Rat Mammary Carcinoma by Infection with Mouse Xenotropic Type C Virus.** (Eng) Weislow, O. S. (Viral Oncology Program, NCI Frederick Cancer Res. Center, Box B, Frederick, MD, 21501); Allen, P. T.; Shepherd, R. E.; Twardzik, D. R.; Fowler, A. K.; Hellman, A. *J Natl Cancer Inst* 61(1): 123-129; 1978.

The possibility that preinfection with a live xenotropic virus may reduce the incidence of chemically induced neoplastic disease was investigated. Female outbred Sprague-Dawley rats were inoculated ip with a viable mouse xenotropic C-type virus, M55 (2 x 10^6 focus-inducing units), 7 days prior to po administration of 7,12-dimethylbenz(a)anthracene (DMBA; 0.1 mg/g). Another group of rats was administered DMBA only. The incidence of tumor formation was 90% in the DMBA-treated rats at day 140. In contrast, tumor incidence in the M55 virus-infected and DMBA-treated group only rose to 35% by day 133 and did not exceed this level. M55 virus inoculation also delayed the onset of tumor formation by 3 wk. Infectious virus could not be isolated from organs of the infected rats, but high titers of circulating and tumor-associated antibodies were detected against the viral internal core protein p30, and a low-grade antibody response to intact virus or envelope glycoprotein was found. A cell-mediated immune response, measured by lymphocyte transformation, was detected with intact virus but not with p30 antigen. No immunity developed after a single inoculation of UV-inactivated virus. It is concluded that infection of a heterologous species with a live, nonpathogenic xenotropic mouse virus protects against DMBA-induced mammary carcinoma. The mechanism of this protection is not known. (62 refs)

78-3746 **Chromosome Changes (Trisomy 15) in Murine T-Cell Leukemia Induced by 7,12-Dimethylbenz(a)anthracene (DMBA).** (Eng) Wiener, F. (Dept. Tumor Biology, Karolinska Institutet, S-104 01 Stockholm 60, Sweden); Spira, J.; Ohno, S.; Haran-Ghera, N.; Klein, G. *Int J Cancer* 22(4): 447-453; 1978.

A series of T-cell leukemias induced in female C57BL/6 mice by 7,12-dimethylbenz(a)anthracene (DMBA: 1 mg/wk x 4, via stomach tube) was studied cytogenetically. After leuemia development, tumor cells were inoculated either iv or p in syngeneic males. Tumors subjected to chromosome anding analysis were from the first, second, or third transplantation generations. The modal number of the leukemia ells fell into three categories: a 41 stemline, a 43 stemline nd a 36 and 37 + 2 two-armed chromosome stemline. All nalyzed leukemia cells had a trisomy of chromosome 15. umor cells with a modal number of 43 displayed additional risomies of chromosomes 19 and 14. These findings suggest hat chromosome 15 trisomy is the first detectable specific hromosome change associated with the development of DMBA-induced T-cell lymphomas. A similar association was previously shown with regard to the development of adiation leukemia virus-induced T-cell lymphomas. These findings support the idea that in tumors of diverse etiologies, ommon cytogenetic changes may appear in the same common target-cell precursor by a convergent microevolution-type process. (20 refs)

78-3747 **Binding of 7,12-Dimethylbenz(a)anthracene to BALB/c Mouse Mammary Gland DNA in Organ Culture.** (Eng) Kundu, A. B. (Tumor Biology Lab., School Life Sciences, Univ. Nebraska, 201 Lyman Hall, Lincoln, NB, 68588); Telang, N. T.; Banerjee, M. R. *J Natl Cancer Inst* 61(2): 465-469; 1978.

Binding of 7,12-dimethylbenz(a)anthracene (DMBA) to BALB/c mouse mammary gland DNA in organ culture was studied, and the ability of DMBA and several other hydrocarbons to induce nodulelike alveolar lesions (NLAL) in the mammary glands in culture was assessed. The second thoracic mammary glands of immature female BALB/c mice were cultivated to alveolar development in a chemically defined medium containing insulin, prolactin, aldosterone, and hydrocortisone (IPAH medium). The glands were treated with labeled (^{3}H or ^{14}C) or unlabeled DMBA for 24 hr between days 3 and 4 of culture. The DNA of glands treated with labeled DMBA contained bound radioactivity. Nearly 100% of the initial radioactivity was retained up to 5 mo, 72% up to 8 mo. Morphological studies after treatment of the glands with equimolar concentrations (0.004 M) of DMBA, dibenz(a,h)anthracene (DBA), benz(a)anthracene (BA), or anthracene in IPAH medium indicated that DMBA was the most potent in inducing NLAL. The incidence of NLAL induced by DBA was about half that induced by DMBA. The NLAL-inducing capacity of BA was even lower, and anthracene showed little effect. Thus, the binding of DMBA to DNA corresponds to its high noduligenic action in the mammary glands in this organ culture model. Similar biochemical interactions between DMBA and cellular DNA are believed to be characteristic of DMBA oncogenesis in other cell types. (31 refs)

78-3748 **Biological Evaluation of Euphorbia Latices in Iran: I. Tumour Producing Effect in Mice.** (Eng) Zarrin, K. (Taj Pahlavi Cancer Inst., Pahlavi Medical Sch., Tehran, Iran); Aynehchi, Y. *Tumor Res (Sapporo)* 12: 47-51; 1977.

The tumor-initiating and -promoting activity of latex derived from eight species of *Euphorbia* was investigated in Iran KZ albino mice. The species included *E. bungi* Boiss., *E. lanta* Sieb., *E. tinctoria* Boiss., *E. virgata* L., *E. microsciadia* Boiss. et Buhse., *E. esula* L., *E. gerardiana* Jacq., and *E. larica* Boiss. In the first experiment, a 10% extract of *Euphorbia* latex in 0.5 ml acetone was applied to the shaved back once weekly for 26 wk. Tumors were noted in mice treated with extracts of *E. tinctoria, E. virgata, E. microsciadia,* and *E. gerardiana.* Papillomas were noted by week 10, and by week 26, 22/76 survivors had papillomas. Ulceration and crusting of the skin were observed in eight animals. All skin lesions eventually healed; no malignant tumors were noted by 40 wk. In a second experiment, mice were treated with either a single dose of 300 μg 9,10-dimethyl-1,2-benzanthracene (DMBA) to the shaved dorsal skin or DMBA plus an *Euphorbia* extract on day 1 and weekly applications of the extract beginning 3 wk later. By 26 wk, 1/79 surviving animals treated with DMBA only had papillomas compared with 60/74 in those treated with DMBA + extract. Papillomas were first noted in the latter group during week 8 of promotion. No malignant tumors were noted by 40 wk. It was concluded that extracts of *Euphorbia* contain a potent tumor-promoting substance. (8 refs)

78-3749 **Effects of Reserpine on the Incidence of 9,10-Dimethyl-1,2-benzanthracene-induced Tumors in Pinealectomised and Thymectomised Rats.** (Eng) Lapin, V. (Inst. Cancer Res., Univ. Vienna, 1090 Vienna IX, Austria). *Oncology* 35(3): 132-135; 1978.

The effects of reserpine administration on the incidence of 9,10-dimethyl-1,2-benzanthracene (DMBA)-induced tumors was investigated in Wistar rats, that had been pinealectomized (PiX) and/or thymectomized (Tx) neonatally. At 3-3.5 mo of age, the rats were given three doses of DMBA (50 mg po) 10 days apart and, 1 mo after the first DMBA dose, some of the rats were also given reserpine (100 μg/kg sc) daily for 40 consecutive days. Another group of rats received reserpine only. In the rats that received DMBA only, there was a 50% tumor occurrence in all animals, with or without surgery. Administration of DMBA + reserpine led to a slight increase in tumor incidence in normal and Tx rats; however, in Pix and Tx-Pix rats, tumor incidence was 100%. None of the animals that received reserpine only suffered any harmful effects. Tumor occurring most frequently were mammary gland and ear duct tumors, both of which are hormone-dependent. Leukemia appeared at a very high rate in Tx-Pix rats after DMBA + reserpine treatment. Mean survival time in these rats was 168.5 days. It is suggested that neuroendocrine alterations in pituitary prolactin secretion due to the neonatal removal of the pineal gland, remain latent and become evident after reserpine administration as reflected in increased tumor incidence. (19 refs)

78-3750 Gangliosides and Their Cell Density-Dependent Changes in Control and Chemically Transformed C3H/10T1/2 Cells. (Eng) Langenbach, R. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NE, 68105); Kennedy, S. *Exp Cell Res* 112(2): 361-372; 1978.

The effect of in vitro chemical transformation on the cellular gangliosides (GS) of C3H/10T1/2 mouse embryo cells was determined, and the possible involvement of GS biosynthesis mechanisms in the density-dependent regulation of cell growth was investigated. The transformants were progeny of line C3H/10T1/2, cloned after treatment with 7,12-dimethylbenz(a)anthracene (DMBA-TCL1 cells) or 3-methylcholanthrene (MCA-TCL15 cells). All three cell types contained a complex pattern of GS, but the transformants had increased levels of the simplest GS (GM_3) and reduced levels of more complex GS. In the C3H/10T1/2 cells, however, as cell-to-cell contact increased, incorporation of ^{14}C-glucosamine into GS showed that GM_3 synthesis was decreased and synthesis of a more complex GS (GD-1a) was increased. In the transformants, the percentage of each individual GS (with respect to total labeled GS) was only slightly altered with changing cell density. As cell density increased, turnover of ^{14}C-glucosamine-labeled GS was approx equal in C3H/10T1/2 cells and MCA-TCL15 cells but more rapid in DMBA-TCL1 cells. Most individual GS turned over at about the same rate except for GD-1a, which increased slightly (relative to percentage of total labeled GS) in all three cell types. Labeling data indicated that the majority of most GD-1a synthesis was de novo. When exogenous complex GS were added to the medium of C3H/10T1/2 and DMBA-TCL1 cells, they inhibited cellular DNA synthesis and caused both lines to incorporate an increased percentage of ^{14}C-glucosamine into GS more complex than GM_3. These and similar findings raise interesting questions about the use of glycolipids for phenotypic reversion of transformed cells, as potential chemotherapeutic agents, and for the immunological detection of transformed cells. (48 refs)

78-3751 Particle-enhanced Membrane Uptake of a Polynuclear Aromatic Hydrocarbon: A Possible Role in Cocarcinogenesis. (Eng) Lakowicz, J. R. (Freshwater Biological Inst., Univ. Minnesota, P.O. Box 100, Navatte, MN, 55392); Englund, F.; Hidmark, A. *J Natl Cancer Inst* 61(4): 1155-1159; 1978.

The effect of a particulate, silica, on the rate of membrane uptake of benz(a)anthracene (BA) was studied. The fluorescence emission spectrum and quantum yield of BA, when adsorbed to silica, underwent spectral shifts upon addition of phospholipid vesicles. These spectral changes appeared to result from the transfer of BA from the silica surface into the lipid bilayer. These spectral changes were used to measure the rates of membrane uptake of BA from the silica-adsorbed state and from the crystalline and microcrystalline states of BA. Adsorption of BA to silica resulted in an increased rate of uptake of BA into dipalmitoyl-L-α-phosphatidylcholine vesicles compared with the uptake rate into these vesicles from crystalline states. This particle-enhanced membrane uptake of chemical carcinogens may be of importance in explaining the enhanced carcinogenicity of polynuclear aromatic hydrocarbons in the presence of particulate matter. (28 refs)

78-3752 Emission of Polycyclic Aromatic Hydrocarbons in the Electrochemical Industry. Accumulation and Effect of Polycyclic Aromatic Hydrocarbons on the Marine Environment. (Fin) Knutzen, J. (NIVA, Finland). *Kjemi* 38(1): 42-43; 1978.

The accumulation and effect on the marine environment of polycyclic aromatic hydrocarbons (PAH) contained in effluents generated in the electrochemical industry are reviewed. PAH have been found in the effluents in concentrations of 0.1-1 ppm. Two PAH, benzanthrone and creosote, caused tumors in red algae and seaweed. (no refs)

78-3753 7,12-Dimethylbenz(a)anthracene-DNA Products in Mammalian Cells and Salmonella Strains (Meeting Abstract). (Eng) Bigger, C. H. (Chemical Carcinogenesis, NCI Frederick Cancer Res. Center, Frederick, MD, 21501); Andrews, A. W.; Moschel, R. C.; Dipple, A. *Mutat Res* 53(2): 152; 1978. (no refs)

78-3754 Microsome-mediated Mutagenicities of the Dihydrodiols of 7,12-Dimethylbenz(a)anthracene: High Mutagenic Activity of the 3,4-Dihydrodiol. (Eng) Malaveille, C. (Unit Chemical Carcinogenesis, International Agency Research Cancer, 150 cours Albert Thomas, 69372 Lyon, France); Bartsch, H.; Tierney, B.; Grover, P. L.; Sims, P. *Biochem Biophys Res Commun* 83(4): 1468-1473; 1978.

7,12-Dimethylbenz(a)anthracene (DMBA) and its 3,4-, 5,6-, 8,9-, and 10,11-dihydrodiols were tested for mutagenicity toward *Salmonella typhimurium* TA100 in the presence of rat liver post-mitochondrial supernatants from Aroclor-treated rats. At substrate concentrations of 5 μM, the non-K-region 3,4-dihydrodiol was the most active of all the compounds tested, being six times more mutagenic than the parent hydrocarbon. At higher concentrations, its mutagenic activity decreased because of cytotoxicity. The other two non-K-region dihydrodiols, the 8,9- and 10,11-dihydrodiols, were less mutagenic than the parent hydrocarbon. The K-region 5,6-dihydrodiol showed weak mutagenic activity at nontoxic concentrations. These results suggest that, of the dihydrodiols tested, the 3,4-dihydrodiol is the one most likely to be involved in the metabolic activation of DMBA. Since this dihydrodiol requires further metabolic activation, one or both of the isomeric forms of the 3,4-dihydrodiol-1,2-epoxide might be involved. (20 refs)

78-3755 **Suppression of the Development of Carcinogen-Induced Mammary Dysplasias in Female Lewis Rats by Early Chronic Suppression of Prolactin Secretion (Meeting Abstract).** (Eng) Brown, C. K. (Dept. Anatomy, Michigan State Univ., East Lansing, MI, 48824); Smith, M. A.; Welsch, C. W. *Physiologist* 21(4): 13; 1978. (no refs)

78-3756 **Specific-Locus Test of the Possible Mutagenic Effect of Polycyclic Hydrocarbons in the Mouse (Meeting Abstract).** (Eng) Russell, W. L. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN). *Mutat Res* 53(2): 256; 1978. (no refs)

78-3757 **Cellular Uptake, Transport, and Macromolecular Binding of Benzo(a)pyrene and 7,12-Dimethylbenz(a)anthracene by Human Cells In Vitro.** (Eng) Ekelman, K. B. (Dept. Physiological Chemistry, Ohio State Univ., Columbus, OH, 43210); Milo, G. E. *Cancer Res* 38(9): 3026-3032; 1978.

Evidence of the uptake, cytoplasmic protein binding, and nuclear translocation of benzo(a)pyrene (BP) and 7,12-dimethylbenz(a)anthracene (DMBA) by monolayer cultures of preconfluent human neonatal foreskin (HNF) fibroblasts is reported. The max concentration of DMBA in the whole-cell fraction was 6% of the max BP concentration in that fraction, and the max DMBA concentration in the nuclear fraction was 2% that of max nuclear-associated BP. Approx 30% of BP present in the cytoplasm of human fibroblasts was bound DMBA. In BP-treated cells, the extent of polynuclear hydrocarbon uptake by the bound cytoplasmic and nuclear fractions was approx the same; BP appeared first in the bound cytoplasmic fraction and then in the nuclear fraction. The binding of BP to cytoplasmic protein and translocation of this bound polynuclear hydrocarbon to the nucleus appeared to be a function of one or several metabolites of the parent hydrocarbon. In DMBA-treated cells, the pattern and extent of uptake by the crude cytoplasmic fraction were approx the same as those of the nuclear fraction, indicating random dispersal of DMBA throughout the cell. Microelectrophoresis of a cell extract prepared from ^{3}H-BP-treated HNF cells suggested that the ^{3}H-BP-cytoplasmic protein association was noncovalent. (41 refs)

78-3758 **Increased Sensitivity for Detection of Carcinogen-induced DNA Repair with the Chain Terminator Dideoxythymidine.** (Eng) Smith, G. J. (Dept. Pathology, Univ. North Carolina Sch. Medicine, Chapel Hill, NC, 27514); Charlton, R. K.; Grisham, J. W.; Kaufman, D. G. *Biochem Biophys Res Commun* 83(4): 1538-1544; 1978.

DNA excision repair was evaluated in cultured primary hepatocytes following benzo(a)pyrene (BP: 5 or 25 μM) treatment with and without the chain terminators 2′,3′-dideoxythymidine (ddThd: 10 μg/ml) and trifluorothymidine (F_3Thd: 5 μg/ml) in the incubation medium. After 18 hr of incubation, the cells were subjected to alkaline sucrose gradient centrifugation. Eighty-three percent of the DNA from control cells sedimented into the bottom fractions of the gradient, compared with 22% of the DNA from cells exposed to 25 μM BP and incubated with ddThd and F_3Thd. DNA from cells exposed to 25 μM BP alone gave a sedimentation pattern similar to that of control cells, with 68% of the DNA reaching the bottom half of the gradient. Therefore, the inhibitors greatly increased the proportion of slowly sedimenting DNA. For cells exposed to 5 μM BP in the presence of ddThd and F_3Thd, 48% of the total DNA was found in the upper half of the gradient. Under similar conditions, 60% of the DNA from cells exposed to 25 μM BP plus the inhibitors was found in these fractions. Thus, the reduction in DNA sedimentation velocity produced by ddThd and F_3Thd exposure is related to carcinogen dose. DNA chain discontinuities caused by chain growth termination at sites of repair are, collectively, the major contributor to the reduced DNA sedimentation rate, the characteristic observed by this assay, which is more sensitive than those currently available. (14 refs)

78-3759 **Role of Particle Size in the Formation of Respiratory Tract Tumors Induced by Benzo(a)pyrene.** (Eng) Stenback, F. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB, 68105); Rowland, J. *Eur J Cancer* 14(4): 321-326; 1978.

The role of particle size in experimental respiratory tumor formation was studied by intratracheal instillation of two different particle sizes of benzo(a)pyrene (BP) into Syrian golden hamsters. The hamsters received 3 mg BP in large particles (64% >10 μm) or small particles (98% <10 μm) once weekly for 18 wk. Respiratory tract tumors were found in 5/48 animals receiving small particles and in 31/48 animals receiving the larger particles. The small particles cleared much more rapidly from the lungs than did the larger particles. These results indicate the important role of particle size in the experimental induction of respiratory tract tumors. (20 refs)

78-3760 **Analysis of Benzo(a)pyrene Metabolism by High-Pressure Liquid Chromatography.** (Eng) Selkirk, J. K. (Cancer and Toxicology Section, Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN). *Adv Chromatogr* 5(16): 1-36; 1978.

The ease with which high-pressure liquid chromatography can obtain reproducible data on the critical biochemical structural information concerning benzo(a)pyrene (BP) metabolism is exemplified. Four BP phenols were established as metabolites, 3-OH-BP and 9-OH-BP and the newly identified 1-OH-BP and 7-OH-BP. Literature on the metabolism of BP is reviewed. (59 refs)

78-3761 **Analysis of Mutagenic Activity of Benzo(a)pyrene.** (Rus) Krasovskii, G. N. (A. N. Sysin Inst. General and Communal Hygiene, Moscow, USSR); Sokolovskii, V. V.; Varshavskaia, S. P. *Gig Sanit* (3): 102-103; 1978.

To test whether minimal carcinogenic doses of benzo(a)pyrene are mutagenic, random-bred mice were treated with a total of 0.001, 0.01, or 0.1 mg BP given in 10 instillations at 2-day intervals. One day after the last injection, the mice were sacrificed and bone marrow specimens were analyzed. The incidence of chromosome aberrations in the three dosage groups (1.34%, 1.54%, and 1.84%, respectively) was similar to that in controls (1.34%). These findings indicate that BP is not mutagenic at these minimal doses. (5 refs)

78-3762 **Formation of Mutagens in Beef and Beef Extract During Cooking.** (Eng) Commoner, B. (Center Biology Natural Systems, Washington Univ., St. Louis, MO, 63130); Vithayathil, A. J.; Dolara, P.; Nair, S.; Madyastha, P.; Cuca, G. C. *Science* 201(4359): 913-916; 1978.

Studies designed to determine the origin of the mutagens detected in bacterial media that contain beef extract are reported. Beef stock was prepared and then boiled (at temperatures not exceeding 105 C) in an open beaker for 10 hr. Samples were taken at intervals and analyzed for mutagenic activity (MA). Methylene chloride extracts of the stock were tested on histidine-negative *Salmonella typhimurium* strain TA1538 in the presence of a microsomal preparation. An initial sample yielded an av of 21 revertants/plate (vs control value of 29), an 8.5-hr sample yielded 181 revertants/plate, and a 10-hr sample (a dark-brown paste similar to commercial beef extract) yielded 1,572 revertants/plate. Since the mutagens in beef extract are produced in conditions that may be encountered in cooking procedures, their occurrence was determined in cooked beef. Lean ground beef was cooked (at temperatures not exceeding 200 C) on an electric hot plate or home hamburger cooker, and samples were taken after 1.5, 3.0, and 5.5 min and analyzed as above for MA. In the absence of a microsomal preparation, the MA's fell within the range of control values, but in the presence of this preparation, all cooked samples yielded substantial MA. The number of revertants increased with cooking time. The mutagens that occurred in the beef extract and cooked beef were compare chromatographically with those formed by pyrolysis o amino acids and protein and with benzo(a)pyrene (BP These analyses indicated that the mutagens that occurred i the beef stock and ground beef (which were indistinguishab from each other) were distinguishable from both BP and th mutagens produced from pyrolyzed amino acids and protein Well-done hamburgers were calculated to contain about 0.1 ppm of the mutagens and beef bouillon cubes that contai beef extract, about 0.1 ppm. Since these mutagens may b potentially carcinogenic and are formed during ordinar cooking procedures, their occurrence raises questions abou possible risks to human health. (10 refs)

78-3763 **Marked Differences in the Carcinogenic Activity of Optically Pure (+)- an (-)-trans-7,8-Dihydroxy-7,8-dihydrobenzo(a)pyrene in Newborn Mice.** (Eng) Kapitulnik, J. (Dept. Pharmacology Hadassah-Hebrew Univ. Medical Sch., Jerusalem, Israel) Wislocki, P. G.; Levin, W.; Yagi, H.; Thakker, D. R.; Akagi H.; Koreeda, M.; Jerina, D. M.; Conney, A. H. *Cancer Re* 38(9): 2661-2665; 1978.

Optically pure (+)- and (-)-trans-7,8-dihydroxy-7,8-dihydrobenzo(a)pyrene [(+)- and (-)-BP 7,8-dihydrodiol] wer tested for carcinogenicity in newborn Swiss-Webster mice The mice were inoculated ip with 20, 40 and 80 nanomole (nmol) of either compound on days 1, 8, and 15 of life, respectively. The animals were killed at 17 wk of age. Control mic had 0.10 pulmonary adenoma per mouse, whereas animal treated with (+)-BP 7,8-dihydrodiol and (-)-BP 7,8-dihydrodiol had 0.16 and 9.28 pulmonary adenomas per mouse respectively. When a fivefold higher dose was administere according to the above dosage schedule, (+)-BP 7,8-dihydrodiol caused 2.34 pulmonary adenomas per mouse an (-)-BP 7,8-dihydrodiol caused 32.2 pulmonary adenomas pe mouse. When 200, 400, and 800 nmol of benzo(a)pyrene (BP or (+)-BP 7,8-dihydrodiol were administered sequentially o days 1, 8, and 15 of life, 4.13 and 18.5 pulmonary adenoma per mouse, respectively, were observed when the mice wer 27 wk of age. This high dose of (-)-BP 7,8-dihydrodiol kille most of the mice. Administration of (-)-BP 7,8-dihydrodi caused a high incidence of malignant lymphomas, wherea (+)-BP 7,8-dihydrodiol and BP had little or no ability t cause malignant lymphomas. (30 refs)

78-3764 **Explant Culture of Human Colon.** (Eng) Autrup, H. (NCI, NIH, Building 37, Room 3A09 Bethesda, MD, 20014); Barrett, L. A.; Jackson, F. E.; Jesudason, M. L.; Stoner, G.; Phelps, P.; Trump, B. F.; Harris, C C. *Gastroenterology* 74(6): 1248-1257; 1978.

Nontumorous colonic tissues from patients with or withou cancer were cultured as explants in a chemically define medium for > 4 to 20 days. The viability of the explants wa shown by the preservation of the ultrastructural features to the colonic epithelial cells and by the active incorporation of radioactive precursors into cellular DNA and protein. A progressive decrease in the number of goblet cells, a decrease in the depth of the crypts, and a change from a columnar to a cuboidal epithelium were observed. After 20 days in culture, the colonic mucosa consisted of a single layer of cuboidal epithelial cells and a few glands. The survival of colonic mucosa in culture was subject to both intra- and interindividual variation. Cultured human colonic mucosa also activated a chemical procarcinogen, benzo(a)pyrene (BP), into metabolites that bound to cellular DNA. BP binding levels in 32 explants (7 from patients with colon cancer) had a unimodal distribution, and the values varied from 0.2 to 20 picomoles/10 mg DNA (median value, 4.5). Possible factors in the one hundredfold interindividual variation in binding levels are discussed. (20 refs)

78-3765 **Carcinogenicity and Metabolic Profiles of 6-Substituted Benzo(a)pyrene Derivatives on Mouse Skin.** (Eng) Cavalieri, E. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB, 68105); Roth, R.; Grandjean, C.; Althoff, J.; Patil, K.; Liakus, S.; Marsh, S. *Chem Biol Interact* 22(1): 53-67; 1978.

Benzo(a)pyrene (BP) and nine 6-substituted derivatives were applied to the shaved back skin of 8-wk-old female Swiss mice in order to analyze the relative carcinogenicity of these compounds. The mice were treated with 0.2 micromole (μmol) of compound in acetone 4x/wk for 20 wk. In addition, the metabolism of the compounds was studied using mouse skin homogenates in vitro and mouse skin in vivo. Only four compounds were carcinogenic, and the order of potency was BP > 6-methylbenzo(a)pyrene (BP-6-CH_3), > 6-hydroxymethylbenzo(a)pyrene (BP-6-CH_2OH) = benzo(a)pyrene-6-carboxaldehyde (BP-6-CHO). Because the latter two compounds were partially in suspension, the small difference in their carcinogenicity could not be assessed. This problem was resolved in a further experiment, in which the carcinogenicity of these two compounds plus that of BP-6-CH_3 and 6-hydroxymethylbenzo(a)pyrene sulfate ester (BP-6-CH_2OSO_3Na) was compared at three dose levels (0.8, 0.2, and 0.05 μmol, 2x/wk for 40 wk). BP-6-CH_2OSO_3Na proved to be a more potent carcinogen than BP-6-CH_2OH, which in turn was more active than BP-6-CHO. The greater carcinogenicity of BP-6-CH_3 relative to that of BP-6-CH_2OH and BP-6-CHO was confirmed, suggesting that BP-6-CH_2OH is not a proximate carcinogenic metabolite for BP-6-CH_3. Since BP-6-CHO was weaker than BP-6-CH_2OH and was efficiently reduced metabolically to BP-6-CH_2-OH, the latter may be a common proximal carcinogenic metabolite. The more potent carcinogenicity of BP-6-CH_2OSO_3Na relative to that of BP-6-CH_2OH suggests that an ester of the latter might be the ultimate alkylating compound reacting with cellular nucleophiles. (30 refs)

78-3766 **Increase of Liver Microsomal Benzo(a)pyrene Monooxygenase Activity by Subsequent Glucuronidation.** (Eng) Bock, K. W. (Institut fur Pharmakologie und Toxikologie der Universitat, Kreuzbergring 57, D-3400 Gottingen, W. Germany). *Naunyn Schmiedebergs Arch Pharmacol* 304(1): 77-79; 1978.

Benzo(a)pyrene (BP) monooxygenase activity in rat liver microsomes was measured with and without subsequent addition of uridine diphosphate (UDP)-glucuronic acid (UDPGA) and/or UDP-N-acetylglucosamine (UDPNAG) to the incubation medium. The microsomes were obtained from rats pretreated with 3-methylcholanthrene (3-MC) or phenobarbital. BP monooxygenase activity was significantly increased when the assay was performed in the presence of UDPGA and further increased by the addition of UDPNAG, which is an allosteric activator of UDP-glucuronyltransferase. Specific enzyme activities found with microsomes from untreated, phenobarbital-, or 3-MC-treated rats were 0.5, 0.5 and 1.6 nanomoles/min/mg protein, respectively. However, the increase of monooxygenase activity by the addition of both UDPGA and UDPNAG was similar (180%, 200%, and 219% with microsomes from untreated, phenobarbital-, and 3-MC-treated rats, respectively). Experiments with guinea pig liver microsomes produced similar results. The results suggest that there is a coupling between microsomal monooxygenase and UDP-glucuronyltransferase. This coupling may be involved in the elimination of toxic compounds. (12 refs)

78-3767 **A Comparative Study of Aryl Hydrocarbon Hydroxylase Induction in Rat Liver and Lungs by 5,6-Benzoflavone.** (Fre) Queval, P. (Charge de Recherches a l'INSERM, Laboratoire de Pathologie pleuropulmonaire F.R.A., 16 INSERM, Hopital Laennec, 42, rue de Sevres, 75007 Paris, France); Beaumatin, J. *Rev Fr Mal Respir* 6(2): 191-194; 1978.

Induction of the microsomal enzyme aryl hydrocarbon hydroxylase (AHH) was measured in the livers and lungs of 3- to 4-mo-old Sprague-Dawley rats inoculated ip with 20, 25, or 30 mg/kg 5,6-benzoflavone (BF). The rats were sacrificed at 24-hr intervals beginning on day 1 and ending on day 4. In the liver, a peak level of AHH activity was reached after 24 hr; in the lung, a peak level was reached after 48 hr and it remained stable for about 10 days. The alterations in lung enzyme levels were attributed to passage of BF from the abdominal cavity to the lung by the lymphatic system. (13 refs)

78-3768 **Nuclear Metabolism of Polycyclic Hydrocarbons and Interaction of Polycyclic Hydrocarbons with Nuclear Components.** (Eng) Bresnick, E. (Univ. Vermont Coll. Medicine, Burlington, VT, 05401). *Adv Enzyme Regul* 16: 347-361; 1978.

The ability of the nucleus to activate polycyclic hydrocarbons was investigated in male Sprague-Dawley rats and male mice of various strains treated ip with 20 mg/kg 3-methylcholanthrene (MC) or corn oil. Twenty-four hours later, the animals were killed and the nuclei isolated by centrifugation. The nuclei were incubated with 2 μg of benzo(a)pyrene (BP) and examined by histofluorescence for BP metabolites. Nuclear aryl hydrocarbon hydroxylase (AHH) was present in the nuclear fraction at 15%-20% of the level found in the microsomal fraction. Cytochrome P_{450} was also present within the nucleus, and both AHH and hemopigments were inducible by MC treatment. Rat liver nuclei were incubated with ^{14}C-BP or ^{3}H-(±)trans-BP-7,8-dihydrodiol (^{3}H-BP-7,8-dihydrodiol) in the presence of an NADPH generating system. Prior administration of MC to the rats stimulated the total nuclear metabolism 11-fold. BP was oxidized to the 9,10-, 4,5-, and 7,8-dihydrodiols, the 3,6- and 1,6-quinones, and the 3- and 9-phenols, and BP-7,8-dihydrodiol formation was enhanced 29-fold. The nuclei were also able to produce BP diol epoxides 1 and 2 from BP-7,8-dihydrodiol; MC pretreatment increased this nuclear activity fourfold. Incubation of ^{3}H-BP with liver nuclei in an NADPH-generating system also resulted in aralkylation of DNA, histones, and nonhistone components; prior MC treatment stimulated the aralkylation of these macromolecules. The alkylated deoxyribonucleosides produced after enzymatic digestion of the incubated DNA were separated by Sephadex LH-20 chromatography. Two peaks of radioactivity noted after incubation with BP, but only one major peak was noted after incubation with the diol derivative. It is suggested that the nuclear metabolism of BP and the subsequent alkylation of the DNA may be related to mutagenesis and/or carcinogenesis. (35 refs)

78-3769 **Appraisal of Fluorimetric Assay of Aryl Hydrocarbon (Benzo(α)pyrene) Hydroxylase in Cultured Human Lymphocytes.** (Eng) Trieff, N. M. (Environmental Toxicology Dept. Preventive Medicine and Community Health, Univ. Texas Medical Branch, Galveston, TX, 77550); Cantelli Forti, G.; Smart, V. B.; Kempen, R. R.; Kilian, D. J. *Br J Cancer* 38(2): 335-338; 1978.

Problems inherent in the fluorimetric determination of aryl hydrocarbon hydroxylase (AHH) activity in human lymphocytes were examined. Six simulated experiments were performed according to the procedure developed by Kellerman and coworkers except that lymphocytes were omitted and generally tritiated benzo(a)pyrene (BP) was used. It was shown that 11.72% of the total radioactivity ended up in the NaOH phase, but the method was developed to ensure that no BP, only 3-BPOH, was being extracted by the NaOH. As the BP fluorescence spectrum has one peak with a wavelength max at 522 nanometers, the same as that for 3-BPOH, there is substantial interference of BP in the measurement of the fluorescence intensity of 3-BPOH, especially at low 3-BPOH concentrations and at small slit widths. The photochemical instability of BP, 3-BPOH, and other metabolites may lead to additional errors. (25 refs)

78-3770 **Aryl Hydrocarbon Hydroxylase in Persons with Lung or Laryngeal Cancer.** (Eng) Ward, E. (Dept. Res. Medicine, Univ. Pennsylvania, Philadelphia, PA, 19104); Paigen, B.; Steenland, K.; Vincent, R.; Minowada, J.; Gurtoo, H. L.; Sartori, P.; Havens, M. B. *Int J Cancer* 22(4): 384-389; 1978.

Aryl hydrocarbon hydroxylase inducibility was measured in cultured lymphocytes of 59 cancer patients (32 lung and 27 laryngeal) whose tumors were completely resected 1-10 yr prior to testing and who were considered disease-free. In these patients, AHH inducibility should not have been affected by the disease state. The mean inducibility ratio was 3.2 for the lung cancer patients, 2.96 for the laryngeal cancer patients, and 3.29 for controls. Induced enzyme activity was also similar for patients and controls: 0.25 picomole of 3-hydroxybenzo(a)pyrene/min/10 μg DNA for patients with laryngeal and lung cancer and 0.27 for controls. Thus, no shift toward higher AHH inducibility was observed in patients who had lung or laryngeal cancer compared with controls. (21 refs)

78-3771 **Control of Benzo(a)Pyrene (BP)-induced Mammalian Cell Mutagenesis and Cytotoxicity by Exogenous Enzyme Fractions (Meeting Abstract).** (Eng) Schechtman, L. M. (Dept. Biochemical Oncology, Microbiological Associates, Bethesda, MD, 20016); Kouri, R. E. *Mutat Res* 53(2): 258-259; 1978. (1 ref)

78-3772 **Stimulatory Effect of Cigarette Smoke on the Metabolism and Covalent Binding of Benzo(a)-pyrene in the Trachea of the Rat.** (Eng) Simberg, N. (Dept. Physiology, Univ. Turku, SF-20520 Turku 52, Finland); Uotila, P. *Int J Cancer* 22(1): 28-31; 1978.

The effect of cigarette smoke on the activities of enzymes involved in benzo(a)pyrene (BP) metabolism was investigated in male Wistar rats, along with the degree of covalent binding of BP in the rat trachea. The rats were exposed to the smoke of five commercial filter cigarettes for 1 hr daily, for 1 or 10 days (Group A and B, respectively). Aryl hydrocarbon hydroxylase (AHH) activity was increased in the tracheas of Groups A and B rats, reaching a max 12 hr after the last exposure to 320% and 290% of controls, respectively. However, this activity returned to control levels within 24 hr. At 12 hr after the last exposure, AHH activity in the pulmonary microsomes was 560% of controls in both groups, and it was still increased (360%) at 24 hr. Concomitantly with the increase in AHH activity, the amounts of covalently bound metabolites of BP were increased twofold in the nucleic acid and protein fractions of the tracheas of Group A rats. There was no significant changes in tracheal epoxide hydratase activity in both groups. Repeated exposures slightly increased the activity of uridine diphosphate-glucuronosyltransferase in both the trachea and lung. The results indicate that the metabolism of polycyclic hydrocarbons present in cigarette smoke is enhanced in the respiratory tract after cigarette smoke exposure. (28 refs)

78-3773 **Longevity, Selective Mortality, and Competitive Risks in Relation to Chemical Carcinogenesis.** (Eng) Hammond, E. C. (Dept. Epidemiology and Statistics, American Cancer Society, 777 Third Ave., New York, NY, 10017); Garfinkel, L.; Lew, E. A. *Environ Res* 16(1/3): 153-173; 1978.

Male cigarette smokers and nonsmokers were compared periodically with respect to longevity, total death rates, deaths from cancer (all sites combined), and deaths from cancer of three specific sites: lung, pancreas, and bladder. The data were obtained through questionnaire surveys of men in 22 states, the starting date for the analyses being July 1, 1960, and the ending dates June 30, 1975, for men born in 1887 or earlier and June 30, 1971, for men born later than 1887. The findings were compared with the results calculated for three hypothetical types of cigarettes: LCA, which induce the same lung cancer rates as ordinary cigarettes but have no other effects; BPCA, which increase death rates from bladder and pancreatic cancer; and CA, which yield the same age-specific death rates from cancer of every site and type as ordinary cigarettes. Life expectancy at age 40 was 37.85 yr for nonsmokers, 34.87 yr for light smokers, and 30.79 yr for heavy smokers. The proportion of men living to age 65 was 86.04% for nonsmokers, 79.61% for light smokers, and 70.55% for heavy smokers. The proportions of men living to age 85 were 29.12%, 20.15%, and 9.7%, respectively, for the three groups. The mean ages at death from cancer were 74.93 for nonsmokers, 73.66 for light smokers, and 70.83 for heavy smokers. CA smokers had the same age-specific cancer death rates as ordinary cigarette smokers; LCA smokers, the same age-specific lung cancer death rates as ordinary smokers; BPCA smokers, the same bladder and pancreas death rates as ordinary smokers. Otherwise, the death rates in these three groups were the same as those for nonsmokers. (no refs)

78-3774 **Pathologic Changes in the Lungs of Mice after Inhalation of Cigarette Smoke.** (Cro) Kambic, V. (Otorinolaringoloska klinika, Ljubljana, Zaloska 2, Yugoslavia); Radsel, Z.; Jutersek, A. *Plucne Bolesti Tuberk* 29(3/4): 181-185; 1977.

A study was made of pathological changes in the lungs of 42 male mice exposed to cigarette smoke for various lengths of time over a 10-wk period. The animals were sacrificed at the end of the experiment. Microscopically, there were pulmonary changes in all animals exposed longest to the smoke, but in only 2/7 animals least exposed. The changes included peribronchitis, bronchitis, pneumonia and pulmonary abscess. Normal epithelium was found in the low-exposure groups, but thickened epithelium, abnormal cilia, and loss of the cilia were seen in the high-exposure groups. The incidence and degree of the changes correlated with the degree of exposure. With prolonged exposure, these changes are believed to be capable of developing into precancerosis and carcinoma. Changes in the pharynx and larynx were less marked. (8 refs)

78-3775 **Determination of Polycyclic Aromatic Hydrocarbons in the Air and in the Work Environment.** (Ger) Otto, J. (Arbeitshygieneinspektion des Rates des Bezirkes, Predigerstrasse 6, DDR-50 Erfurt, E. Germany); Schmidt, E. *Z Gesamte Hyg* 24(8): 584-587; 1978.

A gas chromatographic method for determining polycyclic aromatic hydrocarbons in the air is described. A sample size of 5 μl was optimal. With the aid of a flame ionization detector, the detection limits were 0.005 μg for fluorene and anthracene, 0.013 μg for benzo(a)pyrene, and 0.025 μg for benzo(a)fluorene. The av error was 10%. (no refs)

78-3776 **Embryotoxic Effects of Benzene and Its Methyl Derivatives: Toluene, Xylene.** (Eng) Hudak, A. (Dept. Experimental Pathology, State Inst. Occupational Health Budapest, H-1450 Budapest P.O.B. 22, Hungary); Ungvary, G. *Toxicology* 11(1): 55-63; 1978.

The teratogenic and embryotoxic effects of benzene, toluene, and xylene were determined in CFY rats and CFLP mice. The rats were exposed to inhalation of (a) 1,000 mg/m^3 benzene, 1,500 mg/m^3 toluene, or 1,000 mg/m^3 xylene for 24 hr/day from days to 14 of pregnancy; (b) 1,500 mg/m^3 toluene for 24 hr/day from days 1 to 8 of pregnancy, or (c) 1,000 mg/m^3 toluene for 8 hr/day from days 1 to 21 of pregnancy. The mice were exposed to inhalation of 500 mg/m^3 toluene for 24 hr/day from days 6 to 13 of pregnancy. Untreated animals and groups inhaling pure air served as controls. None of the solvents were teratogenic; ie, the incidence of malformations did not change as a result of exposure. However, the incidence of skeletal anomalies (extra ribs, fused sternebrae) was increased with all three solvents. Benzene and toluene also caused considerable retardation of fetal development. The growth-retarding effect of toluene on fetal development in early pregnancy is of particular importance from the point of view of occupational exposure. (32 refs)

78-3777 **Analysis of Sensitizing Effect of Benzene Administered Simultaneously in Water and in Air.** (Rus) Tepikina, L. A. (A. N. Sysin Inst. General and Communal Hygiene, Moscow, USSR); Gordeeva, M. S. *Gig Sanit* (3): 23-27; 1978.

The sensitizing effect of benzene was studied in random-bred albino rats who were exposed simultaneously to benzene in their drinking water (45.0, 9.0, 1.5 mg/kg) and by inhalation

(15.0, 6.63, 0.51 mg/m^3) for up to 100 days. Sensitization was measured by a microprecipitation reaction, a passive hemagglutination reaction, and by skin allergy tests. Positive immunologic reactions were detected on days 21-50 of the experiment, depending on dose. (9 refs)

78-3778 **Followup Study on the Mortality and the Development of Leukemia in 44 Pancytopenic Patients with Chronic Exposure to Benzene.** (Eng) Aksoy, M. (Buyukciflik sokak 10/2 Ozler, ap., Tesvikiye, Istanbul, Turkey); Erdem, S. *Blood* 52(2): 285-292; 1978.

Further evidence of the leukemogenic effect of benzene (BZ) in humans was obtained from a 2- to 17-yr follow-up study of 44 patients with pancytopenia (PCP) who had been exposed occupationally to 150-650 ppm BZ in various adhesives for 4 mo-15 yr (mean, 6.7 yr). The BZ level in the adhesives ranged from 9% to 88%, and daily exposure ranged from 15 min to 8 hr. Thirty-four patients worked in shoe factories. Complete remission of PCP was obtained in 23 patients, but 21 died, 14 due to complications of PCP, 6 following the development 6 mo-6 yr later of leukemia, and 1 due to myeloid metaplasia. The mean age and duration of exposure were significantly higher in the 21 patients with a fatal outcome (36.0 yr and 93.62 mo, respectively) than in the 23 complete remission patients (26.87 yr, 65.43 mo). Nevertheless, except for the mean neutrophil values 1.51 x 10^9/liter for the remission patients, 0.96 x 10^9/liter for the patients that died), the findings in the peripheral blood, including HbF and HbA$_2$ levels, could not be correlated with the outcome of the PCP. However, one patient who had a 0.67% level of HbA$_2$ and a 48% level of HbF during the PCP period developed preleukemia 9 mo later. This observation suggests that low levels of HbA$_2$ with or without high levels of HbF during PCP may be a sign of leukemia. Twenty-one patients, 5 of whom developed leukemia, had a hypocellular bone marrow; 16 of these patients died. Only 1/13 patients with normocellular bone marrow died. (31 refs)

78-3779 **Clastogenic Effects of Styrene Exposure on Bone Marrow Cells of Rat.** (Eng) Meretoja, T. (Dept. Industrial Hygiene and Toxicology, Inst. Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland); Vainio, H.; Jarventaus, H. *Toxicol Lett* 1(5/6): 315-318; 1978.

Male Wistar rats were exposed to 300 ppm styrene in air for 2-11 wk (6 hr/day, 5 days/wk) to determine its effects on bone marrow cells. The chromosome aberration rate increased significantly between 8 and 9 wk of exposure and remained at this level to the end of the test. Aberrant cell incidence ranged from 8% to 12% (1-6% in controls). Polyploid cells were noted exclusively in exposed animals, and they were found in every sample from animals exposed for 11 wk. (8 refs)

78-3780 **Clastogenic Activity of Styrene in Occupation
Exposure (Meeting Abstract).** (Eng) Meretoi
T. (Dept. Industrial Hygiene and Toxicology, Inst. Occup
tional Health, Helsinki, Finland); Vainio, H.; Sorsa,
Mutat Res 53(2): 229; 1978. (no refs)

78-3781 **Analysis of the Mutations Induced by Styren
Oxide in V79 Chinese Hamster Cells (Meetin
Abstract).** (Eng) Bonatti, S. (Laboratorio di Mutagenesi
Differenziamento, CNR, Pisa, Italy); Corti, G.; Mazzaccar
A.; Abbondandolo, A. *Mutat Res* 53(2): 154; 1978. (no ref

78-3782 **Metabolism of the Proximate Carcinoge
1′-Hydroxysafrole and the Isomer 3′-Hydro
yisosafrole.** (Eng) Peele, J. D. (Dept. Lab. Medicine, Medic
Univ. South Carolina, 80 Barre St., Charleston, SC, 2940
Oswald, E. O. *Bull Environ Contam Toxicol* 19(4): 396-40
1978.

The metabolism of the proximate carcinogen 1′-hydroxys
frole (HS) and the isomer 3′-hydroxyisosafrole (HIS) was i
vestigated in the rat. The neutral urinary extracts from ra
inoculated with HS contained the unmetabolized compou
and HIS, along with 3,4-methylenedioxyphenyl vinyl keto
and two other neutral metabolites that were not fully chara
terized. The basic urinary fraction of rats given HS contain
the dimethylamino and piperidyl Mannich base metabolite
The only observed acidic metabolite was 3,4-methylenedio
yhippuric acid. The neutral urinary extract from rats give
HIS contained the unmetabolized parent compoun
3,4-methylenedioxyacetophenone, and traces of HS. The ba
ic fraction contained no metabolites. The acidic fraction co
tained 3,4-methylenedioxyacetophenone and 3′-(3,4-m
thylenedioxyphenyl)-3′-hydroxypropionic acid. In additio
traces of piperonylic acid and 3,4-methylenedioxyhippur
acid were present. A mechanism for the carcinogenic actio
of HS is offered that supports the electrophilic theory of ca
cinogenesis and associates chemical reactivity with biologic
response. The mechanism involves stabilization of a carbon
um ion intermediate by the resonance effect of the methylen
dioxy group. This resonance stabilization could increase t
electrophilicity of the molecule, allowing it to attack tiss
nucleophiles. (12 refs)

78-3783 **2,3,7,8,-Tetrachlorodibenzo-p-dioxin as Coca
cinogen Causing 3-Methylcholanthrene-initia
ed Subcutaneous Tumors in Mice Genetically "Nonrespo
sive" at *Ah* Locus.** (Eng) Kouri, R. E. (Dept. Biochemic
Oncology, Microbiological Associates, Bethesda, M
20016); Rude, T. H.; Joglekar, R.; Dansette, P. M.; Jerin
D. M.; Atlas, S. A.; Owens, I. S.; Nebert, D. W. *Cancer R*
38(9): 2777-2783; 1978.

The purpose of this study was to determine whether 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), at a dose (100 μg/kg ip or sc) sufficient to induce aryl hydrocarbon hydroxylase (AHH) activity and cytochrome P_1-450 in genetically nonresponsive DBA/2 mice would enhance the susceptibility of these mice to 3-methylcholanthrene (3-MC) tumorigenesis. TCDD alone ip killed 30%-70% of the animals in each group of DBA/2 or responsive C57BL/6 mice, but it produced no tumors in survivors 36 wk later. TCDD given to C57BL/6 mice ip 2 days prior to or simultaneously with sc 3-MC did not affect the 3-MC carcinogenic index (which is about 60 in these mice). TCDD given to DBA/2 mice ip 2 days prior to sc 3-MC increased the carcinogenic index slightly (from 5 to 10; in these mice, the carcinogenic index of 3-MC alone is 1). When TCDD was injected simultaneously (sc, in the same vehicle) with 3-MC into DBA/2 mice, the carcinogenic index was increased markedly (from 5 to 38). In this test system, therefore, TCDD appeared to be carcinogenic. However, it is concluded that TCDD is a cocarcinogen. Its proposed mechanism of action is the induction of AHH activity and the associated P_1-450 at the site of inoculation of 3-MC in genetically nonresponsive mice, thereby resulting in more efficient metabolic conversion of 3-MC to the (proximal or) ultimate carcinogen. (39 refs)

78-3784 **Correlation Between Carcinogenicity and Polarographic Reducibility of Some N-substituted Derivatives of 4-Aminoazobenzene.** (Eng) Carruthers, C. (Orchard Park Labs., Roswell Park Memorial Inst., Buffalo, NY, 14263). *Indian J Cancer* 15(1): 49-52; 1978.

The polarographic behavior of six carcinogenic and noncarcinogenic derivatives of 4-aminoazobenzene (4-AAB) was investigated in buffer-ethanol solns at pH 2.6, 4.8, 6.8, 8.2, 9.5, and 11.0, to determine whether there is a correlation between carcinogenicity and polarographic reducibility. The half-wave potential of the noncarcinogens 4-benzylmethyl-AAB, 4-diethyl-AAB, and 4,β-hydroxyethylmethyl-AAB and the carcinogens 4-ethylmethyl-AAB, N-methyl-AAB, and 3′-chloro-4-dimethyl-AAB was essentially the same at any given pH, and it was comparable to that of 4-AAB, 4-dimethyl-AAB, and 3′-methyl-4-dimethyl-AAB (found previously). The results showed that variation in the substituents at the N-position did not appreciably affect the half-wave potential of 4-AAB. (13 refs)

78-3785 **Electrophilic Reactivity and Mutagenicity of Ring-Methyl Derivatives of N-Acyloxy-N-methyl-4-aminoazobenzene and Related Azo Dyes.** (Eng) Degawa, M. (Dept. Hygienic Chemistry, Pharmaceutical Inst., Tohoku Univ., Aobayama, Sendai 980, Japan); Miyairi, S.; Hashimoto, Y. *Gann* 69(3): 367-374; 1978.

The N-hydroxy, N-benzyloxy, and N-acyloxy derivatives of N-methyl-4-aminoazobenzene (MAB), 3′-methyl-MAB, and 4′-methyl-MAB, dyes that have different carcinogenic activities in rats, were synthesized and the electrophilic reactivity and mutagenicity of the parent dyes and the derivatives were compared. The three parent dyes did not react with amino acids, proteins, or DNA; the N-hydroxy derivatives showed slight reactivity with all these substances except for cysteine. The mutagenicity of these dyes towards *Salmonella typhimurium* TA98 and TA100 was expressed only in the presence of the 9,000-*g* supernatant of a rat liver homogenate (S-9 mix), and the 3′-methyl or 4′-methyl substituent did not affect the mutagenic activity of the parent dyes. In contrast, the N-acetoxy and N-benzoyloxy derivatives of the three dyes showed a high degree of reactivity with amino acids, proteins, and DNA, and they were strongly mutagenic toward the bacteria without S-9 mix. No significant differences were observed among these derivatives either in their electrophilic reactivity or mutagenicity. These findings suggest that neither the rate of metabolic activation to ultimate metabolites by liver enzymes nor the electrophilic and mutagenic activities of the N-acyloxy and N-benzoyloxy derivatives were related to the hepatocarcinogenicity of MAB, 3′-methyl-MAB, and 4′-methyl-MAB. Other possible factors influencing the carcinogenicity of MAB homologs are discussed. (18 refs)

78-3786 **Appearance of Intestinal Type of Tumor Cells in Hepatoma Tissue Induced by 3′-Methyl-4-dimethylaminoazobenzene.** (Eng) Yoshida, Y. (La Jolla Cancer Res. Foundation, P.O. Box 1376, 417 Coast Blvd. S., La Jolla, CA, 92038); Kaneko, A.; Chisaka, N.; Onoe, T. *Cancer Res* 38(9): 2753-2758; 1978.

Hepatomas induced in male Wistar rats by 3′-methyl-4-dimethylaminoazobenzene (3′-Me-DAB: 0.06% of diet for 14 wk) were investigated both morphologically and biochemically. The most prominent histological pattern was that of undifferentiated carcinoma. Although this type of carcinoma, histologically, appeared to be due to a uniform population of cells, electron microscopic examination revealed that the tissue was composed of many types of cells, including cells that contained either the brush border or the mucous droplets seen in goblet cells. In addition, tumor cells that contained serotonin like granules were noticed. An electrophoretogram of alkaline phosphatase activity in the tissue extract of this type of carcinoma revealed distinctly the presence of its intestinal-type isoenzyme. These findings show that carcinomas induced by 3′-Me-DAB contain cells differentiated toward hepatocytes or cholangiolar cells and those differentiated toward intestinal epithelial cells. (19 refs)

78-3787 **Hepatoma Induction in the Rat by the Subcutaneous Administration of Powdered 3′-Methyl-p-dimethylaminoazobenzene.** (Eng) Carruthers, C. (Orchard Park Labs., Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263); Baumler, A.; Pauly, S. *Experientia* 34(6): 788; 1978.

Hepatomas were induced in male Fischer rats with the use of a procedure that does not require special facilities for formulating diets containing azocarcinogens. Powdered 3′-methyl-p-dimethylaminoazobenzene (3′-Me-DAB; 200 mg) was evenly distributed under skin flaps created by a 5- to 6-cm abdominal incision in 6- to 8-mo-old rats. This procedure was repeated every 30 days for 3-7 mo. The rats tolerated the sc application of powdered 3′-Me-DAB very well. In four experiments, 67%-82% of rats developed liver tumors (mixtures of hepatocellular carcinomas and cholangiocarcinomas). It is concluded that the sc procedure for inducing liver tumors should be useful when facilities for incorporating 3′-Me-DAB into the diet are unavailable. However, for personal safety, an isolated laboratory should be used for the monthly 3′-Me-DAB administration. (7 refs)

78-3788 **In Vitro Evaluation of Some Derivatives of the Carcinogen Butter Yellow: Implications for Environmental Screening.** (Eng) Ashby, J. (Central Toxicology Lab., Imperial Chemical Industries Ltd., Alderley Park, Macclesfield, Cheshire, England); Styles, J. A.; Paton, D. *Br J Cancer* 38(1): 34-50; 1978.

The rat liver carcinogen 4-dimethylaminoazobenzene (Butter Yellow, DAB), several structural analogs, benzidine, and benzidine-3,3′-disulfonic acid were tested in vitro for their ability to transform BHK21/C13 cells in the presence of an Aroclor 1254-induced rat liver S-9 mix. Positive results were obtained for DAB; a mixture of DAB and azobenzene; 9-phenylazojulolidine; N-methyl-5-phenylazoindoline; DAB-4′-sulfonic acid, sodium salt (methyl orange); benzidine, 4-dimethylamino-4′-trifluoromethylazobenzene; 4-diethylaminoazobenzene; 4-diethylamino-4′-ethylazobenzene; 4-aminoazobenzene; and 4-aminoazobenzene-4′-sulfonic acid. 3-Methyl-DAB, azobenzene, 3,5-dimethyl-DAB, benzidine-3,3′-disulfonic acid, and 3,5-dimethyl-4-aminoazobenzene were negative. It is suggested that the promotion of DAB-induced transformation by azobenzene is the result of the latter acting as a competitive substrate for one of the enzymes that detoxify the former. These findings are correlated with those of previous experiments. This assay cannot be relied on to predict the in vivo potency of carcinogens. (56 refs)

78-3789 **Biochemical Features of Liver from Low- and High-Cancer Mouse Strains and of Primary Spontaneous Hepatomas. Distribution of Hepatic Enzymes in Induced and Spontaneous Hepatomas and in Normal Liver of Low- and High-Cancer Mouse Strains.** (Rus) Gobeev, V. N. (Dept. Biochemistry, Patrick Lumumba Univ., Moscow, USSR); Khripach, L. V.; Berezov, T. T. *Vopr Med Khim* 24(4): 533-539; 1978.

The activity of various hepatic enzymes was studied in CBA mice with spontaneous hepatomas and in C3HA mice with o-aminoazotoluene(o-AAT)-induced hepatic carcinomas (pills containing o-AAT were implanted sc, approx 1x/mo). Spontaneous hepatomas were detected in 70% of CBA males (compared with 10% of CBA females). Hexokinase (HK) activity was significantly increased in the hepatoma tissue (0.28 IU, compared with 0.11 IU in tumor-free regions of the liver), but cytochrome oxidase (CO) activity was significantly decreased in the hepatomas (1.1 IU, compared with 2.5 IU in the unaffected liver tissue of tumor-bearing animals). Similar changes were detected in C3HA mice with induced hepatomas: the activity of HK and CO in the liver of tumor-bearing animals was 0.37 and 3.1 IU, respectively, compared with 0.11 and 3.6 IU in tumor-free mice. (15 refs)

78-3790 **Responsiveness of Peroxisomal Enzymes of Rat Livers During 3′-Methyl-4-dimethylaminoazobenzene Hepatocarcinogenesis to Clofibrate.** (Eng) Mochizuki, Y. (Dept. Pathology, Cancer Res. Inst., Sapporo Medical Coll., Sapporo 060, Japan); Itabashi, M. *Tumor Res (Sapporo)* 12: 37-46; 1977.

The effect of clofibrate on the activities of the peroxisomal enzymes catalase, D-amino acid oxidase, urate oxidase, and L-α-hydroxy acid oxidase was determined in the livers of male Wistar rats fed a diet containing 0.06% 3′-methyl-4-dimethylaminoazobenzene (3′-Me-DAB). Animals were sacrificed after 10, 13, 15, 17, and 19 wk on the diet; 1% clofibrate was added to the diet 2 wk before sacrifice. Although 3′-Me-DAB depressed catalase activity, clofibrate increased the activity of this enzyme at all time intervals studied. (Preliminary experiments indicated that the induction of catalase by clofibrate was not influenced by the carcinogen.) The highest increase was observed at 13 wk. This suggested that hepatocytes that had degenerated early after 3′-Me-DAB administration were undergoing maturation. The minimum increase in catalase activity in response to clofibrate was observed at 19 wk; this indicated that the lesions that were not responsive to clofibrate had increased in number. Foci that were not responsive to clofibrate were noted as early as week 10; therefore, changes essential to hepatoma formation had occurred by this time. The hepatoma precursor lesions were increased by 17-19 wk. The activity of D-amino acid oxidase was decreased by clofibrate at all times. Neither urate oxidase nor L-α-hydroxy acid oxidase activities were affected by clofibrate during carcinogenesis. (69 refs)

78-3791 **Enhancing and Inhibitory Effects of Some Stilbene and Steroid Compounds on Induction of Hepatoma in Rats Fed 3′-Methyl-4-(dimethylamino)azobenzene.** (Eng) Akao, M. (Res. Inst. Chemobiodynamics, Chiba Univ., Inohana 1-8-1, Chiba 280, Japan); Kuroda, K. *Gann* 69(3): 375-382; 1978.

The enchancement or inhibition of hepatoma induction in 3′-methyl-4-(dimethylamino)azobenzene (3′-Me-DAB)-treated male Donryo rats by stilbene, 4-nitrostilbene (NSB), 4,4′-dihydroxystilbene, diethylstilbestrol (DES), 17β-estradiol, and methyltestosterone was studied. Rats were fed a diet containing 0.06% 3′-Me-DAB to a total of 0.5 or 1 g, followed by 0.25 or 0.5 g of test substance in a basal diet. Comparison of the development and yield of hepatomas indicated that NSB and methyltestosterone enhanced 3′-Me-DAB carcinogenesis, whereas DES and 17β-estradiol retarded it. The other substances had no effect on carcinogenesis. The enhancement by NSB and inhibition by DES was correlated with their effect on liver nucleic acid metabolism. Feeding of NSB selectively inhibited the Mn^{2+}-$(NH_4)_2SO_4$-activated RNA polymerase activity of liver nuclei and reduced liver RNA content. These changes were followed by an increase in the incorporation of ip-injected ^{3}H-thymidine into liver DNA. In contrast, feeding of DES increased liver RNA content without affecting the RNA polymerase activity of liver nuclei; and it also increased incorporation of ^{3}H-thymidine into DNA. Moreover, the increased incorporation ceased when DES was removed from the diet. (19 refs)

78-3792 **Squamous Cell Dysplasia and Carcinoma In Situ of the Cervix and Vagina after Prenatal Exposure to Diethylstilbestrol.** (Eng) Robboy, S. J. (Dept. Pathology, Massachusetts General Hosp., Boston, MA, 02114); Keh, P. C.; Nickerson, R. J.; Helmanis, E. K.; Prat, J.; Szyfelbein, W. M.; Taft, P. D.; Barnes, A. B.; Scully, R. E.; Welch, W. R. *Obstet Gynecol* 51(5): 528-535; 1978.

The prevalence and incidence of squamous cell dysplasia of the vagina and cervix were determined in 1,424 women (aged 13-31 yr) exposed to diethylstilbestrol (DES) in utero. Some of these women had been followed for 6 yr. Six types of squamous cell abnormalities were noted: mature squamous metaplasia, immature squamous metaplasia, mild dysplasia, moderate dysplasia, severe dysplasia, and carcinoma in situ. The prevalence of dysplasia was 2.1%, and the incidence was 0.85/100 person-yr of follow-up. The dysplastic epithelial changes were almost always mild in women with no prior history of dysplasia, and they originated equally in the cervix and vagina. Severe dysplasia and carcinoma in situ were encountered only in those subjects specifically referred because of these abnormalities. The most common problem in diagnosing the squamous cell changes was the misinterpretation of mature and immature metaplastic cells for dysplastic squamous cells. Discordance between biopsy and cytology was commonplace in the detection and follow-up of dysplasia, especially mild forms. There were no instances in which the cytology and biopsy samples from the vagina were both abnormal concurrently. There were five instances in which the initial cervical biopsy specimen and scrape smear were both abnormal. Colposcopically directed biopsies did not increase the frequency of confirmation of cytologic findings. These findings suggest that both cytology and biopsy of abnormal segments of the vagina and cervix remain an integral part of the examination of DES-exposed women. (27 refs)

78-3793 **Angiosarcoma of the Liver Associated with Diethylstilbestrol.** (Eng) Hoch-Ligeti, C. (Registry Experimental Cancer, NCI, Del Ray Bldg., Bethesda, MD, 20014). *JAMA* 240(14): 1510-1511; 1978.

Angiosarcoma of the liver occurred in a 76-yr-old man who had been treated for 13 yr with diethylstilbestrol (3 mg/day) for a well-differentiated adenocarcinoma of the liver. The angiosarcoma was also present in pulmonary and renal arteries. Since in many smaller pulmonary arteries the lumen was completely filled by tumor and surrounding tissue was unaffected, the intima apparently represented a primary site. (8 refs)

78-3794 **Squamous Cell Carcinoma In Situ of the Vagina and Cervix after Intrauterine DES Exposure.** (Eng) Veridiano, N. P. (Dept. Obstetrics and Gynecology, Brookdale Hosp. Medical Center, Linden Blvd. at Brookdale Plaza, Brooklyn, NY, 11212); Tancer, M. L.; Weiner, E. A. *Obstet Gynecol* 52(1, Suppl): 30s-33s; 1978.

Squamous cell carcinoma in situ (CIS) of the cervix and vagina arose in areas of squamous metaplasia that replaced areas of adenosis in two women (20 and 23 yr old) exposed to diethylstilbestrol in utero. Their case reports are presented, along with a summary of data on 201 patients with a history of in utero DES exposure that are currently under observation. Adenosis was detected in 174 patients. In addition to the 2 CIS patients, 15 patients had mild or moderate dysplasia (6, cervix alone; 9, cervix + vagina), and 2 had severe dysplasia of the cervix and vagina. Thus, 19 patients with cervical and/or vaginal intraepithelial neoplasia were seen among 203 patients exposed to DES in utero. However, in only 50% of these 19 patients did a Papanicolaou smear reveal abnormal cells. There appears to be a strong possibility that there is a continuous process beginning with DES exposure and proceeding through the occurrence of adenosis and active squamous metaplasia to dysplastic alteration and, finally, squamous neoplasia. (3 refs)

78-3795 **Stilboestrol and Vaginal Clear-Cell Adenocarcinoma Syndrome.** (Eng) Monaghan, J. M. (Dept. Gynaecological Oncology, Queen Elizabeth Hosp., Gateshead, Tyne and Wear NE9 6SX, England); Sirisena, L. A. *Br Med J* 1(6127): 1588-1590; 1978.

The development of a vaginal clear-cell adenocarcinoma in a 25-yr-old woman who had been exposed in utero to maternal stilbestrol treatment is described. Although this is the first such case to be reported in the UK, 250 cases have been recorded in the literature since 1966. In every case for which a detailed prenatal history was available, stilbestrol had been administered before the 18th week of gestation. The primary presenting symptom was abnormal bleeding or bloodstained intermenstrual discharge, but 28 patients were asymptomatic. The patients ranged in age from 7 to 29 yr, with a peak incidence around 18 yr. Between 1940 and 1971, approximately 7,500 women in the UK were given stilbestrol during pregnancy. In reference to these figures and US registry data, there may be at least 10 latent cases of vaginal adenocarcinoma. The best methods for screening for presymptomatic carcinoma are visualization of the vagina and cervix, cytology of both the vaginal aspirate and cervical scrape, iodine staining, and colposcopy with biopsy of suspicious areas. (3 refs)

78-3796 **Relations Between Bleeding Pattern, Endometrial Histology, and Oestrogen Treatment in Menopausal Women.** (Eng) Sturdee, D. W. (Walsgrave Hosp., Coventry CV2 2DX, England); Wade-Evans, T.; Paterson, M. E.; Thom, M.; Studd, J. W. *Br Med J* 1(6127): 1575-1577; 1978.

The effects of hormone treatment on the endometrium were investigated in 286 English women who had received various regimens of estrogen treatment for an av of 9.7 mo for climacteric symptoms. Two hundred and fifty-seven women had a normal endometrium, 21 had cystic hyperplasia, 7 had adenomatous hyperplasia, and 1 had endometrial adenocarcinoma. The incidence of endometrial hyperplasia was 12% among the 98 women given cyclical unopposed po estrogen treatment, 8% among the 37 patients given an additional 5-day course of progestogen in each cycle, 28% among the 50 women treated with sc estradiol implants and monthly 5-day courses of po progestogen, and 0% among the 102 women on regimens that included 10 or 13 days of progestogen per cycle. Regular withdrawal bleeding during treatment was associated with a lower incidence of endometrial hyperplasia (6%) than unscheduled breakthrough bleeding (28%), but the one patient with carcinoma had experienced only regular bleeding. The risk of developing endometrial carcinoma from estrogen treatment may be reduced by avoiding the use of unopposed estrogen regimens, the addition of > 5 days' treatment with a progestogen, and the recognition that a regular bleeding response to estrogen is no guarantee of a healthy endometrium. (16 refs)

78-3797 **Exposure of Pregnant Mice to Hormones: Effects on the Reproductive Cycles and Organs in Their Female Offspring.** (Eng) Mori, T. (Zoological Inst., Faculty Science, Univ. Tokyo, Bunkyo-ku, Hongo, Tokyo 113, Japan); Bern, H. A.; Mills, K. T. *ICRS Med Sci (Cancer)* 6(7): 275; 1978.

The effect of a single injection of 500 μg estradiol-17β on the offspring of BALB/cfC3H/Crgl and C3H/Tw mice was determined. The former received the hormone on day 15 of pregnancy, the latter on day 12 or 15. Two of 8 BALB/c mice had vaginal lesions by 1 yr, compared with 0/11 controls. In C3H mice, exposure on day 15 resulted in abnormal reproductive cycles and abnormalities in the cervicovaginal epithelium of 3/12 mice, vs 0/8 mice exposed in utero on day 12 and 0/15 controls. It is not known whether the genital tract changes were ovary-dependent or -independent. (5 refs)

78-3798 **Hepatocellular Carcinoma in Young Women on Oral Contraceptives (Letter to Editor).** (Eng) Christopherson, W. M. (Dept. Pathology, Univ. Louisville Sch. Medicine, Louisville, KY, 40201); Mays, E. T.; Barrows, G. *Lancet* 2(8079): 38-39; 1978.

Twenty cases of hepatocellular carcinoma listed in one Kentucky tumor registry are reported. Seventeen of the 20 patients, all women aged 17-46 yr, had taken oral contraceptives for 6-120 mo (av 57 mo). Eleven of the 15 that died had widespread metastases. None of the 20 patients had cirrhosis or fibrosis. (no refs)

78-3799 **Hyperplasia and Prolapse of Hepatocytes into Hepatic Veins During Longterm Methyltestosterone Therapy: Possible Relationships of these Changes to the Development of Peliosis Hepatis and Liver Tumours.** (Eng) Paradinas, F. J. (Dept. of Histopathology, Charing Cross Hosp. and Medical Sch., London, England); Bull, T. B.; Westaby, D.; Murray-Lyon, I. M. *Histopathology* 1(4): 225-246; 1977.

Pathological changes in liver biopsy specimens from 10 patients (4 female transsexuals and 6 impotent males) after they were treated with 150 mg methyltestosterone daily for periods of up to 3 yr and in a hemihepatectomy specimen from a female transsexual who developed a liver adenoma after 37 mo of this treatment are reported. Hepatocyte hyperplasia and mild focal sinusoidal dilatation were found in most cases. In some there were microcysts and a dissociation of hepatocytes. In nine patients, there was an accumulation of hepatocytes between the endothelium and the supporting collagen of hepatic veins that often resulted in partial occlusion of their lumina or disruption of their walls. These vascular changes do not appear to have been reported before. The av length of treatment associated with the development of peliosis was considerably shorter than that associated with tumor development. It is postulated that a single pathological process--hyperplasia--which may be related to the anabolic effect of methyltestosterone, could be partly responsible for the formation of cysts through mechanical obstruction of hepatic veins and for the formation of nodules and tumors. (55 refs)

78-3800 **The Existence of a Benzo(a)pyrene-3,6-quinone Reductase in Rat Liver Microsomal Fractions.** (Eng) Capdevila, J. (Dept. Biochemistry, Univ. Texas Health Science Center at Dallas, 5323 Harry Hines Blvd., Dallas, TX, 75235); Estabrook, R. W.; Prough, R. A. *Biochem Biophys Res Commun* 83(4): 1291-1298; 1978.

Analysis of repetitive-scan difference spectra of incubation mixtures containing liver microsomes from phenobarbital-pretreated rats, benzo(a)pyrene 3,6-quinone (BP 3,6-quinone), and NADPH revealed the rapid reduction of the quinone to a steady-state level of hydroquinone and the subsequent reoxidation of the hydroquinone. This cyclic process results in NADPH oxidation coupled to oxygen reduction and hydrogen peroxide formation. The reduction of the BP 3,6-quinone was not supported by NADPH. The initial rate of the NADPH-supported reaction was inhibited by NADPH+, metyrapone, and anti-NADPH-cytochrome P-450 (*c*) reductase globulin, but not by dicumarol, anaerobiosis, or a gas mixture of carbon monoxide and oxygen (4:1, volume/volume). These results suggest that cytochrome P-450 and its reductase are involved in this reaction. During the steady-state metabolism of BP, the 3,6-quinone is present largely as a diol that may be conjugated to form water-soluble products and excreted. In addition, the reactive oxygen intermediates and semiquinone free radicals formed may have toxic properties or be capable of altering genome function and/or structure. (15 refs)

78-3801 **The Action of Two Sulfhydryl Compounds on the Induction of Sister Chromatid Exchanges (SCEs) by Trenimon in Human Lymphocytes (Meeting Abstract).** (Eng) Gebhart, E. (Institut fur Humangenetik und Anthropologie der Universitat, Bismarckstr. 10, D-8520 Erlangen, W. Germany); Kappauf, H. *Mutat Res* 53(2): 190; 1978. (no refs)

78-3802 **Relation Between the Use of Artificial Sweeteners and Bladder Cancer (Letter to Editor).** (Eng) Connolly, J. G. (Ontario Cancer Treatment and Res. Foundation, Toronto, Ontario, Canada); Rider, W. D.; Rosenbaum, L.; Chapman, J. A. *Can Med Assoc J* 119(5): 408; 1978.

A survey of the environmental and occupational aspects of bladder cancer revealed that 58/341 patients and 130/673 controls used artificial sweeteners. Thus, there was no relation between bladder cancer and consumption of the sweeteners. (no refs)

78-3803 **Saccharin: An Epigenetic Carcinogen/Mutagen?** (Eng) Ashby, J. (Central Toxicology Labs., Imperial Chemical Industries Ltd., Alderley Park, Macclesfield, Cheshire, England); Styles, J. A.; Anderson, D.; Paton, D. *Food Cosmet Toxicol* 16(2): 95-103; 1978.

The carcinogenicity of saccharin (Sa), sodium saccharin (SSa), and 13 related compounds was evaluated in two in vitro tests, and the results were evaluated within the context of the ability of commercial Sa to cause bladder cancer in rats and dominant lethal effects in mice. Five doses (4-2,500 μg/plate) of the test compounds were used in the Salmonella assay. In the cell-transformation assay, baby hamster kidney cells in semisolid agar were exposed to six doses (0.025-2,500 μg/ml) of each compound. Except for 3-dimethylamino-ψ-saccharin (DMASa), all compounds were negative in all four Salmonella testers strains used. DMASa had a positive effect in TA98 and TA100, but these results could not be reproduced. All compounds were negative in the transformation assay and all had a similar low toxicity. The following hypotheses are proposed for the divergence between the in vitro and in vivo findings: (1) an undefined impurity in commercial Sa that is responsible for the carcinogenic effect is unable to produce positive effects in vitro because of its low concentration in the test material; (2) the electrophilic theory of DNA attack linked to the somatic mutation theory of cancer induction may not include all organic carcinogens (Sa is nucleophilic). Based on available evidence, it is suggested that the in vivo effects of commercial Sa are probably due to Sa per se and that they may arise by an epigenetic mechanism. Further, these effects may only be exhibited above a given threshold dose that may be much higher than the "safe" level suggested by analogy with carcinogens operating via a genotypic mechanism. (60 refs)

78-3804 **Saccharin, Cyclamate, and Human Bladder Cancer. No Evidence of an Association.** (Eng) Kessler, I. I. (Dept. Epidemiology and Preventive Medicine, Univ. Maryland Sch. Medicine, 655 W. Baltimore St., Baltimore, MD, 21201); Clark, J. P. *JAMA* 240(4): 349-355; 1978.

The results of a hospital-based case-control study of the possible roles of saccharine and cyclamate in human urinary bladder cancer are reported. The study groups consisted of 519 patients with histopathologically confimed bladder cancer and 519 matched controls (cancer-free patients) from metropolitan Baltimore. All subjects were subjected to an intensive personal interview with respect to smoking habits, occupational history, and use of nonnutritive sweeteners (NNS). The proportions of patients and controls who ever used NNS in any form were the same (39.5%), and the frequency, quantity, or duration of intake of these substances did not differ significantly between the two groups. These normal findings persisted after simultaneous adjustment for the effects of smoking, occupation, age, diabetes mellitus, and several other potentially confounding factors. The failure of the relative risk of bladder cancer to increase with increasing NNS exposure is also inconsistent with an etiologic relationship. However, cyclamates were introduced too recently to permit the detection of carcinogenic effects appearing more than 10 or 15 yr after initial exposure. It is concluded that neither saccharin nor cyclamate is likely to be carcinogenic in man, at least at the moderate dietary levels reported by the patient sample. (18 refs)

78-3805 **Saccharin-induced Sister Chromatid Exchanges in Chinese Hamster and Human Cells.** (Eng) Wolff, S. (Lab. Radiology, Univ. California, San Francisco, CA, 94143); Rodin, B. *Science* 200(4341): 543-545; 1978.

The ability of the sodium salt of saccharin (lot S1022) and purified saccharin to induce sister chromatid exchanges (SCE's) in cultured Chinese hamster ovary cells and human lymphocytes was investigated. In the ovary cells, no growth was noted in cells treated with ≥1.5% S1022 or with > 1.2% pure saccharin. In the lymphocytes, no growth was noted in cells treated with > 0.5% S1022 or pure saccharin. However, both batches of saccharin increased the yield of SCE's in both types of cells. These findings indicate that the weak carcinogen saccharin does induce cytogenetic changes in cultured cells. (14 refs)

78-3806 **Non-mutagenicity of Saccharin (Letter to Editor).** (Eng) Pool, B. (Inst. Toxicology and Chemotherapy, German Cancer Res. Center, Im Neuenheimer Feld 280, 69 Heidelberg, W. Germany). *Toxicology* 11(1): 95-97; 1978.

Assay of the mutagenicity of a relatively pure sample of sodium saccharin (< 10 ppm o-toluolsulfonamide) in *Salmonella typhimurium* TA1535, TA1537, TA98, and TA100 produced negative results. Previous positive responses may have been due to impurities. (10 refs)

78-3807 **The Effect of Chronic Alcohol Intake upon the Hepatic Microsomal Carcinogen-Activation System.** (Eng) Capel, I. D. (Res. Dept., Marie Curie Memorial Foundation, The Chart, Oxted, Surrey RH8 0TL, England); Jenner, M.; Pinnock, M. H.; Williams, D. C. *Oncology* 35(4): 168-170; 1978.

The effect of chronic ethanol (EtOH) administration on the activation and subsequent DNA binding of benzo(a)pyrene (BP) was investigated in TO mice. The effect of EtOH pretreatment on the development of BP-induced tumors was also investigated. Males and females were inoculated ip with 0.2 ml of an aqueous EtOH soln at doses of 0.7, 1.2, 1.5, or 1.8 mg/g on 7 consecutive days. Two days later, the mice were sacrificed and their hepatic microsomes were assayed (Group 1) or they were inoculated ip with 1 mg BP (Group 2). The latter were sacrificed 20 wk later. The third group of males was given 10% (volume/volume) distilled EtOH in the drinking water and sacrificed after 2, 4, or 8 wk. Cytochrome P-450 and aryl hydrocarbon hydroxylase (AHH) levels were reduced in Group 1 mice. Microsomal protein was also significantly reduced at all EtOH concentrations except 1.2 mg/g. In Group 3, the level of microsomal protein decreased initially but after 2 wk it increased until at 8 wk it had recovered to control levels. The cytochrome P-450 level was raised significantly in all Group 3 animals. In both Groups 1 and 3, the binding of BP to DNA was reduced significantly, corresponding to the decrease in AHH activity. Mice pretreated with EtOH and given BP developed more tumors than mice given BP only (8/77 mice vs 1/42). The tumors in the pretreated animals originated in the muscle tissue, but the one that developed in the control group was a mammary tumor. It is hypothesized that the in vivo excretion of carcinogens, which is reduced in animals with EtOH-induced liver damage, is a more important factor in tumor development than the reduction of activation and binding of BP observed in vitro. However, DNA repair mechanisms might also be adversely affected by chronic alcohol intake. (15 refs)

78-3808 **The Effect of Long Term Ethanol Intake on the Hepatic Carcinogen Metabolising Enzyme Systems of Mice.** (Eng) Capel, I. D. (Res. Dept., Marie Curie Memorial Foundation, Chart, Oxted, Surrey RII8 0TL, England); Williams, D. C. *ICRS Med Sci (Cancer)* 6(7): 297; 1978.

Male TO mice were maintained on a diet containing 1% or 10% ethanol for up to 24 and 32 wk, respectively. Both diets increased the microsomal protein and cytochrome P_{450} levels. The lower ethanol concentration induced aryl hydrocarbon hydroxylase (AHH) activity and increased the DNA binding of benzo(a)pyrene (BP). The higher concentrations resulted in an initial decrease in AHH levels followed by an increase to values greater than controls; BP binding was decreased, and it recovered to control levels only after 32 wk. (8 refs)

78-3809 **Nutrition, Alcohol, and Esophageal Cancer.** (Fre) Tuyns, A. J. (Centre International de Recherche sur le Cancer, 150, cours Albert-Thomas, F69372 Lyon Cedex 2, France); Pequignot, G.; Jensen, O. M. *Bull Cancer (Paris)* 65(1): 59-64; 1978.

A study in Brittany, France, compared diet and alcohol and tobacco consumption in a group of 200 men with esophageal cancer with those controls. No major deficiencies were found, either in quantity or quality, in the diet of the cancer group. A definite correlation was observed between alcohol consumption and the incidence of esophageal cancer. Smokers also had a higher incidence of this cancer. A dose-response relationship existed independently for alcohol and tobacco and the incidence of cancer; however, the use of both had a cumulative effect on incidence. Surveys of the role of dietary factors in esophageal cancer in other areas, such as Iran, are reviewed. (24 refs)

78-3810 **Effect of Ethanol and Vinyl Chloride on the Induction of Liver Tumors: Preliminary Report.** (Eng) Radike, M. J. (Univ. Cincinnati, Inst. Environmental Health, Kettering Lab., Cincinnati, OH, 45267); Stemmer,

K. L.; Brown, P. G.; Larson, E.; Bingham, E. *Environ Health Perspect* 21: 153-155; 1977.

A preliminary report of the effect of ethanol on vinyl chloride (VC) carcinogenicity in 320 male Sprague-Dawley rats is presented. Equal numbers of rats were divided into four groups. Group 1 received a normal diet and breathed filtered air for life. Group 2 received a normal diet plus 5% ethanol in the drinking water and breathed filtered air for life. Group 3 received a normal diet but breathed 600 ppm VC 4 hr/day, 5 days/wk, for 1 yr. Group 4 received a normal diet plus ethanol starting 4 wk before VC exposure. The ethanol exposure lasted until the animal died or was sacrificed. Sixty weeks after the first exposure to VC, 55 rats had died or had been sacrificied. No tumors were noted in eight Group 1 animals that had died. Of six dead Group 2 animals, one had a kidney tumor. Of 13 dead Group 3 animals, 5 had liver tumors (2 of them being angiosarcomas) and 2 had lung tumors. Of 28 dead Group 4 animals, 21 had 26 liver tumors (5 angiosarcomas) and 2 had kidney tumors. These preliminary findings suggest a synergism between VC inhalation and ingested alcohol in tumorigenesis. (6 refs)

78-3811 **Development of Hepatic Angiosarcoma in Man Induced by Vinyl Chloride, Thorotrast, and Arsenic. Comparison with Cases of Unknown Etiology.** (Eng) Popper, H. (Stratton Lab. Liver Disease, Mount Sinai Sch. Medicine, City Univ. New York, New York, NY); Thomas, L. B.; Telles, N. C.; Falk, H.; Selikoff, I. J. *Am J Pathol* 92(2): 349-376; 1978.

Human hepatic angiosarcomas that occurred following exposure to vinyl chloride, Thorotrast, or arsenic (medicinal and industrial) and cases, including those in children, of unknown etiology were studied to establish diagnostic criteria and evolutionary features. The uniform evolution suggests that there is also an environmental factor in the cases of unknown etiology. The precursor stage is characterized by areas of combined hyperplasia of hepatocytes and a variety of sinusoidal and perisinusoidal cells associated with an excess of reticulin and with sinusoidal dilatation. Silver stainings indicated reticulum formation by the perisinusoidal cells, presumably the lipocytes. The hepatocytic proliferation suggested a hepatocarcinogenic, but usually not fully expressed, potential. The mixed hyperplasia of the various sinusoidal cells proceeded to an overgrowth of angiosarcoma cells, presumably derived from endothelial cells. In early stages they were usually in contact with hepatocyte (intralobular growth). A trabecular arrangement resulted from loosening of the lobular plate arrangement by dilatation of sinusoids, leading to primary peliosis. With disappearance of the hepatocytes, various growth patterns developed, terminating in nodular, solid angiosarcoma composed of either spindle-shaped or polyhedral cells that underwent necrosis or hemorrhage (secondary peliosis). The interaction between hepatocytes and sinusoidal cells requires elucidation. (110 refs)

78-3812 **Acute Exposure to VCM: a Cytogenetic Study of Two Workers (Meeting Abstract).** (Eng) Hansteen, I. L. (St. Josephs Hosp., Porsgrunn, Norway). *Mutat Res* 53(2): 196; 1978. (no refs)

78-3813 **Effects of Vinyl Chloride Exposure Alone and in Combination with Trypan Blue--Applied Systematically During All Thirds of Pregnancy--on the Fetuses of CFY Rats.** (Eng) Ungvary, G. (Dept. Experimental Pathology, State Inst. Occupational Health Budapest, H-1450 Budapest P.O.B. 22, Hungary); Hudak, A.; Tatrai, E.; Lorincz, M.; Folly, G. *Toxicology* 11(1): 45-54; 1978.

The teratogenic and embryotoxic effects of vinyl chloride and/or trypan blue were investigated in CFY rats. VC was found in the fetal and maternal blood as well as in the amniotic fluid of pregnant CFY rats exposed to an atomospheric concentration of 5,500, 18,000, or 33,000 mg/m^3 VC for 2.5 hr on gestation day 18, indicating the permeability of the placenta to the agent. Investigation of the offspring of pregnant rats exposed continuously to 4,000 mg/m^3 VC during the first, second, or last trimester showed that VC has no teratological or embryotoxic effects when applied during the second or last trimester. However, exposure to VC during the first trimester resulted in an increased fetal mortality and in the manifestation of embryotoxic effects. Fetal losses and the induction of CNS malformations due to trypan blue (50 mg/kg sc on gestation days 7 and 8) were not potentiated by 4,000 mg/m^3 VC. Occupational exposure of women of childbearing age may be hazardous. (34 refs)

78-3814 **Vinyl Chloride and Pulmonary Cancer (Letter to Editor).** (Eng) Wagoner, J. K. (Office Assistant Secretary for OSHA, 200 Constitution Ave., Room N3660, Washington, DC, 20210). *J Environ Pathol Toxicol* 1(3): 361-362; 1978.

In a previous article, the development of an undifferentiated adenocarcinoma of the bronchus in a 45-yr-old organic chemist was attributed to a 2-yr exposure to bis(chloromethyl)ether. This claim was made in spite of the patient's long-term involvement in vinyl chloride (VC) research. Since numerous animal and human studies have indicated a link between VC exposure and pulmonary cancer, it is suggested that VC was the agent that caused the tumor. (10 refs)

78-3815 **Study of the Teratogenic and Embryotoxic Effects of Vinyl Chloride in CFY Rats.** (Hun) Ungvary, Gy. (Orszagos Munka- es Uzemegeszsegtani Intezet, Budapest, Hungary); Hudak, A.; Tatrai, E.; Lorincz, M.; Folly, G. *Egeszsegtudomany* 21(4): 363-369; 1977.

Inhalation of vinyl chloride (4,000 mg/m^3) between the 8th and 14th days of pregnancy increased the absolute and relative wt of the liver of pregnant CFY rats significantly, but there was no significant increase in resorption, embryonal mortality, and developmental anomalies. In addition, vinyl chloride was found in the maternal and fetal blood and amniotic fluid following exposure of the mothers to 5,500-33,000 mg/m^3 vinyl chloride for 2.5 hr on the 18th day of pregnancy. (31 refs)

78-3816 **Pharmacokinetics of Vinylidene Chloride in the Rat.** (Eng) McKenna, M. J. (Toxicology Res. Lab., Health and Environmental Res., Dow Chemical Co., Midland, MI, 48640); Watanabe, P. G.; Gehring, P. J. *Environ Health Perspect* 21: 99-105; 1977.

The effects of inhaled vinylidene chloride (VDC; 1,1-dichloroethylene) were determined in rats and mice. In maleSprague-Dawley fasted and fed rats exposed to either 10 or 200 ppm VDC for 6 hr, fasting augmented the process whereby reactive intermediates formed from VDC induced tissue damage. Rats exposed to 200 ppm labeled VDC exhaled a greater percentage of their acquired body burden of radioactivity as unchanged VDC than did animals exposed to 10 ppm. However, rats exposed to the higher concentration showed a greater percentage of body burden remaining in the carcass at 72 hr after exposure. Retention of radioactivity in the carcass was greater in fasted rats exposed to 200 ppm VDC, despite a smaller fraction of the body burden biotransformed by fasted rats. The presence of mercapturic acid derivatives in the urine of exposed rats indicated a major role for glutathione in the detoxification of VDC. Apparently, VDC metabolism represents a balance between biotransformation pathways leading to detoxification by way of glutathione or to covalent binding to tissue nucleophiles and subsequent tissue damage. Enhanced susceptibility to VDC was noted in Ha(ICR) mice exposed to 10 ppm VDC for 6 hr, probably because of their more rapid metabolism of VDC and greater production of alkylating VDC metabolites over that in the rat. (9 refs)

78-3817 **Effect of Various Treatments on Toxicity of Inhaled Vinylidene Chloride.** (Eng) Short, R. D. (Pharmacology and Toxicology, Midwest Res. Inst., Kansas City, MO, 64110); Winston, J. M.; Minor, J. L.; Seifter, J.; Lee, C. C. *Environ Health Perspect* 21: 125-129; 1977.

The acute toxicity of continuously inhaled vinylidene chloride (VDC) was investigated in CD-1 mice and CD rats, and the effects of various treatments on this toxicity were determined. The animals were exposed to varying VDC concentrations for 22-23 hr/day. VDC was more lethal and more hepatotoxic mice than male rats. Furthermore, male mice were more sensitive to the lethal effects of VDC than females; the respective concentrations causing 50% mortality were 98 and 105 ppm. The effects of disulfiram, diethyldithiocarbamate, thiram, cysteine, methionine, N-acetylcysteine, SKF 525-A, cobaltous chloride, dimercaprol, phenoxybenzamine, propranolol, vitamin C, and DL-α-tocopherol acetate on VDC toxicity were evaluated. Disulfiram, diethyldithiocarbamate and thiram reduced the acute lethal and hepatoxic effects of inhaled VDC. Disulfiram also reduced the levels of covalently bound radioactivity in the liver and kidney following ip administration of ^{14}C-VDC. It is suggested that disulfiram and its metabolites act by not only reducing the activation of VDC, but also by increasing the extent of detoxification. (25 refs)

78-3818 **Damage to Hepatic Cellular Membranes by Chlorinated Olefins with Emphasis on Synergism and Antagonism.** (Eng) Reynolds, E. S. (Dept. Pathology, Univ. Texas Medical Branch, Galveston, TX, 77550, 77550); Moslen, M. T. *Environ Health Perspect* 21: 137-147; 1978.

The biochemical mechanisms responsible for the toxicity of vinyl chloride (VC), 1,1-dichloroethylene (1,1-DCE), trichloroethylene (TCE), and perchloroethylene (PCE) were investigated in male Sprague-Dawley rats pretreated with phenobarbital, 3-methylcholanthrene, hexachlorobenzene, spironolactone, or pregnenolone-16α-carbonitrile. The most nonsymmetrically depolarized compound, 1,1-DCE, was the most hepatotoxic and caused a unique pattern of hepatocellular injury involving mitochondria, plasma membranes, and chromatin. The injury induced by the other chloroethylenes appeared to affect the structural integrity of the endoplasmic reticulum profoundly, with the toxic potential in the following order: TCE > VC > PCE. Pretreatments that increased the cytochrome P-450 content (increased metabolic activation) enhanced or were synergistic to the hepatotoxic potential of TCE, VC, and PCE, but they were protective or antagonistic to 1,1-DCE hepatotoxicity. This suggests that the biologic response to 1,1-DCE may be expressed by a different metabolic pathway. Glutathione appears to be involved in the biologic response to all nonsymmetric chloroethylenes and to act as an antagonist against injury. Marked differences in the patterns of injury and the biologic responses suggest that more than one mechanism is involved in the production of injury by chloroethylenes. (37 refs)

78-3819 **Gas-Liquid Chromatographic Determination of Trichloroethylene Metabolites in Urine.** (Eng) Nomiyama, H. (Dept. Environmental Health, Jichi Medical Sch., Minamikawachi-machi, Tochigi-ken 329-04, Japan); Nomiyama, K.; Uchiki, H. *Am Ind Hyg Assoc J* 39(6): 506-510; 1978.

A gas-liquid chromatography technique using an electron capture detector was developed to determine all the urinary metabolites of trichloroethylene (TCE) on a single column. With urine from TCE-exposed rats, detection limits were as low as 0.1-5 nanograms. (10 refs)

78-3820 **Chemical Structure, Reactivity, and Carcinogenicity of Halohydrocarbons.** (Eng) Van Duuren, B. L. (Lab. of Organic Chemistry and Carcinogenesis, Inst. of Environmental Medicine, New York Univ. Medical Center, New York, NY, 10016). *Environ Health Perspect* 21: 17-23; 1977.

The structural and carcinogenic properties of halohydrocarbons are reviewed, with particular emphasis on trichloroethylene (TCE). Based on studies of the chemical structure, reactivity, and possible metabolic pathways of TCE, the compound was predicted to be carcinogenic, particularly to the liver. These studies also suggested that TCE is metabolized to an epoxide and that this epoxide may be the activated carcinogenic intermediate of TCE in liver carcinogenesis. The binding of TCE to liver microsomal proteins of male B6C3F1 hybrid mice, which are susceptible to TCE-induced liver tumorigenesis, was found to be significantly higher than the binding of TCE to microsomal proteins of male Osborne-Mendel rats, which are resistant to TCE-induced hepatocellular carcinoma. Also, the in vitro binding of TCE to liver microsomal proteins was higher for male than female B6C3F1 mice; females have been reported to show a lower incidence of TCE-induced hepatocellular carcinoma than males. The results of carcinogenicity assays of vinyl bromide and polyvinyl bromide are also given. Neither compound appeared to be carcinogenic when injected sc or when applied to the skin of mice. The carcinogenicity of other chlorinated hydrocarbons that are widely used in the chemical industry and that are structurally analogous to TCE and vinyl chloride is discussed briefly. (43 refs)

78-3821 **Biochemical and Toxicological Effects of Combined Exposure to 1,1,1-Trichloroethane and Trichloroethylene on Rat Liver and Brain.** (Eng) Vainio, H. (Dept. Industrial Hygiene and Toxicology, Inst. Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland); Savolainen, H.; Pfaffli, P. *Xenobiotica* 8(3): 191-196; 1978.

Male Wistar rats inhaled a mixture of 1,1,1-trichloroethane (1,1,1-TCE) (500 ppm) and trichloroethylene (200 ppm) 6 hr/day for 4 days. As a result, 1,1,1-TCE accumulated in the perirenal fat, the hepatic RNA content doubled, hepatic glutathione concentrations decreased slightly, and uridine diphosphate-glucuronosyl transferase activity doubled. A 5-day exposure increased the content of both solvents in various organs rapidly, depressed brain RNA, and decreased in styrene monooxygenase activity. Changes noted on day 5 had also occurred on days 1-4 but had reverted during each postexposure period. (22 refs)

78-3822 **Metabolism and Mutagenicity of Halogenated Olefins--A Comparison of Structure and Activity.** (Eng) Henschler, D. (Institut fur Toxikologie und Pharmakologie, Universitat Wurzburg, D-8700 Wurzburg, W. Germany). *Environ Health Perspect* 21: 61-64; 1977.

The metabolism of various halogenated olefins was studied in vivo, and their mutagenicity was investigated in vitro in a modified Ames testing system. The results of the mutagenicity test were compared with in vivo carcinogenicity findings reported previously. In mammals, chlorinated ethylenes are first metabolized to epoxides, which may then undergo intramolecular rearrangement. This reaction has been studied in the entire series of chlorinated epoxyethanes. The rearrangement products found were acyl chlorides (tetrachloro-, trichloro-, and 1,1-dichloroethylenes) or chlorinated aldehydes (cis- and trans-1,2-dichloroethylene, vinyl chloride). In vivo experiments yielded products that were further derivatives of these rearrangement products, except that with trichloroethylene, the only rearrangement product was chloral. Tetrachloroethylene, trichloroethylene, cis-1,2-dichloroethylene, trans-1,2-dichloroethylene, 1,1-dichloroethylene, and vinyl chloride were then tested for mutagenicity in the *Salmonella typhimurium* assay. Vinyl chloride, trichloroethylene, and 1,1-dichloroethylene were mutagenic, suggesting that asymmetric chlorine substitution renders the epoxides unstable and mutagenic. High electrophilicity may be a prerequisite for the mutagenic and carcinogenic activity of these chlorinated ethylenes. (20 refs)

78-3823 **Mutagenicity of the Halogenated Olefin, 2-Bromo-2-chloro-1,1-difluoroethylene, a Presumed Metabolite of the Inhalation Anesthetic Halothane.** (Eng) Garro, A. J. (Dept. Microbiology, Mount Sinai Sch. Medicine, City Univ. New York, New York, NY, 10029); Phillips, R. A. *Environ Health Perspect* 21: 65-69; 1977.

The mutagenicity of 2-bromo-2-chloro-1,1-difluoroethylene (BCD), a metabolite of the anesthetic halothane, was investigated in *Salmonella typhimurium* and *Bacillus subtilis*. BCD induced base and frameshift mutations in *S. typhimurium*. With *B. subtilis*, an increase in the number of mutants and inactivation of transforming activity were noted. The significance of these findings with respect to morbidity in anesthesia personnel is not known. (26 refs)

78-3824 **Formation of N-Nitrosamines in Cutting Fluids.** (Eng) Rappe, C. (Dept. Organic Chemistry, Univ. Umea, Umea, Sweden); Zingmark, P. A. *IARC Sci Publ* (19): 213-217; 1978.

Two grinding fluid concentrates containing triethanolamine, nitrite, and water were analyzed for N-nitrosodiethanolamine after storage for 5-7 mo. Between 400 and 800 ppm N-nitrosamine were formed, even though the pH values of the fluids were 10.2-11.4. These analyses clearly show that the mixing of nitrite and an amine in a technical product should always be regarded with suspicion concerning

N-nitrosamine formation, especially if the product is stored. (6 refs)

78-3825 **Mutagenicity Screening of Industrial Chemicals: Seven Aliphatic Amines and One Amide Tested in the Salmonella/Microsomal Assay (Meeting Abstract).** (Eng) Hedenstedt, A. (Environmental Toxicology Unit, Wallenberg Lab., Univ. Stockholm, Lilla Frescati, 104 05 Stockholm 50, Sweden). *Mutat Res* 53(2): 198-199; 1978. (no refs)

78-3826 **Genetic Activity of Allyl Chloride.** (Eng) McCoy, E. C. (Dept. Microbiology, New York Medical Coll., Valhalla, NY, 10595); Burrows, L.; Rosenkranz, H. S. *Mutat Res* 57(1): 11-15; 1978.

The mutagenic activity of allyl chloride (3-chloroprene) for *Salmonella typhimurium* TA100 and TA1535, but not TA1538, was demonstrated using an assay procedure that protected against evaporation of the volatile test substance. Allyl chloride also induced gene conversions in *Saccharomyces cerevisiae* and displayed DNA-modifying activity for *Escherichia coli*. These results are in contrast to a recent study that reported that allyl chloride was devoid of genetic activity for *S. typhimurium*, and they indicate that the volatility of the test agent is an important factor that must be taken into consideration. (30 refs)

78-3827 **Potential Mutagenic and Adverse Male Reproductive Effects of 1,2,3,4-Tetrabromobutane: A Dominant Lethal Study in the Rat.** (Eng) Simon, G. S. (Div. Toxicology, Dept. Pharmacology, Medical Coll. Virginia, Virginia Commonwealth Univ., Richmond, VA, 23298); Tardiff, R. G.; Borzelleca, J. F. *Toxicol Appl Pharmacol* 44(3): 661-664; 1978.

The results of a dominant lethal assay in which 1,2,3,4-tetrabromobutane (TBB) was given po to Sprague-Dawley rats (10 or 40 mg/kg/day x 10) are summarized. Statistically significant adverse effects that were not dose-related were induced by both doses of TBB during weeks 6-13. The data suggest that TBB is not a potent mutagen but that it may affect male reproductive function and performance adversely. (3 refs)

78-3828 **Influence of Solvents in the Salmonella Microsome Assay (Meeting Abstract).** (Eng) Hermann, M. (Laboratoire de Toxicologie Genetique, Institut Pasteur, Paris, France); Bedouelles, H.; Hofnung, M. *Mutat Res* 53(2): 199; 1978. (no refs)

78-3829 **Dose-Effect Relationship in Mutagenicity Study in Rats after Acute and Chronic Exposure to a Dithiocarbamate (Meeting Abstract).** (Eng) Vachkova-Petrova, R. (Inst. Hygiene, Sofia, Bulgaria). *Mutat Res* 53(2): 277-278; 1978. (no refs)

78-3830 **Chromosome Investigations of Persons Exposed to Dimethylcarbamoyl Chloride and Diethylcarbamoyl Chloride (Meeting Abstract).** (Eng) Fleig, I. (Arbeitsmedizin und Gesundheitsschutz, BASF, Aktiengesellschaft, 6700 Ludwigshafen, W. Germany); Thiess, A. M. *Mutat Res* 53(2): 188; 1978. (no refs)

78-3831 **Effect of Reovirus Infection on Pulmonary Tumor Response to Urethan in Strain A Mice.** (Eng) Theiss, J. C. (Dept. Community Medicine, M-014, Sch. Medicine, Univ. California at San Diego, La Jolla, CA, 92093); Stoner, G. D.; Kniazeff, A. J. *J Natl Cancer Inst* 61(1): 131-134; 1978.

The effect of reovirus type 3 infection on the pulmonary adenoma response to urethan in strain A mice was examined. Mice were given low (0.25 mg/g), medium (0.5 mg/g), or high (1.0 mg/g) doses of urethan and exposed to reovirus either 6 days before, on the same day as, or 14 days after urethan administration. At all doses of urethan and at all times of reovirus exposure, the lung tumor response was significantly (30%-60%) less than the response obtained with urethan alone. In another experiment, mice were given 1.0 mg/g urethan and exposed to reovirus four times: 7 days before, on the same day as, 7 days after, and 14 days after urethan administration. Multiple exposure to reovirus enhanced the pulmonary adenoma response to urethan. A single exposure of strain A mice to reovirus may increase the capacity of the immune system by a rebound effect, thus suppressing lung tumor formation in response to urethan, whereas multiple exposure causes a continued suppression of the immune system, resulting in an enhancement of the pulmonary adenoma response to urethan. Virus infection may be important in the carcinogenic response of humans to environmental chemicals. (26 refs)

78-3832 **Carcinogenic Effects of Niridazole in Rats.** (Eng) Bulay, O. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB, 68105); Clayson, D. B.; Shubik, P. *Cancer Lett* 4(6): 305-310; 1978.

The carcinogenicity of the drug niridazole [NR: 1-(5-nitro-2-thiazolyl)-2-imidazolidinone]was studied in MRC rats. The rats were chronically fed levels of 0.1%, 0.05%, 0.025%, and 0% NR in the diet for 50-140 wk. Mean life-spans were 47, 70, 80, and 102 wk, respectively. Treated females lived an av of 20 wk longer than treated males. NR primarily induced kidney and forestomach tumors. Kidney tumors developed in a higher percentage of males than females, and they were dose-dependent for those sexes. Histologically, the kidney tumors were primarily tubular, solid, and papillary adenomas; adenocarcinomas; and mesenchymal tumors. Controls did not develop kidney tumors. The stomach tumors were all papillomas. Their induction was also dose-dependent, and no major difference was observed between males and females. At the lowest concentration, stomach tumor incidence in controls and in treated animals was similar. Tumor latency was shortest (21 wk) in males treated with the highest dose. (15 refs)

78-3833 **Report on Bioassay of 2-Amino-5-nitrothiazole for Possible Carcinogenicity.** (Eng) Fredrickson, D. S. (NIH, Bethesda, MD, 20014). *Fed Register* 43(143): 32191; 1978.

2-Amino-5-nitrothiazole (ANT) was administered to Fischer 344 rats (300 or 600 ppm diet for 100 wk) and B6C3F1 mice (50 or 100 ppm diet for 104 wk), and tumor formation was monitored. Tumors of the hematopoietic system (lymphomas, granulocytic leukemia) occurred in male rats, but the compound was not carcinogenic in female rats and in the mice. (no refs)

78-3834 **Comparison Between Radiation and Chemicals in the Time of Formation of Chromosome Aberrations Induced in Male Germ Cells of Mice (Meeting Abstract).** (Eng) Generoso, W. M. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN); Cain, K. T.; Huff, S. W.; Krishna, M. *Mutat Res* 53(2): 191; 1978. (no refs)

78-3835 **High Incidence of Hepatic Tumours in Rats Fed Mouldy Rice Contaminated with *Aspergillus versicolor* Containing Sterigmatocystin.** (Eng) Ohtsubo, K. (Dept. Carcinogenesis and Cancer Susceptibility, Inst. Medical Science, Univ. Tokyo, Shirokanedai, Minato-ku, Tokyo 108, Japan); Saito, M.; Kimura, H.; Tsuruta, O. *Food Cosmet Toxicol* 16(2): 143-149; 1978.

The hepatocarcinogenicity of rice contaminated with *Aspergillis versicolor*, a mold that produces the mycotoxin sterigmatocystin (SMC), was investigated in feeding trials with male Donryu rats. A strain of *A. versicolor* isolated from soya beans was injected into minimally polished rice, cultured, pelleted, and mixed with a basal diet. Three groups of 20 rats each were fed a diet containing 0, 5, or 10 ppm SMC for up to 709 days, when all survivors were killed. The doses of SMC were equivalent to a daily intake of 75 or 150 μg/rat, the lowest levels ever investigated. Of the 13 rats in each test group that died between day 465 (when the first animal with a tumor died) and day 709, 12 of those fed 10 ppm SMC and 11 fed 5 ppm developed hepatic tumors, mostly hepatocellular carcinomas. Three hemangiosarcomas were also observed. All of the tumor bearers had hyperplastic nodes and areas, and one-fourth had multiple pulmonary metastases. Non-tumor-bearing SMC-treated animals that died between days 209 and 435 had areas of hyperplasia only or hyperplastic areas and nodes. It is concluded that SMC is a more potent carcinogen than was previously thought, having nearly the same degree of carcinogenicity as aflatoxin B_1. (27 refs)

78-3836 **Sex and Age Differences in Luteoskyrin Hepatotoxicity.** (Eng) Ueno, I. (Inst. Medical Sciences, Univ. Tokyo, Shirogane-Dai, Minato-ku, Tokyo, Japan). *Ann Nutr Aliment* 31(4/5/6): 789-802; 1977.

Experiments correlating luteoskyrin (LS) accumulation in mouse liver with its toxicity are reported. LS, a hepatotoxic and hepatocarcinogenic mycotoxin from the fungus of yellowed rice (*Penicillium islandicum* Sopp), showed different toxicities depending on the age and sex of the mice. The LD_{50} of sc administered LS was lowest in newborn mice (7.2 mg/kg for males and 6.3 mg/kg for females), and it increased with age to 154 mg/kg for the male and 2 g/kg for the female. When 7.1 μg/g of radiolabeled LS was injected sc into suckling (2-wk-old) and adult (10-wk-old) mice, the radioactivity in the livers of suckling mice was several times higher than that in adult livers. When 0.43 mg of radiolabeled LS was given po to mice of both sexes, the percentage of radioactivity in the liver was higher in male than in female mice (more than double 23 hr after administration). This was also the case when a high dose of 2.52 mg was given po. In all mice, the specific radioactivity of the mitochondria was highest among the subcellular fractions of the liver. LS injected iv was excreted via the feces more rapidly in female than in male mice. These results showed that LS accumulated in the livers of suckling or male mice in larger amounts than in livers of adult or female mice and that this difference in accumulation was correlated with hepatotoxicity. The sex difference in the accumulation of LS in mouse liver is due to the different capacities of biliary excretion of this mycotoxin between the two sexes. A previous long-term feeding experiment demonstrated that the hepatocarcinogenicity of LS was higher in male than in female mice; therefore, the sex difference in the biliary excretion of LS may also have influenced its hepatocarcinogenicity. (34 refs)

78-3837 **Specific and Non-Specific Interactions of Two Carcinogenic Mycotoxins, Luteoskyrin and Rugulosin with Nucleic Acids.** (Eng) Bouhet, J. C. (Service de Biochimie, C.E.N. Saclay, B.P. n.2, 91190, Gif-sur-Yvette, France); Van Chuong, P. P. *Ann Nutr Aliment* 31(4/5/6): 811-830; 1977.

Specific and nonspecific interactions of the stereoisomeric and carcinogenic anthraquinonelike pigments luteoskyrin and rugulosin with nucleic acids are reported. Studies were performed with binary polychelates (pigment-metal) and ternary complexes (pigment-metal-nucleic acids). An unspecific complex with double-stranded polynucleotides resulted from simple adsorption of the polychelate onto the double helix of DNA, whereas a complex specific for single-stranded DNA (or polypurine nucleotides) resulted from the electrostatic association of separate pigment-metal units with the polyphosphate chain. Additional stabilization was obtained by hydrophobic interaction between stacked aromatic moieties of pigment and purine units. A model of interaction is proposed for both of the complexes. (26 refs)

78-3838 **Long-Term Testing of Patulin Administered Orally to Sprague-Dawley Rats and Swiss Mice.** (Eng) Osswald, H. (Institut fur Toxikologie und Chemotherapie, Deutsches Krebsforschungszentrum, Heidelberg, W. Germany); Frank, H. K.; Komitowski, D.; Winter, H. *Food Cosmet Toxicol* 16(3): 243-247; 1978.

Administration of patulin to female Sprague-Dawley rats for 64 wk and to pregnant Swiss mice on gestation days 14-19 did not produce a carcinogenic effect in the rats or in the offspring of the mice. However, 11/51 female and 8/43 male neonates died, all showing similar signs of toxicity. (no refs)

78-3839 **Tumorigenic Effect of 1-Hydrazinophthalazine Hydrochloride in Mice.** (Eng) Toth, B. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42d St. and Dewey Ave., Omaha, NB, 68105). *J Natl Cancer Inst* 61(5): 1363-1365; 1978.

The toxicity and carcinogenicity of 1-hydrazinophthalazine hydrochloride (HPH), a widely used hypertensive drug, were studied in Swiss albino mice. In the toxicity study, the mice were given five dose levels (wt/volume) of HPH (1%, 0.5%, 0.25%, 0.125%, and 0.0625%) in the drinking water for 35 days. On the basis of survival rates, body wt, chemical consumption levels, and histological changes, the 0.125% dose was found suitable for lifelong treatment. This dose in drinking water was then given to 100 6-wk-old mice over their life-spans. Av daily consumption of HPH was 5.4 mg for the females and 7.4 mg for the males. All animals were checked and weighed at weekly intervals, and the gross pathology was noted. Treated animals were allowed to die or were killed when found in poor condition; a sex-matched control was also killed. Thirty treated females developed 81 lung tumors (adenomas and adenocarcinomas) and 23 treated males developed 58 lung tumors. Of the untreated mice, 18 females developed 20 lung tumors and 13 males developed 16. Av age at death was 78 wk for treated females, 79 wk for treated males, 78 wk for untreated females, and 75 wk for untreated males. A few other types of tumors, such as malignant lymphomas and angiosarcomas of the liver, were found in the treate mice, but their incidence was not considered significant. (2 refs)

78-3840 **Detection of Mutagenicity of the Colon Car cinogen 1,2-Dimethylhydrazine by the Host-me diated Assay and its Correlation to Carcinogenicity.** (Eng Moriya, M. (Toxicology Div., Inst. Environmental Toxicology, Suzuki-cho 2-772, Kodaira-shi, Tokyo 187, Japan); Kato, K.; Ohta, T.; Watanabe, K.; Watanabe, Y.; Shirasu, Y. *J Nat Cancer Inst* 61(2): 457-460; 1978.

The mutagenicity of 1,2-dimethylhydrazine (DMH) was evaluated by a host-mediated assay in outbred ICR male mice, using *Salmonella typhimurium* G46 as the tester strain. The mice were given 2 ml G46 ip (2.2 x 10^8 cells/ml) followed immediately by DMH po (4.5, 11.25, 22.5, 33.75, or 45 mg/kg). A strong dose-response relationship was demonstrated in mutation induction by DMH. At 45 mg/kg, the mutation frequency was 900 times the control levels. Pretreatment of the mice for 8 days with disulfiram (DSF: 5-2 mg/kg/day po) markedly reduced the mutagenicity of DMH. At 20 mg/kg/day, mutation induction by 45 mg/kg DMH was reduced to 4% of the control value. The effects of mouse strain and sex on these results were also tested. Mutation induction was significantly lower in C57BL/6 mice than in ICR mice of either sex, and male mice showed a higher mutation rate than the females of each strain. (22 refs)

78-3841 **Inhibition of Testicular DNA Synthesis by Chemical Mutagens and Carcinogens as a Valid Mammalian Short-Term Testing Procedure (Meeting Abstract).** (Eng) Seiler, J. P. (Swiss Federal Res. Station, CH-8820 Waedenswil, Switzerland). *Mutat Res* 53(2): 260; 1978. (1 ref)

78-3842 **A Double-Blind Study on the Effect of Purified Cellulose Dietary Fiber on 1,2-Dimethylhydrazine-induced Rat Colonic Neoplasia.** (Eng) Freeman, H. J. (Gastrointestinal Res. Lab., Veteran's Admin. Hosp., 415 Clement St., San Francisco, CA, 94121); Spiller, G. A.; Kim, Y. S. *Cancer Res* 38(9): 2912-2917; 1978.

The incidence, distribution, size, and histopathology of grossly visible colonic tumors induced by parenteral administration of 1,2-dimethylhydrazine (DMH) were examined in 6-wk-old male Wistar rats. The rats were fed either a chemically defined fiber-free diet or a nutritionally and calorically equivalent diet containing a purified fiber component, microcrystalline cellulose. Twenty rats of each diet group were inoculated sc with DMH (25 mg/kg/wk x 16). Cellulose ingestion reduced the number of animals with colonic tumors and the total number of tumors. This protective effect of cel lulose appeared to be time-dependent and associated with a shift in tumor distribution from the proximal colon to a more distal site. Cellulose fiber had no apparent effect on colonic tumor size, histopathology, or the incidence of other tumors known to occur in this experimental animal model. This study strongly supports the hypothesis that fiber is an important protective agent against colonic neoplasia development. Although the mechanism for this protective effect remains obscure, it appears to be temporally related to the duration of fiber ingestion as well as to a differential fiber effect on either the luminal content or the mucosa of the proximal and distal colon. (27 refs)

78-3843 **Colonic Protection from Dimethylhydrazine by a High Fiber Diet.** (Eng) Chen, W. F. (Quakertown, PA); Patchefsky, A. S.; Goldsmith, H. S. *Surg Gynecol Obstet* 147(4): 503-506; 1978.

Protection from the carcinogenic effects of 1,2-dimethylhydrazine (DMH) on the colon afforded by a diet high in fiber bran was studied in female CF1 mice. Two groups of mice were fed diets that were identical except that wheat bran composed 40% by wt of one diet. After 1 wk on the diets, all mice were injected sc on their backs with 20 mg/kg DMH. This dose was repeated on a weekly basis for 26 wk or until the mouse died. Histological studies were made of the colons of all animals that died between weeks 15 and 26 and all animals sacrificed at the end of 26 wk. Of the 400 mice in the study, 122 in the experimental group and 141 in the controls died during the first 15 wk, leaving 78 and 59 evaluable mice. The incidence of major pathological changes (atypical adenoma, invasive and in situ carcinoma) was much higher ($p < 0.02$) in the controls than in animals fed fiber bran. Adenomas that were not considered major pathology were much more frequent in control mice than in experimental mice. The investigation supports the claim that addition of bran to the diet has the ability to lower the incidence of carcinoma of the large intestine. (4 refs)

78-3844 **Kinetics of Changes in the Crypts of the Jejunal Mucosa of Dimethylhydrazine-treated Rats.** (Eng) Sunter, J. P. (Dept. Pathology, Royal Victoria Infirmary, Queen Victoria Road, Newcastle upon Tyne NE1 4LP, England); Appleton, D. R.; Wright, N. A.; Watson, A. J. *Br J Cancer* 37(5): 662-672; 1978.

The effects of sc administration 1,2-dimethylhydrazine (DMH: 15 mg/kg/wk) on the crypts of the jejunal mucosa were studied in female Wistar rats. In animals killed after 21-27 wk of DMH treatment, the incidence of colonic and small-intestinal neoplasms was 100% and 37%, respectively. The large-bowel tumors were generally multiple, whereas the small-intestinal tumors were frequently solitary. Most of the small intestinal tumors (33/39) were situated in the most proximal part of the jejunum or in the distal duodenum. A striking elongation of the intestinal crypts was seen after 24 wk or more of DMH treatment. This hyperplasia was generalized, and it occurred in the absence of any features of atypia in both tumor-bearing and non-tumor-bearing animals. In the hyperplastic jejunal crypts, the absolute size of the proliferative compartment was increased, but the growth fraction calculated from labeling studies appeared to fall, probably because of a reduction in the relative size of the proliferating population in this compartment. No convincing alteration in actual cell-cycle time was observed in the abnormal crypts, but there was a 25% increase in the cell production rate. (28 refs.)

78-3845 **A Requirement of Pi for the Transitory Uncoupling of Rat Liver Mitochondria by Hydrazine, When β-Hydroxybutyrate Is the Substrate.** (Eng) Hadler, H. I. (Dept. Chemistry and Biochemistry, Southern Illinois Univ., Carbondale, IL, 62901); Cook, G. L. *J Environ Pathol Toxicol* 1(4): 419-432; 1978.

The mitochondrial target of hydrazine (HZ) was studied using rat liver mitochondria. HZ combined with inorganic phosphate (Pi) had a transitory stimulatory effect on mitochondrial respiration (transitory uncoupling), although neither compound alone had this effect. HZ also prevented the subsequent addition of ADP from establishing coupled phosphorylation. Both effects of HZ were eliminated by the addition of ADP or ATP before HZ or during the induction period associated with HZ. The transitory uncoupling effect was also prevented by pyrophosphate, magnesium ion, antimycin or rutamycin when they were added before HZ. Aurovertin delayed but did not block the uncoupling effect. The transitory uncoupling induced by HZ and Pi appeared to be restricted to conditions in which β-hydroxybutyrate was the substrate. In a mitochondrial system that included ATP, gramicidin, potassium ion, and sulfate, HZ, acting as a large cation but not as a nucleophile, blocked the swelling and the increment in ATPase activity associated with potassium ion. The data suggest that when β-hydroxybutyric acid is the substrate, an electrophilic form of Pi is the mitochondrial target of HZ. The data support an experimental confluence between oxidative phosphorylation and chemical carcinogenesis, and they explain the previously observed toxic effect of HZ on mitochondria. (34 refs)

78-3846 **Correlation Among Mutagenesis, Carcinogenesis and Teratogenesis and Their Response to Phenobarbital or Caffeine Treatment (Meeting Abstract).** (Eng) Nomura, T. (Inst. Cancer Res., Osaka Univ. Medical Sch., Osaka, Japan). *Mutat Res* 53(2): 244; 1978. (no refs)

78-3847 **Cytogenetical Effects of Organophosphates in Mouse (Meeting Abstract).** (Eng) Degraeve, N.

(Genetics Dept., Univ. Liege, B-4000 Liege, Belgium); Moutschen, J.; Houbrechts, N.; Moutschen-Dahmen, M.; Gilot-Delhalle, J.; Colizzi, A. *Mutat Res* 53(2): 175-176; 1978. (no refs)

78-3848 **A Cell-free Assay Measuring Repair DNA Synthesis in Human Fibroblasts.** (Eng) Ciarrocchi, G. (Laboratorio di Genetica Biochimica ed Evoluzionistica, Consiglio Nazionale delle Ricerche, 27100 Pavia, Italy); Linn, S. *Proc Natl Acad Sci USA* 75(4): 1887-1891; 1978.

DNA repair synthesis was measured by a cell-free assay that involves osmotic disruption of confluent cultured human fibroblasts that have been irradiated or exposed to chemical carcinogens. Deoxythymidine triphosphate was used as a precursor. Fibroblasts from various xeroderma pigmentosum (XP) cell lines showed the deficiencies of UV-induced DNA synthesis predicted from in vivo studies, but they gave normal responses to methyl methanesulfonate. Dose responses and levels of repair synthesis (relative to UV stimulation) induced by x-radiation, 4-nitroquinoline 1-oxide, and N-acetoxy-2-acetylaminofluorene were comparable to those seen with intact cells by autoradiography. However, the level of synthesis induced by methyl methanesulfonate was much greater, comparable to that obtained with UV radiation . A pyrimidine dimer-specific enzyme, phage T4 endonuclease V, stimulated the rate of UV-induced repair synthesis with normal and XP cell lines. A number of applications are suggested for this assay system: (1) to identify agents that induce DNA repair and the cells that respond abnormally to such induction; (2) as an in vitro complementation assay using repair-defective cells and proteins obtained from repair-proficient cells; and (3) to measure DNA replication in vitro by using actively growing fibroblasts and thymidine in the system. (33 refs)

78-3849 **DNA Repair Synthesis in Rat Retinal Ganglion Cells Treated with Chemical Carcinogens or Ultraviolet Light In Vitro, with Special Reference to Aging and Repair Level.** (Eng) Ishikawa, T. (Dept. Experimental Pathology, Cancer Inst., 1-37-1, Kamiikebukuro, Toshima-ku, Tokyo 170, Japan); Takayama, S.; Kitagawa, T. *J Natl Cancer Inst* 61(4): 1101-1105; 1978.

A system in which the retinal tissues of noninbred Wistar rats were used in combination with autoradiography was developed for measuring of DNA repair synthesis in ganglion cells of the CNS. Retinal tissues in short-term organ culture were treated with 4-nitroquinoline 1-oxide (0.032-3.2 x 10^{-5} M), ethylnitrosourea (0.032-3.2 x 10^{-3} M), or methylmethanesulfonic acid (0.032-3.2 x 10^{-3} M) plus tritiated thymidine, or they were irradiated with UV light (60, 190, 1,900 or 6,000 ergs/mm^2) and then treated with tritiated thymidine. In carcinogen-treated groups, unscheduled DNA synthesis was clearly demonstrated as silver grains over the nuclei of cells in the outer nuclear layer of the retina. The cells in this layer had uniform numbers of grains. A preliminary study revealed that aging had no effect on the levels of DNA repair in retinal ganglion cells. (27 refs)

78-3850 **Difficulties Arising from the Use of Bone Marrow Cells as Test System for Chromosome Breakage (Meeting Abstract).** (Eng) Moutschen, J. (Genetics Dept., Univ. Liege, B-4000 Liege, Belgium); Moutschen-Dhamen, M.; Degraeve, N.; Houbrechts, N.; Gilot-Delhalle, J.; Colizzi, A. *Mutat Res* 53(2): 234-235; 1978. (no refs)

78-3851 **Dose-Rate Effects of Ethyl Methanesulfonate on Induction of 6-Thioguanine-resistant Mutations in Cultured Chinese Hamster Cells (Meeting Abstract).** (Eng) Kuroda, Y. (Dept. Morphological Genetics, Natl. Inst. Genetics, Misima, Sizuoka, Japan); Sugiura, K. *Mutat Res* 53(2): 215-216; 1978. (no refs)

78-3852 **Linear Relation of Alkylation to Mutation in Drosophila (Meeting Abstract).** (Eng) Lee, W. R. (Dept. Zoology and Physiology, Louisiana State Univ., Baton Rouge, LA, 70803); Aaron, C. S.; Janca, F. C.; Skinner, P. M. *Mutat Res* 53(2): 217; 1978. (no refs)

78-3853 **Evaluation of Ascorbic Acid for Mutagenicity by Dominant Lethal Test in Male Wistar Rats (Meeting Abstract).** (Eng) Chauhan, P. S. (Bio-Medical Group, Bhabha Atomic Res. Centre, Bombay 400085, India); Aravindakshan, M.; Sundaram, K. *Mutat Res* 53(2): 166-167; 1978. (no refs)

78-3854 **Different Pathways for the Excision Repair of DNA in Human Cells (Meeting Abstract).** (Eng) Strauss, B. (Dept. Microbiology, Univ. Chicago, Chicago, IL); Karran, P.; Bose, K.; Higgins, P. *Mutat Res* 53(2): 268-269; 1978. (no refs)

78-3855 **EMS- and MMS-induced Meiotic Non-Disjunction in the Male Mouse (Meeting Abstract).** (Eng) Szemere, G. (Dept. Medical Biology, Szeged Medical Sch., Szeged, Hungary). *Mutat Res* 53(2): 272-273; 1978. (no refs)

78-3856 **Alkylation of Deoxyribonucleic Acid by Carcinogens Dimethyl Sulphate, Ethyl Methanesul-**

phonate, N-Ethyl-N-nitrosourea and N-Methyl-N-nitrosourea. Relative Reactivity of the Phosphodiester Site Thymidylyl(3′-5′)thymidine. (Eng) Swenson, D. H. (Natl. Center Toxicological Res., Jefferson, AR, 72079); Lawley, P. D. *Biochem J* 171(3): 575-587; 1978.

Determination was made of the extent of alkylation of the phosphodiester sites of DNA by four carcinogens: dimethyl sulfate (DS), ethyl methanesulfonate, N-methyl -N-nitrosourea, and N-ethyl -N- nitrosourea (ENU). As a first step, the ethyl phosphotriester of thymidyl(3′-5′)thymidine [dTp(Et)dt] was isolated and characterized as a product of the ethylation of DNA by ENU, by procedures similar to those reported previously for the methyl homolog. Enzymic degradation to yield alkyl phosphotriesters from DNA alkylated by these carcinogens was studied quantitatively, and the relative yields of the triesters dTp(Alk)dT were determined. The relative reactivity of the phosphodiester group dTpdT to each of the carcinogens was obtained and compared with that of overall DNA or with that of the N-7 atom of guanine in DNA. Relative reactivity of the phosphodiester group was lowest toward DS, the least electrophilic of the reagents used, and highest toward ENU the most electrophilic-reagent. The nature of the alkyl group transferred also influenced the reactivity of the phosphodiester site, since this site was relatively more reactive toward ethylation than would be predicted simply from the known Swain-Scott s values of the alkylating agents. It was therefore suggested that the steric accessibility of the weakly nucleophilic phosphodiester group on the outside of the DNA macromolecule favors its reaction with ethylating, as opposed to methylating, reagents. The nucleophilicity of overall DNA was less than that of the N-7 atom of guanine in DNA. The reactivity of overall DNA was markedly high toward the alkylnitrosourea, despite their relatively low s values. This was ascribed to an electrostatic factor that favored reaction of the negatively charged polymer with alkyldiazonium cation intermediates. (26 refs)

78-3857 **Some Interactions Affecting the Mutagenicity Potential of Dipyrone, Hexachlorophene, Thiodan and Malathion (Meeting Abstract).** (Eng) Sylianco, C. Y. (Dept. Chemistry, Coll. Arts and Sciences, Univ. Philippines, Quezon City, Philippines). *Mutat Res* 53(2): 271-272; 1978. (no refs)

78-3858 **Induction of Colon Tumors in 1,2-Dimethylhydrazine-resistant Lobund Wistar Rats by Methylazoxymethanol Acetate.** (Eng) Pollard, M. (Lobund Lab., Univ. Notre Dame, Notre Dame, IN, 46556); Zedeck, M. S. *J Natl Cancer Inst* 61(2): 493-494; 1978.

Differences in the susceptibility of Sprague-Dawley (S-D: high) and Lobund Wistar (LW: low) rats to 1,2-dimethylhydrazine (DMH) were studied by determining tumor incidence in both strains upon treatment with the product of DMH metabolism, methylazoxymethanol (MAM). Rats were inoculated sc with 30 mg/kg/wk MAM acetate for 10 wk. Although 7/7 LW rats and 10/10 S-D rats developed intestinal tract tumors, the tumor incidence was much greater in S-D rats. They developed 20.4 tumors/animal, most of which were adenocarcinomas extending through the muscularis and into the serous membrane covering the intestine. LW rats averaged 3.1 tumors/animal, and they were generally smaller in size and less invasive. The livers of all animals were damaged, with cirrhotic changes being seen in LW rats. Assays of NAD+-dependent dehydrogenase activity in colon tissue indicated that LW rats indicate that the differential susceptibility of these two rat stocks to colon tumor induction is partially related to metabolic activation of DMH and to other, as yet undetermined, endogenous factors. (11 refs)

78-3859 **Effect of Colostomy on Intestinal Carcinogenesis by Methylazoxymethanol Acetate in Rats.** (Eng) Matsubara, N. (Dept. Surgery, Gifu Univ. Sch. Medicine, Gifu City 500, Japan); Mori, H.; Hirono, I. *J Natl Cancer Inst* 61(4): 1161-1164; 1978.

The effect of the fecal stream on the induction of intestinal tumors was studied in three groups of SD rats. Rats in Group 1 were subjected to single-barreled colostomies for the complete exclusion of the fecal stream at the proximal one-third level of the colon and were given consecutive iv injections of methylazoxymethanol acetate (MAMA). Rats in Group 2 were given MAMA alone. Rats in Group 3 were not treated and served as controls. Tumors were noted in the small and large intestines of almost all Group 1 and 2 rats. Only the multiplicity of the lesions in the entire intestinal tract and tumor incidence in the oral one-third of the colon were significantly higher in Group 2 (9.3, 72.2%) than in Group 1 (5.4, 28%). In spite of the single-barreled colostomies, 80% of the Group 1 animals developed tumors in the colon distal to the colostomy, where the mucosa did not have contact with the fecal stream. These results indicate that carcinogens can probably reach the intestinal mucosa via the vascular system as well as by biliary transport. (14 refs)

78-3860 **Experience with Mutagenic Tests as Indicators of Carcinogenic Activity (Meeting Abstract).** (Eng) Cinkotai, K. I. (Pharmaceuticals Div., Imperial Chemical Industries, Macclesfield, Cheshire, England); Jones, C. C.; Topham, J. C.; Watkins, P. A. *Mutat Res* 53(2): 167-168; 1978. (no refs)

78-3861 **Selection of Species for Cancer Bioassay for Naphthylamine-containing Food Colours on the Basis of Tissue-mediated Mutagenicity (Meeting Abstract).** (Eng) Stoltz, D. R. (Toxicology Res. Div., Health Protection

Branch, Tunney's Pasture, Ottawa K1A OL2, Canada); Bendall, R.; Stavric, B.; Munro, I. C. *Mutat Res* 53(2): 267-268; 1978. (no refs)

78-3862 **A Method to Test Plant Metabolism on the Mutagenicity of Pesticides (Meeting Abstract).** (Eng) Morpurgo, G. (Istituto Superiore di Sanita, Rome, Italy); Iachetta, R.; Camoni, I.; Ortali, V. A.; Benigni, R.; Carere, A.; Gualandi, G. *Mutat Res* 53(2): 232; 1978. (3 refs)

78-3863 **Selection of an In Vitro Carcinogenicity Test for Use with Derivatives of the Carcinogen Hexamethylphosphoramide (HMPA) (Meeting Abstract).** (Eng) Ashby, J. (Central Toxicology Lab., Imperial Chemical Industries Ltd., Alderley Park, Macclesfield, Cheshire SK10 4TJ, England); Styles, J. A.; Anderson, D. *Mutat Res* 53(2): 149; 1978. (no refs)

78-3864 **Evaluation of the Rat-Liver DNA Damage by Parathion in Relation with its Non-Mutagenicity (Meeting Abstract).** (Eng) Decloitre, F. (Institut de Recherches Scientifiques sur le Cancer, Villejuif, France). *Mutat Res* 53(2): 175; 1978. (no refs)

78-3865 **Blastocyte Recovery in the Evaluation of Mutagenic Hazard (Meeting Abstract).** (Eng) Tesh, J. M. (Life Science Res., Stock, Essex, England); Ross, F. W. *Mutat Res* 53(2): 275-276; 1978. (no refs)

78-3866 **Further Studies on Cytogenetic Effects of Folpet (Meeting Abstract).** (Eng) Sirianni, S. R. (Dept. Experimental Biology, Roswell Park Memorial Inst., Buffalo, NY, 14263). *Mutat Res* 53(2): 264-265; 1978. (no refs)

78-3867 **Metabolic Fate of the Precursors of N-Nitroso Compounds (III). Urinary Excretion of Nitrate in Man.** (Eng) Ishiwata, H. (Natl. Inst. Hygienic Sciences, 18-1 Kamiyoga 1-chome, Setagaya-ku, Tokyo, Japan); Mizushiro, H.; Tanimura, A.; Murata, T. *J Food Hyg Soc Jpn* 19(3): 318-322; 1978.

The amount of nitrate ingested in foods and beverages and the amount excreted in urine were determined in normal male and female volunteers. Urine specimens were collected three times from 11 random subjects on different days, and the mean nitrate concentration was 74.4 ppm. The amount of nitrate excreted in 24-hr urine samples from nine subjects who ingested a conventional diet containing 44.1-864.2 mg nitrate was 45.6-236.9 mg, which indicates that the urinary excretion of nitrate varies widely among individuals (27.4%-133.8%). No nitrate was detected in any urine specimens. Three subjects were fed a low-nitrate diet from day -1 to day 4. On days 0 and 2, subject A ingested 200 mg sodium nitrate and on day 0 subjects B and C ingested salted Chinese cabbage containing 75.1 mg nitrate at breakfast. In subject A, the urinary excretion of nitrate was not increased on the control days, but it was increased 78.3% and 86.2% on days 0 and 2. Almost the same excretion pattern was observed in subjects B and C. The amount of nitrate excreted increased by >25 mg every 2 hr on the experimental days in all three subjects. No nitrite was detected in any urine specimens, and neither nitrate nor nitrite was detected in any feces. (20 refs)

78-3868 **Alpha-Tocopherol: Uses in Preventing Nitrosamine Formation.** (Eng) Mergens, W. J. (Hoffmann-La Roche Inc., Nutley, NJ); Kann, J. J.; Newmark, H. L.; Fiddler, W.; Pensabene, J. *IARC Sci Publ* (19): 199-212; 1978.

The ability of the fat-soluble reducing agent α-tocopherol (TP, vitamin E) to destroy nitrite ion, thereby preventing nitrosamine formation, was evaluated in vitro, in vivo, in cigarettes, and in bacon. TP was emulsified in water and spray-dried to yield a free-flowing solid containing 33% by wt of TP. TP reacted with nitrosating agents in lipophilic and aqueous environments, and it destroyed nitrite in simulated gastric fluid. In rats, the elevation of serum glutamic pyruvic transaminase normally seen in animals treated with sodium nitrite [0.43 millimole (mmol)], and aminopyrene (0.15 mmol) was inhibited completely when TP (0.51 mmol) was included in the treatment. N-Nitrosodimethylamine (NDMA) yields were reduced by an av of 50% in cigarettes treated with 30 mg TP. Addition (via the cure soln) of TP to bacon inhibited N-nitrosopyrrolidine formation significantly after the bacon was fried and it also reduced the formation of NDMA, but to a lesser extent. Inhibition was greater with TP + sodium ascorbate than with the latter alone. The mechanism whereby TP reacts with a nitrosating intermediate is not fully understood. The data obtained in this study indicate that the reaction could take place completely within a lipid micelle. (24 refs)

78-3869 **Transnitrosation: An Important Aspect of the Chemistry of Aliphatic Nitrosamines.** (Eng) Singer, S. S. (NCI-Frederick Cancer Res. Center, Frederick, MD); Lijinski, W.; Singer, G. M. *IARC Sci Publ* (19): 175-181; 1978.

Investiations into the kinetics and mechanism of transnitrosation by aliphatic nitrosamines imply that a reactive intermediate (ie, thiocyanate ion), an Sn2-type displacement of the nitroso group, and a suitable receptor (urea, ammonia, or free amino acids) are required for the reaction. At pH 1.5-2, transnitrosation is rapid in the presence of thiocyanate ion, which is present in human saliva. The reaction could occur in the human stomach, where transnitrosating agents could form new carcinogenic nitrosamines by transfers to appropriate amines. (13 refs)

78-3870 **Problem of Artifacts in the Analysis of N-Nitroso Compounds.** (Eng) Krull, I. S. (Cancer Res. Div., Thermo Electron Corporation, 45 First Ave., Waltham, MA, 02154); Fan, T. Y.; Fine, D. H. *Anal Chem* 50(6): 698-701; 1978.

Various procedures that ensure the accuracy and precision of results obtained in the analysis of N-nitroso compounds are outlined. These techniques are designed to prevent both false-positive and false-negative findings during an analysis and to improve the reliability of N-nitroso analyses. Specific examples of the various types of causes of artifact formation are presented. (34 refs)

78-3871 **Salted Fish and Nasopharyngeal Carcinoma in Southern Chinese (Letter to Editor).** (Eng) Ho, J. H. (Medical and Health Dept., Inst. Radiology and Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong); Huang, D. P.; Fong, Y. Y. *Lancet* 2(8090): 626; 1978.

Extracts of salted fish and the urine of four pairs of albino rats fed salted fish, a staple food of the southern Chinese, produced dose-dependent mutagenicity in *Salmonella typhimurium* strains TA100 and TA98. Some of the mutagens in the fish may be N-nitroso compounds. These findings may explain the previously noted induction of carcinoma in rats fed salted fish and the high incidence of nasopharyngeal carcinoma in the southern Chinese. (14 refs)

78-3872 **Induction of Gastric Tumors in BD-VI Rats by Single Application of N-Methyl-N′-nitro-N-nitrosoguanidine in Dimethylsulfoxide (DMSO).** (Eng) Habs, M. (Inst. Toxicology and Chemotherapy, German Cancer Res. Center, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany); Deutsch-Wenzel, R.; Preussmann, R.; Schmahl, D. *Z Krebsforsch* 91(2): 183-188; 1978.

In an acute toxicity test, the LD_{50} of N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) administered po to BD-VI rats in a 10% aqueous soln of dimethyl sulfoxide (DMSO) was 90 mg/kg. After a single po administration of 50 mg/kg (Group I), 75 mg/kg (Group II), or 100 mg/kg (Group III) of MNNG in a 10% aqueous soln of DMSO, generalized forestomach papillomatoses were found in all BD-VI rats after 500 days. Squamous cell carcinomas were detected in 7/24 rats in Group I, 12/26 in Group II, and 7/23 in Group III. Benign adenomatous dysplasia of the glandular stomach was diagnosed in 2 Group I rats, 1 Group II rats, and 12 Group III rats. Adenocarcinomas of the glandular stomach were induced in one animal of Groups I and II and in eight rats of Group III. From 20% to 30% of the MNNG-treated animals also showed degenerative cystic alterations in the liver with bile-duct proliferation. (25 refs)

78-3873 **The Influence of Ischemia and N-Methyl-N′-nitro-N-nitrosoguanidine on Rat Gastric Mucosa.** (Eng) Duris, I. (Dept. Internal Medicine, Medical Sch., Comenius Univ., Bratislava, Czechoslovakia); Popperova, E.; Zaviacic, M. *Tumor Res (Sapporo)* 12: 52-57; 1977.

The effect of ischemia on N-methyl-N′-nitro-N-nitrosoguanidine (MNNG)-induced carcinogenesis in the gastric mucosa of Dobra Voda (Czechoslovakia) strain male Wistar rats was determined. The rats were divided into five experimental groups: Group A underwent ligation of the gastric artery and, 8 wk later, received drinking water containing 83 mg MNNG for 8 wk; Group B received MNNG for 8 wk immediately after ligation of the gast artery; Group C received MNNG for 8 wk only; Group D underwent ligation of the gastric artery only; and Group E received no treatment. All animals were sarcificed 16 wk after termination of MNNG administration. Gastric mucosal lesions were noted in 11/16 Group A animals, 14/18 Group B animals, and 4/13 Group C animals; no tumors were noted in Groups D and E. Only two adenocarcinomas were noted: one in a Group A animal at sacrifice and one in a Group C animal that died 4 wk after termination of MNNG treatment. These findings indicate that ligation of the gastric artery potentiated the influence of MNNG. (17 refs)

78-3874 **Nitrosoguanidine Mutagenesis During the Yeast Cell Cycle.** (Eng) Carter, B. L. (Dept. Genetics, Univ. Dublin, Trinity Coll., Dublin 2, Ireland); Dawes, I. W. *Mutat Res* 51(2): 289-292; 1978.

Exponential cultures of yeast cells (*Saccharomyces cerevisiae*) were harvested by centrifugation and resuspended in 0.2 M potassium acetate buffer (pH 5.4) containing 500 μg/ml N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) for 15 min at 30 C. Since all stages of the cycle are represented in an exponential culture, treating such a culture with MNNG for a fraction of the cycle is equivalent to treating cells at different stages of the cycle for a fraction of the cycle. The culture was then centrifuged, washed, separated according to size and stage of the cycle by zonal centrifugation, and plated for resistance to 0.4 mg/ml erythromycin. Mutation to nuclear erythromycin resistance was enhanced during nuclear DNA replication, and this enhancement depended on DNA replication taking place at the time of mutagenesis. If replication was inhibited 15 min prior to the application of mutagen by hydroxyurea, no peak of mutation was observed in that region of the cell cycle where nuclear DNA replication ordinarily occurs. In addition, in untreated cells actively replicating their DNA, there was no detectable enhancement of spontaneous mutation during the S phase. (15 refs)

78-3875 **Neoplastic Transformation of Canine Embryo Cells in Vitro by N-Methyl-N′-nitro-N-nitrosoguanidine.** (Eng) Rhim, J. S. (Microbiological Associates, Inc., Bethesda, MD, 20016); Park, D. K.; Arnstein, P.; Nelson-Rees, W. A. *Int J Cancer* 22(4): 441-446; 1978.

A cell line derived from a normal beagle embryo was treated in vitro with 0.01 or 0.1 μg/ml N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) or dimethyl sulfoxide (DMSO, control). Cultures were treated for 7 days, washed, refed with carcinogen-free growth medium, and passaged every 7 days. Morphologic alteration and an abnormal growth pattern were observed in MNNG-treated cells in the seventh subculture (75-79 days after treatment); no such changes were observed in control cells. Saturation densities of cells transformed by 0.01 and 0.1 μg/ml MNNG were 3.9 x 10^5 and 2.1 x 10^5 cells/cm^2, respectively, two to four times higher than that of control cells. Transformed cells formed larger aggregates than DMSO-treated cells, and they grew in this aggregate form when suspended in liquid growth medium above an agar base. All nude mice inoculated sc with 5 x 10^6 cells treated with 0.01 μg/ml MNNG developed transplantable fibrosarcomas within 2 wk at the injection site. No tumors developed in mice inoculated with DMSO-treated cells or cells treated with 0.1 μg/ml MNNG. (21 refs)

78-3876 **Induction of Intestinal Metaplasia in the Stomachs of Rats by N-Methyl-N′-nitro-N-nitrosoguanidine.** (Eng) Matsukura, N. (Natl. Cancer Center Res. Inst., Tsukiji, Chuo-ku, Tokyo 104, Japan); Kawachi, T.; Sasajima, K.; Sano, T.; Sugimura, T.; Hirota, T. *J Natl Cancer Inst* 61(1): 141-144; 1978.

The induction of intestinal metaplasia was studied in the stomachs of male Wistar rats given N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) (83 μg/ml in the drinking water) for 2, 4, 5, or 7 mo. MNNG intakes were estimated at 150, 300, 375, and 525 mg, respectively. Most of the rats were killed at 15 mo. Intestinal metaplasia was found in the stomachs of 80%-100% of the rats treated for ≥ 4 mo, of 37.5% treated for 2 mo, and of 10% of the controls. Metaplastic glands, composed of goblet cells and columnar cells with striated borders, were found in the pyloric region. Paneth's cells were found at the bottom of the metaplastic glands in a rat treated for 4 mo. The incidence of well-differentiated adenocarcinomas of the stomach was 63%-90% in rats treated with MNNG for ≥ 4 mo and 25% in those treated for 2 mo. This indicates that with po administration of MNNG, almost the same length of time is required to induce intestinal metaplasia and well-differentiated adenocarcinomas. (12 refs)

78-3877 **Croton Oil Enhancement of Skin Tumor Initiation by N-Methyl-N′-nitro-N-nitrosoguanidine: Possible Role of DNA Replication.** (Eng) Hennings, H. (In Vitro Pathogenesis Section, Experimental Pathology Branch, NCI, NIH, Bethesda, MD, 20014); Michael, D.; Patterson, E. *Proc Soc Exp Biol Med* 158(1): 1-4; 1978.

The effect of a single croton oil application between 6 hr before and 20 hr after skin tumor initiation by N-methyl -N′- nitro -N- nitrosoguanidine (MNNG) was studied to determine the relationship of DNA replication to the croton oil-induced enhancement of initiation. Female CD-1 mice received a single application of 0.5% croton oil 6 hr before or 1, 4, 8, or 20 hr after application of 4-micromoles of MNNG. There was a threefold increase in papilloma yield and a substantial decrease in the latent period (from 30 to 17-24 wk) when croton oil was applied at any of the times tested. Since DNA replication was not stimulated by croton oil until 2-4 days after application of MNNG, this enhancement cannot be explained by an increased uptake of the MNNG by DNA-synthesizing cells or by an increased susceptibility of these cells. If DNA replication is involved in initiation, these results suggest that the critical DNA replication occurs after the interaction of MNNG with DNA but before the initial damage can be repaired, perhaps resulting in a nonrepairable mutation. (16 refs)

78-3878 **Kinetic Studies on the Hepatic Microsomal Metabolism of Dimethylnitrosamine Diethylnitrosamine, and Methylethylnitrosamine in the Rat.** (Eng) Chau, I. Y. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA, 02139); Dagani, D.; Archer, M. C. *J Natl Cancer Inst* 61(2): 517-521; 1978.

The kinetics of the oxidative dealkylation of dimethylnitrosamine (DMN), diethylnitrosamine (DEN), and methylethylnitrosamine (MEN) by male Sprague-Dawley rat liver microsomes and the mutual interactions of these compounds were studied by a colorimetric assay that allows simultaneous measurement of formaldehyde and acetaldehyde formation. The rats were treated with 0.1% phenobarbital for 7 days before sacrifice. The substrate concentration ranges used, 38-193 mM DMN, 4-44 mM DEN, and 10-104 mM MEN, were determined primarily by the sensitivity of the aldehyde assays. For each substrate, liberation of aldehyde followed simple Michaelis-Menten kinetics. DEN had an apparent Michaelis constant (Km) value that was more than an order of magnitude smaller than that for DMN. The Km values for the demethylation and deethylation of MEN were intermediate between those for DMN and DEN. Thus, MEN was dealkylated efficiently by liver enzyme; this is consistent with the observation that, under similar conditions, DMN, DEN, and MEN are all liver carcinogens in the rat. DMN demethylase was inhibited by DEN and DEN deethylase was inhibited by DMN; in both cases, the inhibition was of a mixed type. (28 refs)

78-3879 **Selective Sex-related Modification of Diethylnitrosamine-induced Carcinogenesis in BALB/c Mice by Concomitant Administration of Butylated Hydroxytoluene.** (Eng) Clapp, N. K. (Biology Div., Oak Ridge Natl. Lab., P.O. Box Y, Oak Ridge, TN, 37830); Tyndall, R. L.; Satterfield, L. C.; Klima, W. C.; Bowles, N. D. *J Natl Cancer Inst* 61(1): 177-182; 1978.

Tumor induction by diethylnitrosamine (DENA) and its modification by butylated hydroxytoluene (BHT) were studied in BALB/c mice. BHT (0.75%) was added to the regular chow of one group of 8-wk-old mice and administered for life. A second group was given DENA in the drinking water (4 mg/100 ml) beginning at 11 wk of age for 7 wk (mean dose consumed, 330 mg/kg). Another group received BHT + DENA, and a fourth group was given untreated food and water. Female mice killed at 13.5 mo after DENA exposure had higher incidences of forestomach and lung tumors than did males, and they also had higher numbers of lung tumors per tumor-bearing mouse. Neither sex developed stomach tumors spontaneously, but males had higher incidences of spontaneous lung tumors than females (45% vs 26%). Females receiving BHT + DENA had an increased survival (68% vs 43%) and a reduced incidence of forestomach squamous cell carcinomas (77% vs 100%) compared with females There was no such protective effect on survival or on the occurrence of stomach tumors in males given BHT + DENA, and there was no such effect on lung tumorigenesis in either sex. In males, but not in females, BHT given alone increased survival time of treated mice over untreated control. The results suggest an hormonal influence and/or dependence on the protective action of BHT. (18 refs)

78-3880 **Preliminary Observations on Amines and Nitrosamines in Non-normal Human Gastric Contents.** (Eng) Lakritz, L. (U.S. Dept. Agriculture, Eastern Regional Res. Center, Philadelphia, PA); Wasserman, A. E.; Gates, R.; Spinelli, A. M. *IARC Sci Publ* (19): 425-431; 1978.

Gastric contents from 35 patients undergoing routine clinical gastric examination were assayed for amines and nitrosamines (NA's). A number of samples were tested for NA's before and/or after stimulation with histamine or pentagastrin, making a total of 57 samples analyzed. N-Nitrosodiethylamine (5-30 μg/kg) was present in four samples, N-nitrosodimethylamine (2 μg/kg) in two, and N-nitrosopyrrolidine (6 μg/kg) in one. Twenty-two of the gastric samples in which NA's were absent averaged 1.1 mg/kg nitrite and 23.7 mg/kg nitrate. Three samples in which NA's were detected were also analyzed for nitrite and nitrate, and 2/3 (from the same patient) contained about 70 mg/kg nitrite. There did not appear to be a relationship between the NA content and the pH of the gastric samples. The volatile amines found included dimethylamine, trimethylamine, and histamine. The nonvolatile amines detected were cadaverine, putrescine, ethanolamine, and tryptamine. A partial breakdown of the diagnoses for these patients includes: duodenal ulcer, 8; gastric ulcer, 1; peptic ulcer, 2; esophagitis, duodenitis, and gastritis, 4; and diabetes, cirrhosis, and anemia (numbers not given). There was no correlation between medical diagnosis and the presence of NA's in the gastric contents. (11 refs)

78-3881 **Tumor Induction by N-Nitroso Compounds in Bivalve Mollusks *Unio pictorum*.** (Eng) Khudoley, V. V. (N. N. Petrov Res. Inst. Oncology, Leningrad 188646, USSR); Syrenko, O. A. *Cancer Lett* 4(6): 349-354; 1978.

Exposure of bivalve mollusks (*Unio pictorum*) to diethylnitrosamine (DENA) and dimethylnitrosamine (DMNA) dissolved in the tank water resulted in tumor formation. DENA induced tumors in 18% of the animals exposed to 200 ppm for 64 days and 68% of those exposed to 400 ppm for 62 days; the av latent periods were 71 and 61 days, respectively. DMNA induced tumors in 8% of the animals exposed to 200 ppm for 51 days and 27% of those exposed for 152 days, and av latent periods were 85 and 81 days, respectively. Neoplasms were observed in the digestive gland (basophilic cell tumors; 38 cases), the hemopoietic system (hemocytoblastoma; 12 cases), and the kidney (poorly differentiated carcinoma; 1 case). Tumors arose from the digestive glands on a background of diffuse and focalproliferation of basophilic cells. They were invasive and characterized by extraordinary pleomorphisms and atypia. Tumor incidence was dose-dependent.It is suggested that mollusks can be used to determine the carcinogenicity of N-nitroso compounds and to indicate possible water pollutants. (9 refs)

78-3882 **Effect of Dimethylnitrosamine Concentration on Its Demethylation by Liver Microsomes from Control and 3-Methylcholanthrene Pretreated Rats, Hamsters and Guinea Pigs.** (Eng) Lotlikar, P. D. (Fels Res. Inst., Temple Univ. Medical Sch., Philadelphia, PA, 19140); Hong, Y. S.; Baldy, W. J. *Cancer Lett* 4(6): 355-361; 1978.

The effect of dimethylnitrosamine concentration on its demethylation by liver microsomes from control and 3-methylcholanthrene pretreated (100 mg/kg ip 24 hr before sacrifice) Sprague-Dawley rats, Syrian hamsters and albino guinea pigs was investigated. At a low substrate concentration (2 mM), liver microsomes from pretreated rats and hamsters showed 30%-50% lower demethylation activity than their respective controls. No such difference was found in the guinea pig. At a high substrate concentration (100 mM), all three pretreated species showed 50%-100% higher enzyme activity than their respective controls. Enzyme activities among the three species showed the following order of activity: hamster > guinea pig > rat. The DMN concentration-dependent differential DMN demethylating activities found may be due to the presence of enzymes having different affinities for DMN. (22 refs)

78-3883 **Differential Effects of β-Naphthoflavone and Pregnenolone-16α-carbonitrile on Dimethylnitrosamine-induced Hepatocarcinogenesis.** (Eng) Argus, M. F. (Seamen's Memorial Res. Lab., Biochemistry Section, U.S. Public Health Service Hosp., 210 State St., New Orleans, LA, 70118); Hoch-Ligeti, C.; Arcos, J. C.; Conney, A. H. *J Natl Cancer Inst* 61(2): 441-449; 1978.

The effect of simultaneous administration of β-naphthoflavone (NF) or pregnenolone-16α-carbonitrile (PCN) on the hepatocarcinogenicity of dimethylnitrosamine (DMN) was investigated in male SD rats. Both NF and PCN are potent inhibitors of DMN demethylase I, a low-Michaelis-constant, mixed-function oxidase believed relevant to the in vivo metabolic activation of DMN. DMN in tap water was administered via stomach tube 5x/wk for 28 wk (400 μg/rat/administration). NF or PCN (25 mg/kg) was administered in laboratory chow. Three groups of rats were treated with DMN, NF, or PCN alone. DMN-induced hepatocarcinogenesis was inhibited by PCN and enhanced by NF. Liver tumors developed in 14/43 DMN rats, 32/43 DMN + NF rats, and 7/45 DMN + PCN rats. Neither untreated controls, 32 rats receiving NF, nor 28 rats receiving PCN developed liver tumors. Although DMN + NF increased the number, invasiveness, and dissemination of tumors compared with DMN alone, the latent period was not affected. PCN decreased the absolute number of liver tumors and increased the latent period, but tumor invasiveness and dissemination were increased. Of the total 53 tumors, 28 were vascular in origin, a change in the usual morphology of DMN-induced liver tumors. Four possible explanations for the unexpected enhancement of DMN-induced hepatocarcinogenesis by NF are given. The differential effects of NF and PCN prompt a reexamination of the unique role attributed to DMN demethylase in the metabolic activation of DMN. (58 refs)

78-3884 **The Influence of DDT on Metabolism of Dimethylnitrosamine in Rats.** (Eng) Syrowatka, T. (State Inst. Hygiene, 00-791 Warsaw, Poland); Wiadrowska, B. *Toxicol Appl Pharmacol* 44(2): 219-224; 1978.

The in vitro metabolism of dimethylnitrosamine (DMNA) by liver cell preparations from untreated rats and rats given two to eight ip injections of DDT (1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane: 50 mg/kg, every second day) or a single ip injection of 3-methylcholanthrene (MC: 100 mg/kg) was studied. The activity of O-demethylase in the liver postmitochondrial supernatant was measured as an overall indicator of microsomal enzyme induction. Compared with controls, only the preparations from rats given eight injections of DDT showed significant differences in the rate of DMNA metabolism. O-Demethylase activity was significantly different among preparations from all treated groups, and the activity in these preparations differed significantly from that of controls. The greatest increases in activity were in the preparations from rats treated with MC or two doses of DDT. However, there was no correlation between the rate of DMNA metabolism and O-demethylase activity with the preparations. When DMNA (40 mg/kg) was administered ip to the DDD-treated rats 24 hr after the last injection, no DMNA was detected in the blood 8 hr later. The data suggest that the induction of liver microsomal enzymes by DDT exerts only a slight effect on the rate of DMNA metabolism in vitro and in vivo. (13 refs)

78-3885 **A Sensitive Method for the Colorimetric Determination of Volatile Nitrosamines in Food Products and Air.** (Eng) Ceh, L. (Dept. Biochemistry, Veterinary Coll. Norway, P.O. Box 8146, Dep. Oslo 1, Norway); Ender, F. *Food Cosmet Toxicol* 16(2): 117-121; 1978.

Volatile nitrosodialkylamines (NDA's) were extracted from foods containing at least 0.1 μg NDA (preferably 1-10 μg) with freshly distilled water and purified and concentrated by distillations from alkaline and acid soln. The nitrosamines were then reduced with granulated Zn and HCl and oxidized to monoalkylhydrazines by heating and evaporating the soln to a volume of 3-5 ml. Following this, the oxidized compounds were coupled with p-dimethylaminobenzaldehyde. The absorbance of the resulting alkylaldazine complex was measured at 458 nanometers (nm). A calibration curve was obtained by carrying out this procedure with N-nitrosodimethylamine (NDMA), the most common NDA found in food and feed materials. Photometry at 458 nm allowed the determination of as little as 0.1 μg NDMA. Higher nitrosoalkylamines were as efficiently reduced by Zn and HCl as was NDMA. The method was equally sensitive when it was used to determine nitrosamines in laboratory and storeroom air. (16 refs)

78-3886 **Dimethylnitrosamine Inhibits Enzymatic Removal of O^6-Methylguanine from DNA.** (Eng) Pegg, A. E. (Dept. Physiology, Milton S. Hershey Medical Center, Pennsylvania State Univ., 500 University Drive, Hershey, PA, 17033). *Nature* 274(5667): 183-184; 1978.

Liver and kidney extracts from control rats and rats treated with dimethylnitrosamine (DMN: 20 mg/kg) were tested for their ability to catalyze the loss of 0^6-methylguanine (O-MG) from acid-precipitable DNA in vitro. The extracts were incubated with calf thymus DNA that had been alkylated by reaction with ^{3}H-N-methyl-N-nitrosourea or with rat liver

DNA that had been alkylated in vivo by an injection of H-DMN (50 μg/g) shortly before death. None of the exracts resulted in the loss of 7-methylguanine or guanine from he acid-precipitable DNA. Liver extracts were more active han kidney extracts in catalyzing the removal of O-MG, but with both tissues the activity was lost if the rats had been pretreated with 20 mg/kg DMN. Extracts from the nitrosamine-treated rats were still inactive after a purification step nvolving chromatography on 2-(diethylamino)ethanol celulose. The findings suggest that high doses of alkylating carcinogens inhibit the hepatic and renal enzyme systems that are involved in the removal of O-MG from DNA. This inhibitory action may be important in the induction of tumors by nitroso compounds. (13 refs)

78-3887 **Formation of Nitrosodimethylamine by Microorganisms Used in the Baking Industry or Isolated from the Raw Materials of Bakery Products.** (Eng) Uibu, J. (Inst. Experimental and Clinical Medicine, Tallinn, Estonia); Bogovski, P.; Tauts, O. *IARC Sci Publ* (19): 247-256; 1978.

The production of N-nitrosodimethylamine (NDMA) by microorganisms used in the baking industry and the presence of NDMA in the raw materials of bakery products were investigated. Among the 22 strains of bacteria and fungi studied, 7 produced NDMA: 2 of *Saccharomyces cerevisiae* used in the baking industry; 2 of *Pseudomonas*, one from rye flour and one from compressed baker's yeast; and a strain of *Bacillus subtilis* isolated from leaven. NDMA production was not a property of a particular species but rather of various biotypes. All bacterial NDMA producers showed nitrate reductase activity, but not all nitrate-reducing microorganisms produced NDMA. Two strains of *S. cerevisiae* produced NDMA without any of this enzyme activity. Prolonged incubation time was desirable for detecting microbial producers of nitrosamines. The yield of NDMA produced by a *Pseudomonas* strain was reduced in the presence of 0.1% and 1% flour; however, at higher percentages, the yield increased and remained constant to 10%. In contrast, NDMA production by *P. herbicola* decreased as the amount of crushed wheat was increased, a result that may be related to the tendency of the pH to increase when more wheat was added. Although the cultivation conditions used were far from those occurring in a bakery plant, the data strongly indicate the need for more research concerning nitrosamine formation in bakery products. (17 refs)

78-3888 **Interactions of 4-Nitroquinoline 1-Oxide with Four Deoxyribonucleotides.** (Eng) Winkle, S. A. (Dept. Chemistry, Univ. California, Berkeley, CA, 94720); Tinoco, I. *Biochemistry (Washington)* 17(7): 1352-1356; 1978.

The interactions of 4-nitroquinoline l-oxide (NQO) with the four 5′-deoxyribonucleotides were probed using absorption spectra of the charge transfer bands and ^{1}-H and ^{13}C nuclear magnetic resonance spectra of nucleotide-NQO mixtures. Using Benesi-Hildebrand-type plots, the equilibrium constants for the formation of 1:1 complexes of deoxyguanosine 5′-monophosphate, deoxyAMP, deoxythymidine ′-monophosphate, and deoxycytidine 5′-monophosphate and NQO were obtained. These equilibrium constants, which are similar in value to those obtained for the deoxynucleosides, indicate the preference of NQO for the guanine residues of DNA. NQO, rather than a possible metabolite such as 4-hydroxyaminoquinoline 1-oxide, could possibly be the species that interacts with the bases of DNA. A previous electron spin resonance study on NQO free radicals showed that DNA enhanced the formation of NQO free radicals, which might suggest that DNA helps metabolize the NQO to an adduct-forming species. These studies indicate the feasibility of using NMR and optical methods for studying complexing of NQO with dinucleotides. (17 refs)

78-3889 **Lethal and Mutagenic Action of a Carcinogen 4-Nitroquinoline 1-Oxide on *Chaetomium aureum* Chivers.** (Eng) Ghora, B. K. (Washington Univ., Sch. Medicine, Dept. Microbiology, Box 8093, St. Louis, MO, 63110); Chaudhuri, K. L. *Science Culture* 43(8): 357-359; 1977.

The effects of 4-nitroquinoline 1-oxide (4NQO) on *Chaetomium aureum* fungi were investigated. Doses of 16.0 x 10^{-6} M 4NQO drastically reduced the survival rate of *C. aureum*, but a much smaller reduction was noted with 16.0 x 10^{-7} M. The mutagenic action of the compound also increased with increasing dose; max mutagenicity was noted at 16.0 x 10^{-6} M. All mutants lost their cellulase producing ability. (12 refs)

78-3890 **N-Nitroso-3-hydroxypyrrolidine in Fried Bacon and Fried Out Fat.** (Eng) Lee, J. S. (Dept. Food Science and Technology, Oregon State Univ., Corvallis, OR); Libbey, L. M.; Scanlan, R. A.; Barbour, J. *IARC Sci Publ* (19): 325-332; 1978.

A new method for the trace analysis of N-nitroso-3-hydroxypyrrolidine (NHPYR) in fried bacon and fried-out fat is described. For the identification study, five commercial brands of raw bacon were obtained. For recovery studies, six replicate analyses were performed on fried nitrite-free bacon and fried-out fat that had been spiked with 10 ppb NHPYR. NHPYR was extracted from the sample with water:methanol (3:2). After elimination of residual nitrite with ammonium sulfamate, NHPYR was removed from the water:methanol by continuous extraction with dichloromethane. Identification was accomplished by conversion of NHPYR to the trimethylsilyl derivative, followed by separation on a capillary column and characterization by gas chromatography-mass spectrometry. Concentration and recovery studies were conducted without derivatization. Concentrated dichloromethane extracts were analyzed by a gas chromatograph-thermal energy analyzer system. Recovery of NHPYR from fried bacon and fried-out fat was 62% and 63%, respectively. NHPYR levels in five samples of commercial bacon were 0.4-3.0 ppb in fried bacon and 0.3-2.3 ppb in fried-out fat. Although N-nitrosopyrrolidine is a well-known carcinogen, it is not known whether NHPYR is also carcinogenic. (12 refs)

78-3891 **Trends in Levels of N-Nitrosopyrrolidine in Fried Bacon.** (Eng) Havery, D. C. (Dept. Health, Education and Welfare, Washington, DC); Fazio, T.; Howard, J. W. *IARC Sci Publ* (19): 305-310; 1978.

Nine popular brands of bacon on the Washington, DC, retail marked were analyzed for N-nitrosopyrrolidine (NNP) content from 1971 to 1977. The results show that there was a significant decline in NNP content in six brands during 1971-1974. Since 1974, there has been little change in NNP levels. Additional progress in eliminating NNP may be difficult if botulinum control is to be maintained. Substitutions for nitrite are currently under investigation. (5 refs)

78-3892 **Comparative Mutagenesis of Nitrosopiperidines (Meeting Abstract).** (Eng) Nix, C. E. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37820); Rao, T. K.; Larimer, F. W.; Epler, J. L. *Mutat Res* 53(2): 243; 1978. (no refs)

78-3893 **Determination of N-Nitrosoproline in Meat Samples.** (Eng) Baker, J. K. (Dept. Medicinal Chemistry, Sch. Pharmacy, Univ. Mississippi, University, MS, 38677); Ma, C. Y. *J Agric Food Chem* 26(5): 1253-1255; 1978.

A combination of high-pressure liquid chromatography and thermal energy analysis for the determination of N-nitrosoproline (NPRO) in meat was shown to have sufficient accuracy and moderate precision in spiked samples containing 100 ppb NPRO. Application of the method to commercial meat samples revealed that some contained very low levels of NPRO. (12 refs)

78-3894 **The Results of Pancreatography During Pancreatic Carcinogenesis.** (Eng) Takahashi, M. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB, 68105); Pour, P. *Am J Pathol* 91(1): 57-70; 1978.

Systematic histologic-pancreatographic examinations were performed during induced pancreatic carcinogenesis in Syrian golden hamsters to define relevant criteria for tumor detection at early stages. The hamsters were given weekly s injections of N-nitrosobis(2-oxopropyl)amine (10 mg/kg) fo up to 9 wk. Animals were killed beginning 2 wk after the fift injection and at 2-wk intervals thereafter. Immediately afte death, India ink was injected into the pancreatic duct, an the specimen was processed for photography and histologi evaluation. Ductal changes due to hyperplasia of ductal epithelium were recognized as distention rather than narrowing but this condition occurred in limited numbers during advanced ductal alteration and was an insignificant criterion The most reliable tumor marker was complete ductal obstruction: ductal deterioration (displacement, deformity) occurred in 63% of the tumor cases, a pooling effect in 44% and a filling defect in 42%. The "chain of lakes" caused b ductular proliferation or adenomas, both of which seem t represent precursor stages of adenocarcinomas, was seen i both histologic and pancreatographic studies. Some neoplasms escaped diagnosis because of their small size, anatomic location, or specific histologic patterns. It is concluded tha pancreatography is unreliable as an early detection techniqu for induced pancreatic cancer and that additional clinica parameters are required. (19 refs)

78-3895 **Zinc Deficiency and Methylbenzylnitrosamine-induced Esophageal Cancer in Rats.** (Eng Fong, L. Y. (Dept. Biochemistry, Univ. Hong Kong, Hon Kong); Sivak, A.; Newberne, P. M. *J Natl Cancer Inst* 61(1) 145-150; 1978.

The role of zinc deficiency in esophageal cancer induced b methylbenzylnitrosamine (MBN) was studied in male outbred Charles River CD rats fed a control (containing 60 pp Zn) or a Zn-deficient (7 ppm Zn) diet. Groups of control an Zn-deficient rats were given 24, 17, 8, or 4 doses of MBN b intragastric intubation (2 mg/kg 2x/wk). All rats were kille and examined at 58-93 days after the beginning of treatment All rats receiving 24 doses of MBN beginning at 15 wk developed tumors; however, 43% of the tumor in Zn-deficien group were invasive carcinomas compared with 15% in th controls. After 17 doses beginning at 7 wk of age, all of th Zn-deficient rats at both 58 and 93 days and 33% and 66% of the control rats developed tumors. However, none of th controls developed invasive carcinoma, compared with 83% of the Zn-deficient rats sampled at 58 days and 33% sample at 93 days. Following ip injection of methyl-^{14}C-MBN, th DNA and RNA of esophagus and liver were more noticeabl labeled than the DNA and RNA of kidney, lung, and smal intestine. In vitro incubation of tissue slices from esophagus liver, kidney, and small intestine with ^{14}C produced simila results. These results indicate that Zn deficiency increases th incidence of and shortens the lag time for induction of esophageal tumors by MBN. Since Zn usually interacts wit other trace elements, these findings imply that the observe effects involve a trace element imbalance on a broader scale (20 refs)

78-3896 **Effect of Phenacetin and Caffeine on the Urinary Bladder of Rats Treated with N-Butyl-N-(4-hydroxybutyl)nitrosamine.** (Eng) Nakanishi, K. (First Dept. Pathology, Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467, Japan); Fukushima, S.; Shibata, M.; Shirai, T.; Ogiso, T.; Ito, N. *Gann* 69(3): 395-400; 1978.

The effect of phenacetin and caffeine on urinary bladder carcinogenesis was evaluated in male Wistar rats treated with N-butyl-N-(4-hydroxybutyl)nitrosamine (BBN). Animals were given a high (0.05%) or (0.01%) low dose of BBN for 4 wk and then phenacetin (2.5% in diet) or caffeine (0.1% in drinking water) was administered for 30 and 32 wk, respectively. All the animals were examined histologically after 36 wk. The incidence of papillary or nodular hyperplasia, papilloma, and cancer of the urinary bladder was significantly higher in the groups treated with BBN + phenacetin, especially in the group treated with 0.05% BBN + phenacetin, than in those treated with BBN alone. Simple hyperplasia and papillary or nodular hyperplasia developed in the urinary bladder of rats treated with phenacetin alone. Papillary proliferative growth of the renal pelvis was seen in one rat treated with a 0.01% BBN + phenacetin. Treatment with BBN + caffeine had no enhancing effect, and caffeine alone caused no remarkable changes. The results show that phenacetin enhanced BBN carcinogenesis and that this effect was additive. (18 refs)

78-3897 **Chemical Studies on Tobacco Smoke. LVI. Tobacco Specific Nitrosamines: Origins, Carcinogenicity and Metabolism.** (Eng) Hecht, S. S. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY); Chen, C. B.; Ornaf, R. M.; Hoffmann, D.; Tso, T. C. *IARC Sci Publ* (19): 395-413; 1978.

The formation, occurrence, and carcinogenicity of tobacco-specific nitrosamines are reviewed. The major tobacco-specific nitrosamine is N′-nitrosonornicotine (NNN), which is present in both unburned tobacco (0.3-90.6 ppm) and cigarette mainstream smoke (127-238 nanograms/cigarette). Model studies of the reaction of nicotine and nitrite showed that two other nitrosamines could be formed under mild conditions: 4-(N-methyl-N-nitrosamino) -4- (3-pyridyl) -1- butanal (NAA) and 4-(N-methyl -N-nitrosamino) -1- (3-pyridyl) -1- butanone (NNK). NNK was detected in both chewing tobacco and snuff. A comparative bioassay of NNN, NNK, and NNA in strain A mice indicated that NNK was more tumorigenic than NNN and that NNA was inactive. Sc injections of NNN into male and female Syrian golden hamsters (3x/wk for 25 wk; total dose, 375 mg) induced tracheal tumors in 12/19 hamsters within 83 wk. In a study of the metabolism of cyclic nitrosamines, metabolic α-hydroxylation of nitrosopyrrolidine, which is thought to be the critical step in activation of this compound, was demonstrated by detection in vitro and in vivo of 2-hydroxytetrahydrofuran, the decomposition product of α-hydroxynitrosopyrrolidine. (38 refs)

78-3898 **The Mutagenicity of Heterocyclic N-Nitrosamines for *Salmonella typhimurium*.** (Eng) Zeiger, E. (Environmental Mutagenesis Branch, Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC, 27709); Sheldon, A. T. *Mutat Res* 57(1): 1-10; 1978.

Fourteen carcinogenic and noncarcinogenic heterocyclic N-nitrosamines were evaluated for mutagenicity to *Salmonella typhimurium* TA1535, which responds to mutagens inducing base-pair substitutions. The nitrosamines tested were N-nitrosomorpholine (NM), 2,6-dimethyl-N-nitrosomorpholine (DMNM), N-nitrosothiomorpholine (NTM), 1-nitrosopiperazine (NPIPZ), 1,4-dinitrosopiperazine (DNPIPZ), 1-methyl-4-nitrosopiperazine (NMPIPZ), 2,6-dimethyl-1,4-dinitrosopiperazine (DMDNPZ), N-nitrosoazetidine (NAZ), N-nitrosopyrrolidine (NPYRR), N-nitrosopiperidine (NPIPD), N-nitrosohexamethyleneimine (NHMI), N-nitrosoheptamethyleneimine (NHPMI), N-nitrosooctamethyleneimine (NOMI), and N-nitrosododecamethyleneimine (NDMI). Both suspension and plate tests were used with mouse and rat liver metabolic activation systems. All nitrosamines tested, both carcinogenic and noncarcinogenic, showed a positive response in at least one test system. Among the nitrosomorpholine derivatives, NM was the most active mutagen in all systems, followed by DMNM and NTM. NPIPZ was the least mutagenic nitrosopiperazine tested. The series of compounds between NAZ through NOMI had a high level of mutagenic activity, but NDMI did not. The mutagenic responses obtained with mouse liver were generally equal to, or greater than, the responses obtained with rat liver in both the suspension and plate tests. Although it is difficult to make quantitative comparisons between plate and suspension tests, both systems appeared to be responsive to the same dose ranges for the individual nitrosamines. (29 refs)

78-3899 **N-Nitroso-5-methyl-1,3-oxazolidine Identified as an Impurity in a Commercial Cutting Fluid.** (Eng) Stephany, R. W. (Natl. Inst. Public Health, Bilthoven, Netherlands); Freudenthal, J.; Schuller, P. L. *Rec Trav Chim Pays Bas* 97(6): 177-178; 1978.

A commerical mineral oil cutting fluid concentrate was found, by various chromatographic analyses, to contain 30 mg/kg N-nitroso-5-methyl-1,3-oxazolidine. Identification was by gas chromatography-mass spectrometry using reference compounds. The N-nitrosamine, whose parent compound is a strong liver carcinogen, may have been formed from 1-amino-2-propanol. (6 refs)

78-3900 **The Effect of Carcinogens on the Nuclear Size of HeLa Cells.** (Eng) Agrelo, C. E. (British Industrial Biological Res. Assoc., Carshalton, Surrey, England). *Toxicology* 9(1/2): 21-27; 1978.

The possibility that changes in nuclear size might be used as an index of the carcinogenicity of compounds to which cells have been exposed was examined using HeLa S-3 cells. Optimal reaction conditions were the use of concentrations of chemicals that had a low order of cytotoxicity and the measurement of nuclear size 48 hr after pulse treatment of the cells for 30-60 min. When necessary for activation, rat liver microsome preparations were added. After exposure of HeLa S-3 cells to N-methyl-N-nitrosourea, N-dimethylnitrosamine, vinyl chloride, aflatoxin B$_1$, mitomycin C, or acetoxydimethylnitrosamine, nuclear size increased and the frequency distribution pattern of nuclear size changed. The increase in nuclear size was usually dose-related. These effects were not seen with noncarcinogens. The results suggest that changes in nuclear size may be an index of chemical carcinogenicity. (18 refs)

78-3901 **Induction of Tumors in Heterotopic Bladder by Topical Application of N-Methyl-N-nitrosourea and N-Butyl-N-(3-carboxypropyl)nitrosamine.** (Eng) Oyasu, R. (Dept. Pathology, Northwestern Univ. Medical Sch., Chicago, IL, 60611); Iwasaki, T.; Matsumoto, M.; Hirao, Y.; Tabuchi, Y. *Cancer Res* 38(9): 3019-3025; 1978.

Bladders from CD Fischer rats were transplanted into the gluteal muscle of rats of the same strain and connected to a reservoir implanted into the sc tissue at the back. This system is a potentially useful model for bladder carcinogenesis studies. Two carcinogens, N-methyl-N-nitrosourea (MNU) and N-butyl-N-(3-carboxypropyl)nitrosamine (BCPN), were instilled repeatedly into the reservoir connected with the heterotopic bladder. Transitional cell carcinomas developed in 25/33 heterotopic bladders exposed to cumulative doses of 1.5, 3.0 or 6.0 mg MNU for 20-30 wk, but heterotopic bladders exposed to cumulative doses of 150 or 300 mg BCPN failed to develop tumors. However, 11/27 rats with heterotopic bladders that were exposed to BCPN for $>$ 20 wk developed tumors in their homotopic or natural bladders. MNU probably acted directly on the bladder epithelial cells to induce neoplastic change. The reason(s) for the development of tumors in the homotopic but not heterotopic bladders when BCPN was administered directly into the heterotopic bladders could not be ascertained. (18 refs)

78-3902 **Pathogenesis of Rat Colon Carcinomas Induced by N-Methyl-N-nitrosourea.** (Eng) Lev, R. (Dept. Pathology, Roger Williams General Hosp., 825 Chalkstone Ave., Providence, RI, 02908); Herp, A. *J Natl Cancer Inst* 61(3): 779-786; 1978.

Colon specimens were obtained from 45 young male and female inbred CDF rats between 11 and 42 wk after they began to receive intrarectal injections of the carcinogen N-methyl-N-nitrosourea (MNU: 43-108 mg, total dose). Groups of animals were given 2 mg/wk for 1 mo, then 2.5 mg 1x or 2 x/wk for 20 wk. Specimens were also obtained from 26 control rats. Well-developed tumors from rats receiving MNU generally presented grossly as polyps and histologically as invasive adenocarcinomas that had not metastasized. These tumors resembled their human counterparts. Focal epithelial atypias were found in grossly normal mucosa both from rats that had not yet developed tumors and from tumor-bearing rats. These foci typically consisted of low columnar cells with enlarged nuclei, increased number of mitoses, cytoplasmic basophilia, and reduced cytoplasmic mucus. Serial section studies of minute foci of atypia indicated that some of them arose from normal deep crypt cells. Transitions were frequently found between atypical foci and in situ or invasive carcinomas. Less commonly, adenomatous epithelium was identified in the early lesions or within the frank adenocarcinomas. It was concluded that most invasive carcinomas in this rat model originate in foci of epithelial atypia, which are found with increasing frequency in the flat mucosa during treatment with MNU, but that some carcinomas also arise from adenomatous epithelium. (25 refs)

78-3903 **In Vivo DNA Repair after N-Methyl-N-nitrosourea Administration to Rats of Different Ages.** (Eng) Arfellini, G. (Istituto di Cancerologia, Universita di Bologna, via S. Giacomo 14, 40126 Bologna, Italy); Grilli, S.; Prodi, G. *Z Krebsforsch* 91(2): 157-164; 1978.

In vivo DNA repair was studied in female Wistar rats inoculated ip with N-methyl-N-nitrosourea (MNU: 0.1 mg/g) at age 10 days, age 2 mo, or on the 18th day of pregnancy. The rats received 12 im injections of ^{3}H-thymidine (0.75 μCi/g) before being killed 8, 15, or 22 hr after MNU treatment. Hydroxyurea (0.5 mg/g) was also administered prior to sacrifice (3 ip injections) to inhibit scheduled DNA synthesis. In young adult liver, DNA repair was complete by 22 hr, the time at which repair began in newborn rat liver. Liver DNA repair was max at 15 hr in the pregnant rats, but it was undetectable in their fetuses. There was no evidence of DNA repair in the kidney and brain (MNU target organs) of rats from the different age groups. The higher sensitivity of the fetus and the neonate to the oncogenicity of MNU may be partly due to an impairment in DNA repair. (22 refs)

78-3904 **Histochemistry of Acid Mucopolysaccharides During the Genesis of Brain Tumors Induced in Rats by Methylnitrosourea.** (Ger) Engelhardt, A. (Abteilung fur Cytopathologie, Institut fur Experimentelle Pathologie am Deutschen Krebsforschungszentru, Postfach, D-6900 eidelberg, W. Germany); Bannasch, P. *Acta Neuropathol* *Berl)* 42(3): 197-204; 1978.

arly histologic and histochemical changes that occurred uring the genesis of brain tumors induced in 66 Wistar rats y chronic iv administration of methylnitrosourea (5 ng/kg/wk) were studied. After a lag period of 225-300 days, 0 CNS tumors developed: 8 gliomas, 33 gliomatous microtunors, 8 sarcomas, 5 sarcomatous microtumors, and 6 eurinomas. In addition, 15 areas with characteristic histohemical changes, but without cell proliferation, were found. cid mucopolysaccharides could be demonstrated in these reas by the iron binding reaction or by Alcian Blue staining eaction (Alcian-positive areas without cell proliferation). he same histochemical changes were seen in all gliomatous nicrotumors (Alcian-positive areas with cell proliferation) nd in fully developed gliomas. The positive staining reaction or acid mucopolysaccharides was diffuse or weblike. In the enter of the larger gliomas, Alcian positivity tended to disapear. Alcian-positive areas with and without cell proliferation nd fully developed gliomas showed a similar localization: hey involved preferentially the white matter and regions near he lateral ventricles. These observations show that gliomas riginate from Alcian-positive areas rich in acid mucopolyaccharides. The histochemical findings point to an early disegulation of carbohydrate metabolism during the neoplastic ransformation of glial cells. In contrast to gliomas, sarcomas ever accumulate acid mucopolysaccharides. This histohemical difference may help to distinguish between gliomas nd sarcomas. (36 refs)

8-3905 **Nitrosourea-induced Brain Tumors: An In Vivo and In Vitro Tumor Model System.** (Eng) Yohida, J. (Dept. Neurosurgery, Sch. Medicine, New York Jniv. Medical Center, 550 First Ave., New York, NY, 0016). *J Natl Cancer Inst* 61(2): 365-374; 1978.

he tumorigenicity of methylnitrosourea-induced glioma nd an ethylnitrosourea-induced neurinoma was subtantially increased when the tumors were serially subultured in vitro or serially transplanted sc in 30-day-old DF rats. In early passages, the two tumor types were readily listinguishable, but after 10 in vivo passages and 20 in vitro assages, the cells became indistinguishable. However, basic lifferences could be demonstrated electron microscopically fter as many as 87 in vitro passages. The tumorigenicity (in ransplantation experiments) of these tumors was classified nto four grades: (I) negative tumorigenicity; (II) positive but ot consistent tumorigenicity; (III) consistent 100% umorigenicity but erratic tumor latency period; (IV) consistnt 100% tumorigenicity with consistent tumor latency perid. The neurinoma required a lower number of in vivo and n vitro passages to obtain Grade IV tumorigenicity than did he glioma. In transplantation experiments, immunosuppresion (nonlethal whole-body x-radiation or neonatal thymectomy) of the mice hastened tumor appearance significantly compared with that in untreated controls. (24 refs)

78-3906 **Analysis of N-Ethyl-nitrosourea-induced Brain Carcinogenesis by Sequential Culturing During the Latent Period. I. Morphology and Tumorigenicity of the Cultured Cells and Their Growth in Agar.** (Eng) Roscoe, J. P. (Sch. Pathology, Middlesex Hosp. Medical Sch., Riding House St., London W1P 7LD, England); Claisse, P. J. *J Natl Cancer Inst* 61(2): 381-390; 1978.

Pregnant BD IX rats were given ip injections of 40-50 mg/kg N-ethyl -N- nitrosourea on gestation day 15 or 16, and brain cultures were prepared from fetuses and offspring at 2, 3, 5, 34-35, 57-60, 90-91, 111-112, and 138-145 days postinjection (PI). The cultures were examined for morphology, growth in soft agar, and tumorigenicity upon sc injection into syngeneic rats. From 90 days PI onward, cells resembling glioma cells were observed. These cells, termed basal layer cells (BLC), were not observed in control cultures. The BLC in cultures prepared 90-91 days PI did not persist and they did not grow in soft agar or produce tumors. From 111 days PI onward, however, the BLC predominated and they behaved like malignant cells. In general, the longer the time lapse before removal of the cells for culture, the more closely the cells resembled tumor cultures. Three main morphologic types of BLC that occurred in different proportions in the cultures were observed: fried eggs, crescents, and pyramids. The pyramidal type was associated with a shorter tumor latency; the fried egg type was predominant in cultures prepared 90-91 days PI, in which BLC disappeared upon passaging. The apparent progression with increasing malignancy is suggested to be (fried egg, crescent) -> pyramid -> less obviously pyramidal with further reduction in size. Cultures prepared < 90 days PI did not contain BLC and did not grow in agar or in animals at early passages. However, cultures derived 2 days PI became tumorigenic after prolonged culturing (>44 passages), which indicated that they had already acquired malignant potential. (18 refs)

78-3907 **Analysis of N-Ethyl-N-nitrosourea-induced Brain Carcinogenesis by Sequential Culturing During the Latent period. II. Morphology of the Tumors Induced by Cell Cultures.** (Eng) Claisse, P. J. (Sch. Pathology, Middlesex Hosp. Medical Sch., Riding House St., London W1P 7LD, England); Lantos, P. L.; Roscoe, J. P. *J Natl Cancer Inst* 61(2): 391-397; 1978.

Pregnant BD IX rats were inoculated ip with 40-50 mg/kg N-ethyl-N-nitrosourea (ENU) on gestation day 15 or 16. Cultured cells derived from brains of fetuses and offspring 2, 111-112, and 138-145 days postinjection (PI) were injected sc into syngeneic rats, and the histology of the induced tumors was compared. Some of the cultures taken at each of the PI times formed malignant growths that invaded and destroyed surrounding tissues. The tumors exhibited different structural patterns in various areas. A central area of loosely distributed, large, polygonal cells contrasted with bundles of smaller fusiform cells at the periphery. These patterns might reflect two processes occurring within the tumors: tissue organization and cellular differentiation. Three morphologic groups of cells could be distinguished: bipolar-fusiform (B-F), polygonal-stellate (P-S), and binucleate-multinucleate (B-M). Phosphotungstic acid/hematoxylin (PTAH) stains allowed separation of the B-F cells into glioblasts with weakly positive glial fibers and collagen-producing cells. P-S and B-M cells were of astrocytic derivation with evidence of glial fiber production. Varying degrees of pleomorphism were observed in all tumors; however, decreasing pleomorphism correlated well with increasing time between ENU treatment and culture initiation. Tumors from cultures derived at 2 days were the most pleomorphic although they were unmistakably glial, as demonstrated by the presence of many PTAH-staining fibrils. Cellular pleomorphism decreased with increasing number of transfers in tumors from some cultures prepared 111-112 days and 138-145 days PI, but not in those derived 2 days PI. (17 refs)

See also:

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*(Phys.): 78-3912, 78-3914, 78-3921, 78-3943.

*(Viral): 78-3968, 78-3974, 78-3975, 78-4052.

*(Immun.): 78-4064, 78-4075, 78-4079, 78-4082.

*(Path): 78-4099, 78-4103, 78-4108, 78-4120, 78-4123, 78-4126, 78-4127, 78-4135.

*(Epid.-Biom.): 78-4141, 78-4144, 78-4145, 78-4146, 78-4156, 78-4158, 78-4161, 78-4164, 78-4165, 78-4166, 78-4171, 78-4176, 78-4178, 78-4180, 78-4181, 78-4184, 78-4185, 78-4186.

78-3908 **Chromosome Aberrations and Unscheduled DNA Synthesis in X- and UV-irradiated Lymphocytes from a Boy with Bloom's Syndrome and a Man with Xeroderma Pigmentosum.** (Eng) Evans, H. J. (MRC Clinic and Population Cytogenetics Unit, Western General Hosp., Crewe Road, Edinburgh, EH4 2XU, Scotland); Adams, A. C.; Clarkson, J. M.; German, J. *Cytogenet Cell Genet* 20(1-6): 124-140; 1978.

Blood lymphocytes from a 6.5-yr-old boy with Bloom's syndrome (BS) and a 51-yr-old man with a mild form of xeroderma pigmentosum (XP) were examined for their response to UV radiation, as measured by unscheduled DNA synthesis levels, and to x radiation, as measured by chromosome aberrations. Although the child with BS showed facial skin sensitivity to sunlight, his lymphocytes had a normal unscheduled DNA synthesis response. In contrast, cells from the XP patient showed a 50% reduction in unscheduled DNA synthesis following UV exposure. Cytogenetic studies of phytohemagglutinin-stimulated BS lymphocytes revealed a typical increase in the incidence of aberrations. Many of the cells showed breakages located at the centromere and equal, symmetrical chromatid interchanges involving homologous chromosomes. The aberrations were not produced in control cells cultured in serum from the BS patient. In contrast, the frequency aberration spontaneous in XP lymphocytes was similar to that in controls. When G_1 cells from both patients were exposed to a range of x-ray doses, they did not differ from controls in the incidence of induced chromosome-type aberrations. The significance of this finding is discussed in relation to the response of BS cells to chemical mutagens. (39 refs)

78-3909 **Repair Replication in Replicating and Non-replicating DNA after Irradiation with UV Light.** (Eng) Slor, H. (Dept. Human Genetics, Sackler Sch. Medicine, Tel-Aviv Univ., Tel-Aviv, Israel); Cleaver, J. E. *Nucleic Acids Res* 5(6): 2095-2098; 1978.

The ability of replicating and nonreplicating Hela cell DNA damaged by UV light to undergo repair replication was studied. The percentage of dimers in parental DNA strands from replicated DNA (0.147) was higher than that from the unreplicated DNA (0.032). The results suggest that more dimers are formed in replicons that are about to replicate their DNA than in the bulk DNA. About seven times more repair replication occurred on the parental strands of replicating DNA than in the bulk DNA, and it appeared that not all the parental strands were repaired to the same extent. The data indicate that replicons that replicate their DNA during the first 2-3 hr after UV irradiation contain more dimers and undergo more repair replication, involving larger patches, than do replicons that do not replicate at that time. It is possible that special conformations that may be associated with DNA replication cause a greater efficiency in dimer formation and a concomitant increase in repair replication. (11 refs)

78-3910 **Effect of Caffeine on DNA Polymerase I from *Escherichia coli*. Studies In Vitro and In Vivo.** (Eng) Solberg, K. A. (Dept. Biochemistry, Univ. Bergen, Bergen, Norway); Ovrebo, S.; Kleppe, R. K.; Kleppe, K. *Mutat Res* 51(1): 1-10; 1978.

The effect of caffeine on the activity of DNA polymerase I from *Escherichia coli* was studied. Caffeine had no effect on polymerizing activity, but it did inhibit both 5' --> 3' and 3' --> 5' nuclease activities of DNA polymerase I. The inhibition was most marked with d(A-T)n as substrate and less marked with T_7 DNA. At 10mM caffeine, inhibition was 50%. Caffeine had little effect on the ratio between mono- and dinucleotides. Sensitivity to UV irradiation and the effect of caffeine on dark repair was studied in vivo in three isogenic strains of *E. coli* carrying different mutations in the DNA polymerase I gene. The effect of caffeine on survival after UV irradiation was most marked for the wild-type, *pol+*, and less marked for the mutants defective in 3' --> 5' (*polA1*) and 5' --> 3' (*polA107*) nuclease activities. Caffeine had little effect on the survival of the *resA1* mutant, which lacks both 5' --> 3' and 3' --> 5' nuclease activities. The data support the concept that the influence of caffeine on dark repair may be explained in part by its effect on the nuclease activities of DNA polymerase I. (38 refs)

78-3911 **Postreplication Repair of DNA in Ultraviolet Light-irradiated Normal and Malignantly Transformed Mouse Epidermal Cell Cultures.** (Eng) Bowden, G. T. (NCI, NIH, Building 37, Room 3A19, Bethesda, MD, 20014); Giesselbach, B.; Fusenig, N. E. *Cancer Res* 38(9): 2709-2718; 1978.

Alkaline sucrose gradient analysis of pulse-labeled single-stranded DNA was used to study the effect of UV irradiation (wavelength, 254 nanometers; rate, 1 joule/m^2/sec) on the size of newly synthesized DNA in primary cultures of newborn C3H mouse epidermal cells and in an in vitro malignantly transformed epidermal cell line. The latter was derived by treatment of a primary culture of B10Lp mouse epidermal cells with 10^{-6} M dimethylbenz(a)anthracene. In both cell types, the DNA that was synthesized after UV irradiation was smaller. This smaller size may have been due to gaps in the newly synthesized DNA or to a slower rate of DNA polymerization. Therefore, the pulse time for the treated cells was increased so that equal amounts of DNA synthesis occurred in control and treated cells. Under these conditions, the pulse-labeled DNA's were very similar in size, which suggested that the rate of polymerization had been slowed by UV and that no gaps were present. However, it was possible that gaps were present but were being closed rapidly. Caffeine was therefore used to inhibit gap filling under conditions of equalized DNA synthesis in control and treated cells. When caffeine was present during the extended pulse, the newly synthesized DNA was again smaller in the UV-treated cells than in the control cells. According to these results, gaps do occur in newly synthesized DNA of UV-irradiated epidermal cells. Pulse-chase experiments showed that the gaps in the newly synthesized DNA from irradiated cells were filled in and that the DNA was joined and elongated in both cell types. These experiments demonstrate that both normal primary and malignantly transformed epidermal cells exhibit a postreplication gap-filling mechanism in response to UV irradiation. (46 refs)

78-3912 **Mutation to Ouabain-Resistance in Chinese Hamster Cells: Induction by Ethyl Methanesulphonate and Lack of Induction by Ionising Radiation.** (Eng) Thacker, J. (MRC Radiobiology Unit, Harwell, Didcot, Oxon OX11 0RD, England); Stephens, M. A.; Stretch, A. *Mutat Res* 51(2): 255-270; 1978.

The effect of ionizing radiation on the frequency of ouabain-resistant (OUAr) mutants in Chinese hamsters V79-4 cell cultures was examined. Ethyl methanesulfonate (EMS) was used as a positive control mutagen. In uncloned V79-4 cells, the spontaneous frequency of OUAr was approx 5×10^{-5}/viable cell. This frequency was initially reduced to approx 1×10^{-6} in 1 mM ouabain in freshly isolated clones or cultures started from a few cells. No increase in frequency of OUAr mutants was observed in cultures treated with γ-rays. Variation in radiation dose, ouabain concentration, posttreatment interval prior to selection, cell density in selective medium, and clonal state of the cells at the time of ouabain administration (in situ vs respreading technique) had no effect. Negative results were also observed with accelerated helium ions. In cultures treated with various concentrations of EMS, reproducibly large increases in the frequency of OUAr were observed. The relevant literature is reviewed, and the suggestion that ionizing radiation cannot induce OUAr mutants because of the severe genetic damage that it causes is discussed. (35 refs)

78-3913 **Xeroderma Pigmentosum Neurological Abnormalities Correlated with Colony-forming Ability after Ultraviolet Radiation.** (Eng) Andrews, A. D. (Dept Dermatology, Columbia Univ., Coll. Physicians and Surgeons, New York, NY, 10032); Barrett, S. F.; Robbins, J. H *Proc Natl Acad Sci USA* 75(4): 1984-1988; 1978.

The relationship between the neurological abnormalities o xeroderma pigmentosum (XP) and the sensitivity of cell from XP patients to UV radiation was examined in 24 X fibroblast strains. Sensitivity to UV radiation was studied i vitro by measuring the ability of each strain to divide an form colonies after irradiation. The most sensitive strain were derived from patients who had an early onset of neurological abnormalities, less sensitive strains were from patient with a later onset, and the most resistant strains were fro patients without neurological abnormalities. The UV sensitivities of strains from each member of a sibling pair wit XP were identical, indicating that UV sensitivity of X strains is determined by the patient's inherited DNA repai defect. The results suggest that effective DNA repair is required to maintain the functional integrity of the huma nervous system by preventing premature death of neurons (18 refs)

78-3914 **Inhibition and Enhancement of the Action o Mutagens by Chemical, Physical an Viral-modulating Factors (Meeting Abstract).** (Eng) Lo, L (Dept. Medical Genetics, Univ. British Columbia, Vancouver, British Columbia, Canada); Lam, P.; Stich, H. F. *Muta Res* 53(2): 221-222; 1978. (no refs)

78-3915 **The Use of Cytosine Arabinoside for Studyin the Rate and Extent of DNA Repair in Mammalian Cells Following Irradiation or Chemical Treatmen (Meeting Abstract).** (Eng) Preston, R. J. (Biology Div., Oa Ridge Natl. Lab., Oak Ridge, TN, 37830); Hiss, E. A. *Muta Res* 53(2): 250; 1978. (no refs)

78-3916 **Serologic Assay of DNA Base Damage. I 5-Hydroxymethyldeoxyuridine. A Radiatio Product of Thymidine.** (Eng) Lewis, H. L. (Lab. Nuclea Medicine and Radiation Biology, Univ. California, Los Angeles, CA, 90024); Muhleman, D. R.; Ward, J. F. *Radiat Re* 75(2): 305-316; 1978.

A serologic assay was developed for 5-hydroxymethyldeoxyuridine (5-HMUdR), an ionizing radiation product o thymidine (Thd). Antibodies were prepared to a bovin serum albumin conjugate of 5-hydroxymethyluridin (5-HMUR). Using a phage-neutralization assay, 0.1 nanomole of 5-HMUR could be detected. The antiserum cross-reacted with Thd but with no other naturally occurrin DNA component. There was cross-reaction with uridine 6-methyluracil, and the halogenated analogs of Thd. Interference by cross-reaction with Thd was eliminated by saturation of the assay with excess Thd. This also simulates the conditions for radiation studies, in which a small quantity of 5-HMUdR is determined in the presence of an excess of the parent compound, Thd. The assay was applied to the estimation of 5-HMUdR produced by irradiation of Thd in O_2. Irradiation at pH 14 increased the yield nine times over that obtained at neutral pH. Postirradiation heating at pH 9 or treatment with 10^{-3} ferrous ions, procedures that convert Thd methyl peroxides to 5-HMUdR, also increased product yield. Estimation of 5-HMUdR in DNA irradiated in O_2 gave results similar to those obtained with Thd. This yield is based on the assumption that binding of the antibody to the 5-hydroxymethyluracil moiety in DNA and to free 5-HMUR is similar. 5-MHUdR was detected at one nucleotide per 3 x 10^6 daltons of DNA. The results suggest that this serologic approach will be generally applicable to the detection and quantitation of a number of radiation products in DNA. (45 refs)

78-3917 **Radiation Damage to DNA in Aqueous Solution: A Comparison of the Responses of the Single-stranded Form with That of the Double-stranded Form.** (Eng) Ward, J. F. (Lab. Nuclear Medicine and Radiation Biology, Univ. California, Los Angeles, CA, 90024); Kuo, I. *Radiat Res* 75(2): 278-285; 1978.

The responses of single-stranded (ss) and double-stranded (ds) DNA to radiation in aqueous soln were compared. Comparison of the absorption spectra of transient DNA-OH radicals immediately after pulse radiolysis indicated that the spectrum of the transient DNA-OH radical formed from ssDNA has a higher absorbance than that of the dsDNA-OH radical. After 400 μsec in the presence of oxygen, there was little change in the spectrum from dsDNA, but an appreciable decay was seen in the spectrum from ssDNA. This decay was complete in 400 μsec and did not occur in the absence of oxygen. These results suggest that the dsDNA-OH transient does not react with molecular oxygen but that ssDNA-OH radical does. The yield of strand breaks in neutral soln was 2.5 times as high for dsDNA as for ssDNA suggesting greater relative sugar damage in dsDNA. The number of alkali-produced strand breaks was higher than that seen in neutral solns for both DNA's but those for ssDNA were almost double those seen with dsDNA. The higher yield of alkali-produced breaks indicates more base damage. In the presence of oxygen, the yield of free thymine was almost the same in both DNA's; this yield can be correlated with sugar damage. The yields of damaged bases were much lower in dsDNA than in ssDNA. It is hypothesized that the nucleic acid bases are protected inside the double helix against the reactions of OH free radicals. Thus, in assessing damage to native DNA, radiation damage to deoxynucleotides does not provide an accurate model. (19 refs)

78-3918 **Spontaneous and Radiation-induced Benign Tumors in Parabiont Rats.** (Eng) Warren, S. (Cancer Res. Inst., New England Deaconess Hosp., Boston, MA, 02215); Gates, O.; Brown, C. E.; Chute, R. N.; Porter, M. W. *JAMA* 75(1): 98-107; 1978.

The incidence of spontaneous benign tumors in NEDH rats, as well as of those induced by parabiosis and radiation, is reported. NEDH rats of the same age and sex were parabiosed at about 35 days of age. At 110 days of age, the right-hand members were irradiated with a single whole-body exposure of 1,000 R of 250-kilovolt peak x-rays. The left-hand members were shielded to ensure survival of their partners from the otherwise fatal x-ray dose. There were three series of controls: single untreated rats; parabiosed, untreated rats; and the shielded partners of the irradiated rats. The radiation produced high incidence rates of benign tumors in several radioresponsive organs: ovary, 49.7%; adrenal medulla (males, 23.9%; females, 15.2); mammary tissue (female 19.6%); islands of Langerhans (males, 15.3%); liver (cholangiomas: males, 7.4%; females, 13.8%). Both hormonal imbalance and radiation effects appear to be involved. Endometrial polyps, although more frequent in irradiated than in shielded partners, probably resulted from hormonal imbalance. In females, the incidence of pituitary adenomas was four times as high in the shielded partners (16%) as in the irradiated partners (4.2%), probably the result of sterilization of the ovaries in the latter. Parabiosis decreased the incidence of pheochromocytoma, but irradiation of a partner increased it. Mammary tumors occurred an av of 200 days earlier in irradiated rats than in their unirradiated controls. Among the many benign lesions studied, only a few showed changes indicating transition to a malignancy. (21 refs)

78-3919 **Dose-Incidence Variations of Reticulum Cell Sarcoma in Mice with Irradiated Bone Marrow.** (Eng) Covelli, V. (Lab. Radiation Pathology, Centro di Studi Nucleari della Casaccia, Comitato Nazionale per l'Energia Nucleare, Casella Postale 2400, 00100 Rome, Italy); Ballardin, E.; Di Majo, V.; Bassani, B.; Metalli, P. *J Natl Cancer Inst* 61(4): 1131-1133; 1978.

The incidence of reticulum cell sarcoma (RCS) in irradiated and limb-shielded [C57BL/Cne(female) x C3H/Cne(male)]-F1 male mice as a function of radiation dose to the shielded marrow was determined. When used, the shields were kept in place over one or both hind legs for only part of the time required to irradiate the rest of the body. The av final incidence of RCS in untreated control mice was 57%, and it was not significantly changed by single acute whole-body doses of x-rays up to 400 rads. Higher doses sharply decreased this incidence to very low levels. In a syngeneic chimera system, however, irradiation (400 rads) of bone marrow prior to transplantation significantly increased the frequency and rate of RCS relative to transplantation of unirradiated marrow. An endogenous repopulating system was tested; marrow was irradiated in situ by a limb-shielding technique. The results indicated a biphasic dose-incidence curve with a peak at 500 rads. The rising part of the curve agreed with and extended the conclusion from the exogenous system, whereas the descending portion at higher doses paralleled the trend observedin whole-body x-irradiated animals. Finally, the irradiation of two hind legs resulted in a higher frequency of RCS than did irradiation of only one leg at the same dose levels. The results strongly indicated that the frequency of radiation-induced tumors in marrow-irradiated mice is correlated with the mass and, hence, with the numbers of cells at risk of being transformed by irradiation. (9 refs)

78-3920 **Decline of Natural Killer Cell Activity in Sublethally Irradiated Mice.** (Eng) Hochman, P. S. (Dept. Pathology, State Univ. New York, Buffalo, NY, 14214); Cudkowicz, G.; Dausset, J. *J Natl Cancer Inst* 61(1): 265-268; 1978.

Spleen cells of (C57BL/6 x C3H/He)F$_1$ mice were assayed for natural killer (NK) cell activity against YAC-1 and FBL-3 lymphoma targets at intervals after total-body exposure to a high sublethal dose of ^{137}Cs or ^{60}Co x-rays. NK cell activity did not decline for the first 12 days, but there was a marked loss of cellularity in the spleen (80%-90%). During the subsequent period of rapid regeneration of splenic cellularity, NK cell activity decreased sharply, so that the values of specific lysis were $< 10\%$. The earliest drop of NK cell activity was detected on day 14; activity became minimal on days 17 and 19. During the fourth and fifth weeks, cellularity was 60%-90% of controls and NK cell activity was low but tendency toward recovery was noted. Near normal levels of NK activity were reached 6-8 wk after irradiation. Suppressor cells were detectable during the period of lowest NK cell activity, and they may have been responsible for the delayed and slow recovery. It is concluded that (1) mature effectors of natural cytotoxicity are relatively radioresistant renewable cells with a life-span of 2 wk whose progenitors are radiosensitive cells and (2) the kinetics of decline and recovery of NK cell activity may be influenced by suppressor cells. (12 refs)

78-3921 **Effect of Ovariectomy on X-Ray Carcinogenesis in Rats.** (Eng) Takizawa, S. (Dept. Cancer Res., Res. Inst. Nuclear Medicine and Biology, Hiroshima Univ., Kasumi 1-2-3, Hiroshima 734, Japan); Naito, Y.; Watanabe, H.; Hirose, F. *Gann* 69(3): 353-360; 1978.

Tumor induction by fractionated whole-body x-radiation (400 rads) was studied in ovariectomized (OV-X), OV-X and ovary-grafted (OV-X,OV-G), and sham OV-X (S-OV-X) Sprague-Dawley rats. Ovariectomy was performed 3 days before the last (4th) radiation treatment. OV-X rats gained more body wt and responded more quickly (but transiently) in recovery of WBC levels after the last x-irradiation than the other rats. A total of 26 tumors developed in 21/47 OV-X rats, 11 tumors in 6 /13 OV-X,OV-G rats, and 44 tumors in 25/29 S-OV-X rats during the observation period (up to 64 wk after the starting of x-irradiation). In the latter two groups, 80% of the tumors were of mammary gland origin. In contrast, 61.1% of tumors in the OV-X rats were derived from sc mesenchymal tissue and hematopoietic tissue. This indicates that some forms of mesenchymal tumors, including leukemia, may be under the suppressive influence of female sex hormones. (12 refs)

78-3922 **Ovarian Tumor Types and Their Incidence in Intact Mice Following Whole-Body Exposure to Ionizing Radiation.** (Eng) Clapp, N. K. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830). *Radiat Res* 74(3): 405-414; 1978.

In a large-scale experiment, 3,135 8-wk-old RF/Un mice were exposed to 0-400 rads of whole-body radiation with 60-million electron volt protons or 300-kilovolt peak x-rays in single doses at a rate of 80-100 rads/min, and the incidence and types of ovarian tumors produced were determined. Since there were only minor differences in effect between the two treatments, both groups were combined by dose for analysis of the data. Ovarian tumors developed in 3.8% of the untreated mice and 50%-60% of those given 50-100 rads; the incidence declined gradually with 100-400 rads. Tumor cell types were identified histologically in 2,400 mice. There were 479 single cell tumors [309 tubular adenomas (TA), 85 granulosa cell (GC) tumors, 60 luteomas (L), 17 hemangiomas, 6 adenocarcinomas, 2 fibrosarcomas] and 467 mixed cell tumors (123 bilateral mixed tumors, 112 of which were L; 289 L in one ovary, 42 GC + L and/or TA; 13 miscellaneous types). At 0-100 rads, the relative proportions of mixed cell tumors increased and single cell tumors decreased; they were about equal at 100-400 rads. Approx 90% of the ovarian tumors at 50 rads contained TA elements; this decreased to 70% at 400 rads. L increased to 40% at 50 rads and 60% at 100 rads, but showed no dose effect at 100-400 rads. GC tumors showed no dose effect, remaining at an incidence of 15%-20%. At 50 rads, TA were most common, L were intermediate, and GC tumors were lowest in occurrence. The prevalence of tumor cell types suggests a relative sensitivity of ovarian tissues to radiation-induced tumorigenesis after whole-body ionizing radiation. (14 refs)

78-3923 **Role of Age in Tumorigenesis in Irradiated Animals.** (Rus) Pavlenko-Mikhailov, Iu. N. (No affiliation given); Stre'tsova, V. N.; Strashnenko, S. I.; Oshchepko, A. B. *Radiobiologiia* 18(2): 218-222; 1978.

The association between age and the tumorigenic effect of radiation was studied in random-bred albino rats. Newborn rats were divided into five groups: Groups 1-4 (treated at 1, 2, 4 wk and at 4 mo of age, respectively) were given a single dose of 200 R radiation, and Group 5 served as controls. Irradiation significantly increased the frequency of ovarian, pituitary, and mammary gland tumors. Ovarian tumors were detected only in Groups 1-3: 10/18, 11/30, and 11/28 rats, respectively, on day 800 postirradiation. On day 540, pituitary tumors were found in 5/18 rats in Group 1, 5/30 rats in Group 2, 6/28 rats in Group 3, and 36/50 rats in Group 4, compared with 1/30 rats in Group 5. The incidence of mammary gland tumors was significantly greater in Group 4 (38/50, vs 5/18, 7/30, and 8/28 in Groups 1, 2 and 3, respectively). The incidence in Group 5 was 4/20. The latent period of mammary gland tumors in Groups 1-4 was decreased by 500, 470, 380, and 120 days, respectively. (11 refs)

78-3924 Characteristic Features of the Development of Radiation-induced Tumors of the Stomach and Intestine. (Rus) Lebedeva, G. A. (No affiliation given). *Vopr Onkol* 24(7): 73-77; 1978.

The carcinogenic effect of ionizing radiation was studied in dogs and rats. Animals were divided into three groups: Group 1 consisted of 52 dogs subjected to mixed gamma-neutron radiation at doses ranging from 272 to 2,351 biological equivalent of roentgen (ber). Group 2 consisted of 25 dogs subjected to sc implantation of the alpha-emitter ^{210}Po (2.5 μCi/kg). Group 3 consisted of 250 rats who received the beta-emitter ^{144}Ce (9, 12, 15, and 18 μCi/g) intragastrically. Dogs were sacrificed 11.5 mo to 11.4 yr later, rats 1-26 mo later. The av latent period of tumor development in Groups 1, 2, and 3 was 7.4 yr, 9.2 yr, and 11.0 mo, respectively. Dogs in Groups 1, 2, and 3 was 7.4 yr, 9.2 yr, and 11.0 mo, respectively. Dogs in Groups 1 and 2 had predominantly benign tumors (100% and 93.3%, respectively), but 78.3% of the rats developed malignant tumors. Approx 50% of the malignant tumors were located in the stomach and intestine. (11 refs)

78-3925 Gamma-Ray-induced Dominant Mutations That Cause Skeletal Abnormalities in Mice. II. Description of Proved Mutations. (Eng) Selby, P. B. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Selby, P. R. *Mutat Res* 51(2): 199-236; 1978.

Breeding tests were used to determine if γ-radiation-induced dominant skeletal mutations in male 101 strain mice can be transmitted. Spermatogonia irradiated with 100 R + 500 R γ-rays, the mice were bred with C3H females, and the F1 males were outcrossed with three (101 x C3H)F1 females. The F1 males were killed, and their skeletons were cleared and examined for abnormalities. If a male had one or more unusual skeletal abnormalities, his progeny (FOP) were saved and approx half of them were examined for abnormalities. If the FOP clearly had abnormalities transmitted from the sire, they were further outcrossed and their offspring examined for anomalies. Thirty-one dominant skeletal mutations were observed, each of which is described in detail. The data provide the basis for determining which mutations cause effects that would, if they occurred in humans, cause a serious handicap. Most of the mutations resulted in multiple abnormalities. They exhibited low penetrance for many of their effects. Many dominant skeletal mutations produced a general stunting of body growth. Externally visible effects found in some heterozygotes for a few mutations included hydrocephalus, circling behavior, increased nervous activity, gray color coat, webbing of digits, and small size. The results indicate that some of the mutations may be reciprocal translocations; however, cytological testing of most of the mutant lines gave no indication of chromosome aberrations. (14 refs)

78-3926 Induction of Hepatocellular Tumor by X-Ray Irradiation at Perinatal Stage of Mice. (Eng) Sasaki, S. (Div. Radiation Hazards, Natl. Inst. Radiological Sciences, Anagawa 4-9-1, Chiba 280, Japan); Kasuga, T.; Sato, F.; Kawashima, N. *Gann* 69(3): 451-452; 1978.

The incidence of hepatocellular carcinoma after x-ray irradiation was determined in the F_1 hybrids of C57BL/6 x WHT/Ht (B6WF$_1$) mice. The mice were grouped as follows: Group 1, unirradiated controls; Group 2, irradiated in utero at 17 days' postcoitum with 150 or 300 R; Group 3, irradiated within 24 hr after parturition with 400 R; Group 4, females only, irradiated at 5 wk of age with 400 R. The incidence of hepatocellular carcinoma was markedly enhanced by irradiation, especially in Group 3; the incidence in males and females was 68% and 44%, respectively, vs 7% and 1% incidences in Group 1. Age at death with hepatocellular tumor was 612 days in Group 3 males, (vs 877 days in Group 1, 793 and 623 days in Group 2) and 626 days in Group 2 females (vs 812 days in Group 1, 927 and 756 days in Group 2). Perinatal female mice were more susceptible than 5-wk-old females (14% incidence) to the x-ray-induced tumors. The high susceptibility of perinatal mice to x-ray-induced hepatocellular tumorigenesis may be related to the high frequency of mitosis at and/or after x-ray irradiation. (5 refs)

78-3927 Effects of Fast Neutrons on Rabbits--I. Comparison of Pathologic Effects of Fractionated Neutron and Photon Exposures of the Head. (Eng) Bradley, E. W. (708 Ross Hall, 2300 Eye St., N. W., Washington, DC, 20037); Zook, B. C.; Casarett, G. W.; Bondelid, R. O.; Maier, J. G.; Rogers, C. C. *Int J Radiat Oncol Biol Phys* 2(11/12): 1133-1139; 1977.

Adult New Zealand white rabbits received one of four head irradiation doses of neutrons [mean energy 15 million electron volts; 1,680-6,720 rads] or photons (4,800-19,200 rad). Each of the photon dose levels was 2.86 rads larger than the corresponding neutron dose level. The fast neutrons were produced at the Naval Research Cyclotron in Washington, DC, by bombarding a thick beryllium target with 35-MeV deuterons. The photon irradiation was produced by a 4-megavolt linear accelerator at the George Washington Univ. Medical Center. Control rabbits received no irradiation. The neutron and photon rabbits were irradiated twice weekly for 6 wk, the fractionation schedule used for treatment of advanced malignancies by the Middle Atlantic Neutron Therapy Association at the time of initiation of this experiment. The onset and severity of skin lesions and mucosal reactions in irradiated regions appeared to be dose-dependent and more severe in photon-irradiated rabbits. Death was commonly caused by complications of mucosal reactions. Three of the five rabbits that had received the lowest neutron dose (1,680 rads), a dose commonly administered to neutron-irradiated patients, developed invasive and metastasizing osteosarcomas within the irradiated field < 1 yr after the irradiation was completed. (34 refs)

78-3928 **Second Neoplasms Following Megavoltage Radiation in a Pediatric Population.** (Eng) Haselow, R. E. (Dept. Therapeutic Radiology, Box 494, Univ. Minnesota Hosp., Minneapolis, MN, 55455); Nesbit, M.; Dehner, L. P.; Khan, F. M.; McHugh, R.; Levitt, S. H. *Cancer* 42(3): 1185-1191; 1978.

The long-term oncogenic effects of exposure of children to megavoltage treatment with cobalt-60 were reviewed based on the records of 88 patients followed for 6-22 yr. Criteria for inclusion in the study were age < 16 yr at the time of treatment, min of 10 yr survival after inital treatment or follow-up until second neoplasm (1 case), and absence of diseases with known susceptibility to cancer. Patients could have been treated additionally with orthovoltage radiation, and the primary lesion did not have to be malignant. During follow-up period (av, 14 yr) seven second neoplasms were discovered, five benign and two malignant. Five of these tumors were not related to the prior radiation. Both radiation-induced tumors(1 benign, 1 malignant) were associated with low doses (< 1,500 rads) and one was without significant morbidity. The benign tumor was a chondromatous hamartoma of the lung. The malignant tumor was an ependymoma of the fourth ventricle in a patient treated 15 yr previously with 500-600 rads to the fourth ventricle for histiocytosis. This patient had also received long-term treatment with multiagent chemotherapy. (49 refs)

78-3929 **Hyperparathyroidism after Radiotherapy.** (Eng) Erkocak, E. U. (Dept. Surgery, Cukurova Univ. Medical Faculty, Adana, Turkey); Taylor, S. *Br Med J* 2(6133): 327-328; 1978.

A 66-yr-old man who had undergone a 6-mo course of external radiation therapy for tuberculous lymph nodes at age 5 presented with hyperparathyroidism. Both parathyroid glands on the irradiated side of the neck showed hyperplasia. There was also a neuroma and a nodular goiter in this area, both of which are recognized as consequences of radiation. (5 refs)

78-3930 **Cancer in Parents of Children with Leukemia (Letter to Editor).** (Eng) Marchetto, D. J. (Sidney Farber Cancer Inst., Boston, MA, 02115); Li, F. P.; Meadows, A. T. *J Pediatr* 93(3): 537; 1978.

A report of irradiation-associated thyroid carcinomas in parents of children with acute leukemia prompted the investigation of 1,274 parents (906 mothers, 368 fathers) of such children. None of the 1,274 had thyroid carcinoma, and the incidence of other neoplasms was not thought to be excessive (0.8% vs 0.7% expected). These findings suggest that thyroid carcinoma of any cause is rare among parents of children with acute leukemia. (7 refs)

78-3931 **Radiation-induced Hypoplasia and Carcinoma of the Breast.** (Eng) Kleinfeld, G. (No affiliation given). *Breast* 4(1): 25-26; 1978.

Two cases of mammary hypoplasia caused by therapeutic irradiation in childhood are described. One patient was a 34-yr-old woman who had been treated for a hemangioma of the right breast at age 3 yr by applications of radium. The second, a 34-yr-old woman who had received external radiation at age 3 yr for bursitis of the shoulder, was also found to have intraductal carcinoma at surgery. It is concluded that carcinogenicity of the breast is related to concentrated exposure to radiation. This exposure occurs with therapeutic, not diagnostic, radiation dosages. Exposure of young women and prepubertal girls appears to be more dangerous than exposure of more mature women. There also appears to be a risk of inducing supervening breast cancer in premenopausal women treated for breast cancer by whole-breast irradiation without mastectomy. (6 refs)

78-3932 **Thorotrast Carcinoma of the Lung that Developed after a Bullet Wound and Thorotrast Application.** (Eng) Buchholz, A. (Institut fur Pathologie, Bezirkskrankenhaus/Poliklinik, Pfaffenstrass 24, DDR-20 Neubrandenburg, E. Germany). *Z Erkr Atmungsorgane* 150(1): 76-89; 1978.

A 50-yr-old man developed a bronchogenic carcinoma at the site of a scar from a bullet wound he received in 1945, 27 yr before his death. Histological examination disclosed considerable deposits of thorotrast in the carcinoma and scar tissue. It is probable that, in connection with treatment of the wound, bronchography with the use of thorotrast or preparation of a fistula were performed. All available literature cases on the development of carcinoma after a pulmonary bullet wound as well as all cases of pulmonary carcinoma development after thorotrast incorporation were collected, sorted, and tabulated. Theories concerning the etiology and pathogenesis of these tumors are presented. (62 refs)

78-3933 **Tissue Dose in Thorotrast Patients.** (Eng) Kaul, A. (Klinikum Steglitz, Freien Universitat, Berlin, W. Germany); Noffz, W. *Health Phys* 35(1): 113-121; 1978.

Absorbed α-ray doses to the liver, spleen, red marrow, lungs, and kidneys and to various parts of bone tissue were calculated for long-term burdens of iv-injected Thorotrast (TT). The calculations, performed for injection of 10, 30, 50, or 100 ml of colloidal thorium dioxide ($^{232}ThO_2$), were based upon best estimates of ^{232}Th tissue distribution and steady-state activity ratios between the subsequent daughters. Correcting for α-particle self-absorption within TT aggregates (up to 100 μm across, giving rise to substantial self-absorption), the mean α-ray dose to a standard 70-kg human at 30 yr after the injection of 25 ml TT is 750 rads to the liver, 2,100 rads to the spleen, 270 rads to the red marrow, 60-620 rads to the lungs and 13 rads to the kidneys. Dose rates to bone tissue were estimated by applying a model on alkaline earth metabolism to the continuous translocation of Th daughters to bone and to the formation of Th daughters by decay within bone tissue. The av dose to calcified bone from translocated radium-224 and its daughters is 18 rads at 30 yr after the injection of 25 ml TT. Considering a previously calculated risk coefficient of 0.9%-1.7% bone sarcomas/100 rads of av skeletal dose from ^{224}Ra and its daughters, the induction of 1.6-3.1 bone sarcomas per 1,000 TT patients is predicted. (22 refs)

78-3934 **Endosteal Dose to Thorotrast Patients.** (Eng) Mays, C. W. (Radiobiology Lab., Bldg. 522, Univ. Utah, Salt Lake City, UT, 84112). *Health Phys* 35(1): 123-125; 1978.

The endosteal dose rate was calculated for a typical iv injection of 25 ml of Thorotrast (TT) (0.55-0.65 μCi thorium-232) into a 70-kg human with 5,000 g of calcified bone, 1,500 g of red marrow, and 125 g endosteal tissue. The dose rate to the endosteum, 0-10 μg from bone surfaces, was estimated to be 16 rads/yr, 7 rads/yr from radium-224, ^{228}Th, and ^{228}Ra translocated from Thorotrast to calcified bone and 9 rad/yr from Thorotrast on bone surfaces and in red marrow. These figures were obtained by multiplying the av organ dose rate by the previously tabulated ratio of endosteal dose to av dose. Punctate sources of Thorotrast irradiate fewer bone cells but kill a higher proportion of those that are irradiated, compared with the more uniform irradiation from calcified bone. It is thus possible that the endosteal dose from Thorotrast is less effective in osteosarcoma induction than that from ^{224}Ra. The endosteal dose rate to a typical Thorotrast patient would then be equivalent to an endosteal dose rate of 7-16 rads/yr from protracted ^{224}Ra. (8 refs)

78-3935 **Lymphoid Tumours and Leukemia Induced in Mice by Bone-seeking Radionuclides.** (Eng) Loutit, J. F. (MRC Radiobiology Unit, Harwell, Didcot, Oxon, England); Carr, T. E. *Int J Radiat Biol* 33(3): 245-263; 1978.

Harwell stock CBA and C3H mice and their F_1 hybrids were inoculated ip with single carcinogenic doses of ^{90}Sr (7, 13, or 20 μCi/mouse), ^{226}Ra [50, 150, or 500 nanocuries (nCi)/mouse], or ^{239}Pu (2, 4, 10, 30, or 100 nCi/mouse) in soln and examined for the development of lymphoid tumors and leukemia. Soluble ^{90}Sr and monomeric ^{239}Pu induced generalized lymphomatosis in laboratory mice. Leukemogenesis due to soluble ^{226}Ra was more uncertain. Clinical expression was variable, but as a rule, the disease was a lymphosarcoma with hematogenous (leukemic) spread. The cell type was lymphoblastic of undifferentiated null form. The av doses of α or β radiation that accumulated in the bone marrow at the time of diagnosis were usually $>$2,500 rads, but if the cases occurring after Ra or low activities of Pu are accepted as being induced, doses of α radiation could have been as low as 300-1,500 rads. Chimeric mice rarely exhibited any lymphoma, general or local, following treatment. Abdominal lymphomas were not numerically increased by these radionuclides, although some may have been prematurely induced. (35 refs)

78-3936 **Radiation Leukaemogenesis: Is Virus Really Necessary?** (Eng) Loutit, J. F. (M.R.C. Radiobiology Unit, Harwell, Didcot, OX11 ORD, England); Ash, P. J. *Br J Cancer* 38(1): 24-33; 1978.

Generalized nonthymic lymphosarcomas from mice injected with ^{90}Sr, ^{239}Pu, or ^{226}Ra were maintained in unirradiated compatible mice and used as a source of tumor-associated antigen to determine whether repeated administration of this antigen would lead to progressive resistance to the tumor. If the tumor-associated antigens are virally determined, they should evoke a protective immunity. Subthreshold doses of living syngeneic tumor, large doses of living allogeneic tumor, and large doses of killed syngeneic tumor did not protect compatible mice from the development of lymphosarcomas. This suggests that the viruses seen electron microscopically in these tumors were not the etiological agents. The ultrastructure of the tumors and the characteristics of the viruses (mainly A-type) they contained are also presented. (24 refs)

78-3937 **Dose to Endosteal Cells and Relative Distribution Factors for Radium-224 and Plutonium-239 Compared to Radium-226.** (Eng) Marshall, J. H. (Center Human Radiobiology, Argonne Natl. Lab., Argonne, IL, 60439); Groer, P. G.; Schlenker, R. A. *Health Phys* 35(1): 91-101; 1978.

Information forming the basis for calculations of the dose to the endosteal cells from iv injections of radium-224 in adult humans is reviewed, and relative distribution factors (RDF's) for ^{224}Ra, and plutonium-239 relative to ^{226}Ra are derived. A model of alkaline earth metabolism in adult humans predicts that 10% of injected ^{224}Ra atoms decay on bone surface, 1.5% in bone volume. New measurements of the total surface/volume ratio of bone from 10 human skeletons gave a preliminary value of 50 cm^2/cm^3. RDF's were determined by calculating the ratio of the endosteal doses for equal av doses to bone for both ^{224}Ra and ^{239}Pu relative to ^{226}Ra (from a volume source). These factors predict the relative toxicity of α-emitting radionuclides in humans relative to ^{226}Ra on the assumption that the dose to endosteal cells is the causative factor. The value of RDF (^{224}Ra/^{226}Ra) was 20 and that of RDF (^{239}Pu/^{226}Ra) was 28 for a surface source of Pu and 0.96 for a vol source. The RDF's for α-emitters relative to ^{226}Ra were almost independent of distance for cell targets within 30 μm of the bone surface. Taking the fraction of injected ^{224}Ra atoms that decay in bone volume or on bone surface as 11.5%, assuming 100% retention of thoron and its daughters at bone surface, and using previously reported integrations, it was estimated that an iv injection of 1 μCi ^{224}Ra in a 70-kg standard human with 5 kg of bone results in an av dose to endosteal cells 0-10 μm from bone surfaces of 1.46 rads, slightly less than the dose of 1.77 rads calculated in another study. (25 refs)

78-3938 **Post-irradiation Sarcoma of the Clavicle: A Report of Two Patients.** (Eng) Evans, M. J. (Orthopaedic Dept., Middlesex Hosp., London, England); Hughes, S. P. *Clin Oncol* 4(2): 131-138; 1978.

A 61-yr-old woman and a 27-yr-old man developed a sarcoma of the clavicle 9 and 10 yr, respectively, after receiving radiation to the same area. Radiation was initiated following mastectomy for a spheroidal cell carcinoma in the woman and as treatment for a reticulum cell sarcoma of the neck in man. Total radiation doses were 4,740 and 5,000 rads, respectively. Four similar cases in the literature are reviewed. (14 refs.)

78-3939 **Pulmonary Connective Tissue Modifications Induced by Internal α Irradiation. I. Effect of Time and Dose on Alterations Following Inhalation of Plutonium-239 Dioxide Aerosol in Rat.** (Eng) Metivier, H. (Commissariat a l'Energie Atomique, Departement de Protection, Laboratoire de Toxicologie Experimentale, Boite Postale 561, 92542 Montrouge Cedex, France); Masse, R.; Legendre, N.; Lafuma, J. *Radiat Res* 75(2): 385-396; 1978.

Female Wistar rats were exposed by inhalation to an aerosol of relatively insoluble $^{239}PuO_2$ to quantitate the relationship between pulmonary radiation dose and dose rate and induced fibrosis. Lung wt, whole proteins, collagen, and uronic acids were determined as functions of dose and time. Total pulmonary collagen was increased up to 200% of that in control animals at 200 days after exposure at an α radiation dose to the lung of 100-10,000 rads. At 400 days, however, the total pulmonary collagen was similar to that of controls. The same evolution was observed with uronic acids. This biphasic effect was specific, since it was not observed for total proteins in the lung. Modifications of the fresh lung/dry lung ratio or significant changes in blood gases were not observed. Scars appeared and foci of the usual interstitial pneumonia were observed around trapped particles 100 days after inhalation. In animals given doses of 3,000-10,000 rads, the lesions consisted of small focal areas scattered in the lung. Most of the connective tissue deposits in these areas were due to strongly argyrophilic fibers. In the group followed 400 days after inhalation, there was no decrease of this scarring effect. Metaplasia and adenomatosis were constant at doses > 1,000 rads, and some cancers were seen. It was concluded that the decrease in connective tissue components observed with biochemical methods at later times correlates only with an evenly distributed tissue alteration; ie, the strongly argyrophilic fibers. (23 refs)

78-3940 **A Routine Procedure for the Determination of Trace Amounts of Plutonium in Urine.** (Eng) Veselsky, J. C. (International Atomic Energy Agency, Lab. Seibersdorf, A-2444 Siebersdorf, Austria). *Mikrochim Acta* 1(1/2): 79-88; 1978.

A method for determining trace amounts of plutonium in urine was developed to monitor personnel involved in nuclear energy production. The method allows large samples to be processed without time-consuming evaporation steps and no unusual or expensive reagents are required. The chemical recovery is ≥90%; the detection limit is 40 femtocuries ^{239}Pu/liter. (40 refs)

78-3941 **Relationship Between Physicochemical Form of Plutonium and Its Behavior in Tissues and in the Mouse Reticuloendothelial System. I. Distribution of Monomeric and Polymeric Pu after Sc Injection in Mice.** (Jpn) Kashima, M. (Div. Radiation Hazards, Natl. Inst. Radiological Sciences, 9-1, Anagawa 4 chome, Chiba, 280, Japan); Joshima, H.; Matsuoka, O. *Nippon Acta Radiol* 38(1): 65-80; 1978.

The distribution of plutonium was studied in a section of human skin that was accidentally punctured by a needle contaminated with $^{239}Pu(NO_3)_4$ and in mice given sc injections of monomeric or polymeric Pu. The human skin was surgically removed 2 hr after the accident and subjected to autoradiography. A total of 24 nanoCi of ^{239}Pu were detected. Although most of the alpha tracks in the malpighian layer and cornium of the skin were single tracks, several star tracks and clusters of tracks were also observed. The polymerization, translocation, and deposition of Pu were studied in mice following sc injection of fresh monomeric Pu soln at pH 1, 4, 7.2, and 13; monomeric Pu soln stored for 348 days at pH 7.2; and pure polymeric Pu. The whole-body distribution of monomeric Pu at pH 4 and 7.2 and of stocked Pu showed a similar pattern. However, there was a difference in the diffusible Pu pattern at the injection site between monomeric Pu at pH 4 or 7.2, and that at pH 1 or 13 and polymeric Pu. The former was spread throughout the sc tissue, but the latter was not. Several star tracks were observed at the site of monomeric Pu injection; this may have been the result of polymerization of monomeric Pu in the skin. Although low Pu levels were found in the blood 3 hr after injection, Pu, especially the polymeric form, was most likely to be removed from the skin by way of the lymphatics. Translocation of Pu to the bone, liver, spleen, bone marrow and lymph nodes was observed by 3 hr; distribution was uniform in the bone marrow only. In the liver, the highest concentrations were found in the zone surrounding the central vein. (32 refs)

78-3942 **Local Dose to Lung Tissue from Inhaled $^{238}PuO_2$ Particles.** (Eng) Diel, J. H. (Inhalation Toxicology Res. Inst., Lovelace Biomedical and Environmental Res. Inst., P.O. Box 5890, Albuquerque, NM, 87115). *Radiat Res* 75(2): 348-372; 1978.

A new method for computing local dose to tissue from an α-emitting particle in deep lung is described. Syrian hamsters were exposed by inhalation to 0.2-μm real diameter $^{238}PuO_2$ particles and then sacrificed 8-35 days later. The trachea was cannulated; the lungs were inflated and fixed in situ, excised, paraffin-embedded, and sectioned at 5 μm thickness; and autoradiographs were prepared. Seven particles from three animals were chosen at random from these autoradiographs, and the location of the particle and the configuration of the tissue surrounding the particle were analyzed by a computer. The local dose rate and fraction of α-particles that penetrate to or pass through tissue at a given distance from the source were computed. The local dose rate was used to compute the effect of this radiation pattern based on four empirical formulas. The av effects obtained were similar to those calculated assuming a structureless lung of uniform density (0.2 g/cm^3), but they were different from calculated effects based on uniform dose over the irradiated region. The relationship between dose rate and distance from an α-emitting particle in deep lung was virtually independent of the location of that particle. Under the dose effect assumptions used, the results indicate that there is an inverse or possibly constant relationship between the activity contained in a $^{238}PuO_2$ particle deposited in lung and its effectiveness in producing tumors. They also indicate that there is a direct relationship between cell turnover time and tumor production. (34 refs)

78-3943 **Foreign-Body Tumorigenesis in Mice: DNA Synthesis in Surface-attached Cells During Preneoplasia.** (Eng) Thomassen, M. J. (Dept. Pediatrics, Case Western Reserve Univ., Cleveland, OH, 44106); Buoen, L. C.; Brand, I.; Brand, K. G. *J Natl Cancer Inst* 61(2): 359-363; 1978.

DNA synthesis throughout the stages of foreign body (FB) tumorigenesis was investigated to provide insight into the role of macrophages in the development of neoplasia. Four unplasticized vinyl chloride-vinyl acetate copolymer films (15 x 22 mm) were implanted sc into the flanks of (CBA/H x CBA/H-T6)F_1 mice. The animals were pulsed with ^{3}H-thymidine at various times during the next 15 mo. DNA synthesis occurred consistently in the film-attached cells, predominantly macrophages, throughout the preneoplastic phase in both male and female mice. The labeling rate was calculated to be 5-15 cells/1,000, the cell turnover rate to be 1.5%-4.5%/day, and the mitotic rate to be <3 cells/10,000. Giant cells with <10 nuclei were labeled synchronously and asynchronously. No DNA synthesis was detected in giant cells with >10 nuclei. The results show that the macrophages were not dormant with respect to DNA synthesis. (16 refs)

See also:
*(Rev.): 78-3609, 78-3612, 78-3626, 78-3648, 78-3649, 78-3655, 78-3656, 78-3657, 78-3658, 78-3659, 78-3660, 78-3661, 78-3662.
*(Chem.): 78-3689, 78-3703, 78-3716, 78-3724, 78-3811, 78-3834, 78-3846, 78-3848, 78-3849, 78-3854.
*(Viral): 78-3962, 78-3973.
*(Path): 78-4103, 78-4106, 78-4135.
*(Epid.-Biom.): 78-4149, 78-4162, 78-4169, 78-4182, 78-4183.

78-3944 **Unwinding-like Activity Associated with Avian Retrovirus RNA-directed DNA Polymerase.** (Eng) Collett, M. S. (Dept. Pathology, Univ. Colorado, Medical Sch., Denver, CO, 80262); Leis, J. P.; Smith, M. S.; Faras, A. J. *J Virol* 26(2): 498-509; 1978.

An activity associated with purified preparations of avian retrovirus reverse transcriptase that is capable of dissociating hydrogen-bonded regions from duplex nucleic acid molecules is described. This "unwindinglike" activity appears specific, affecting RNA-DNA and DNA-DNA duplex molecules but not RNA-RNA duplexes. It can remove a portion of the hydrogen bonds from double-stranded structures, but it cannot separate complementary strands completely. The unwindinglike activity was sensitive to temperature and monovalent and divalent cation concentrations. It could also remove a specific large oligonucleotide from the 5′ end of the viral genome subsequent to RNase H hydrolysis of viral RNA complexed to DNA present at that terminus. It is speculated that this activity may be involved in the removal of RNA oligoribonucleotides from RNA-DNA hybrid intermediates by the viral RNase H activity during proviral DNA synthesis. (27 refs)

78-3945 **Neoplasms Resembling Lymphoid Leukosis in Chicken Flocks Free of Infection with Exogenous Lymphoid Leukosis Virus.** (Eng) Crittenden, L. B. (Regional Poultry Res. Lab., U.S.D.A., ARS, East Lansing, MI); Neiman, P. E.; Okazaki, W.; Sharma, J. M.; Witter, R. L. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York Elsevier/North Holland Biomedical Press): 503 pp.; 456-457; 1978.

An attempt was made to determine the etiologic agent involved in 12 cases of apparent lymphoid leukosis (LL) that occurred spontaneously in an inbred line of chickens that consistently produces the endogenous Rous-associated virus RAV-O. The LL occurred in sublines of line 7 or in F_1 hybrids of line 7. In 8/12 birds, the bursa of fabricius was grossly neoplastic. Tests excluded LL virus, Marek's disease virus, and reticuloendotheliosis virus as etiologic agents. DNA from these tumors hybridized with RNA extracted from RAV-O, but the reaction kinetics were not different from those observed with normal chicken cell DNA. A subgroup E leukosis virus isolated from infected chickens was injected into recipient birds, but no lymphoid tumors were induced by >300 days. Analysis of one of two transplantable tumor lines indicated that this transplant was of T- cell origin and thus atypical, since LL is thought to be a B-cell-dependent neoplasm. An etiologic role for RAV-O was not proved. (3 refs)

78-3946 **Suppression of the Mitogen-stimulated Blastogenic Response During Reticuloendotheliosis Virus-induced Tumorigenesis: Investigations into the Mechanism of Action of the Suppressor.** (Eng) Carpenter, C. R. (Dept. Microbiology, Univ. Texas at Austin, Austin, TX, 78712); Rubin, A. S.; Bose, H. R. *J Immunol* 120(4): 1313-1320; 1978.

The in vitro and in vivo kinetics of reticuloendotheliosis virus (REV)-induced suppression of the phytohemagglutinin (PHA) response of splenic lymphocytes were investigated. Using PHA-induced ^{3}H-thymidine uptake as an indication of the ability of spleen cells from REV-infected chickens to undergo blastogenesis, severe suppression of the response was demonstrated within 72 hr of REV infection. This suppression required active viral infection, since ip or iv injection of UV-inactivated REV did not result in inhibition of the blastogenic response. Suppressor cells from the spleens of REV-infected birds severely inhibited the ability of spleen cells from uninfected chickens to respond to PHA at a ratio of 1:20, suggesting that each suppressor cell may be capable of suppressing more than one target cell. Contact between suppressor and target cells was required for the rapid inhibition of the normal PHA response. The suppressive mechanism is cytostatic and apparently of host origin, since neither REV nor REV-infected (or transformed) cells mediated the suppression. The ability of the suppressor cells to impair the blastogenic response of spleen cells was sensitive to trypsin, suggesting that an inhibitory protein is exposed on the surface of the suppressor cells. In view of previous indications that early cytostatic suppressive events might inhibit cytotoxic immune reactions, the early appearance of this suppressor activity should favor tumorigenesis. (35 refs)

78-3947 **Morphological and Biochemical Differentiation in RSV Transformed Chick Embryo Myoblasts.** (Eng) Fiszman, M. Y. (Dept. Biologie Moleculaire, Institut Pasteur, 75015 Paris, France). *Cell Differ* 7(1/2): 89-101; 1978.

Morphological and biochemical differentiation was studied in chick embryo myoblasts transformed by a temperature-sensitive mutant of Rous sarcoma virus (RSV ts88). At the permissive temperature (36 C), the transformed myoblasts lost their ability to form myotubes and to express the biochemical markers of myogenic differentiation. No acetylcholine receptors were detected, few myosin heavy chains and no myosin light chains were synthesized, and only the B form of creatine phosphokinase was present in the cells. After a shift to the nonpermissive temperature (41 C), the normal differentiation process was restored; myotubes were formed that expressed muscle-specific proteins. Two mechanisms by which viral transformation may prevent myogenic differentiation are summarized. (20 refs)

78-3948 **Avian Retrovirus RNA-directed DNA Synthesis: Transcription at the 5′ Terminus of the Viral Genome and the Functional Role for the Viral Terminal Redundancy.** (Eng) Collett, M. S. (Dept. Pathology, Univ. Colorado Medical Sch., Denver, CO, 80262); Faras, A. J. *Virology* 86(2): 297-311; 1978.

The size and structure of the DNA synthesized in vitro on 35S RNA-transfer RNA-trp (tRNA-trp) template-primer complexes in reconstructed reactions by avian retrovirus RNA-directed DNA polymerase were elucidated. Under standard enzymatic reaction conditions, three major DNA species were initiated on tRNA-trp at the 5′ end of the viral genome. Two of these were single-stranded transcripts 70 and 100 nucleotides in length. The third was hairpin in structure and 185 nucleotides in length. A precursor-product relationship was being demonstrated between DNA_{70}, DNA_{100}, and the hairpin DNA_{185} species. Furthermore, the hairpin DNA_{185} appeared to form during DNA synthesis, rather than after DNA synthesis, as a consequence of the intramolecular folding of complementary sequences present in the DNA_{185} transcript. In addition to conversion of DNA_{100} to hairpin DNA, the DNA_{100} species could attach to the 3′ end of the viral genome, presumably by base pairing with the terminally redundant sequences at the 3′ terminus, to provide a primer for the continuation of DNA transcription at this end of the viral RNA. These data provide a functional role in proviral DNA synthesis for the terminally repeated genomic sequences. (46 refs)

78-3949 **Persistence, Induction and Transfection of Virus Genome in RSV-transformed Mammalian Cells.** (Eng) Svoboda, J. (Inst. Molecular Genetics, Czechoslovak Acad. Sciences, Prague, Czechoslovakia). *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 30-37; 1978.

The role of the avian sarcoma virus (ASV) *gag* gene in viral transfection was studied using Brown Leghorn chicken fibroblasts transfected by DNA isolated from duck cells that had been transformed by the ASV mutant LA 334. Following transfection, four viral isolates were obtained that were temperature-sensitive (ts) to transformation. The tests of ts lesion formation at the level of virus synthesis using chicken fibroblasts gave equivocal results. However, when the viral isolates were tested on duck cells, virus production was markedly increased at the nonpermissive temperature (41 C). These and other results suggest that the region of the virus genome responsible for coding for the *gag* gene products is specifically transferred during transfection. The importance of these findings to the molecular characteristics of provirus expression is outlined. (38 refs)

78-3950 **Preliminary Identification of the Number of *Sarc* Gene Protein Products of Rous Sarcoma Virus.** (Eng) Kamine, J. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA); Burr, J. G.; Buchanan, J. M. *Adv Enzyme Regul* 16: 379-390; 1978.

The protein products made from virion RNA of transformation-defective (*td*) and nondefective (*nd*) Rous sarcoma virus (RSV) in a cell-free translation system were compared. Previously, two proteins of 25,000 and 18,000 daltons had been isolated by sodium dodecyl sulfate-polyacrylamide gelelectrophoresis from *nd* RSV but not from *td* RSV, which lacks the *sarc* gene. In these experiments, separation of RSV virion RNA on sucrose gradients showed that these two putative *sarc* gene products are synthesized as doublets from a messenger RNA (mRNA) of approx 18S. In addition to these two proteins, the 18S viral mRNA from *nd* RSV also directed the synthesis of a 60,000-dalton protein; this protein was absent or produced in much smaller amounts from the RNA of *td* RSV, suggesting that it may also be a *sarc*-specific protein. Three additional sizes of viral mRNA were found in *nd* and *ts* virions: the 38S genomic RNA that yields the 80,000-dalton *gag* gene precursor, a 28S RNA that yields a 55,000-dalton protein, and an RNA slightly smaller than 18S that yields a 37,000-dalton protein. Two-dimensional electrophoresis allowed the resolution of both bands of the 25,000 dalton doublet into several differently charged species. (34 refs)

78-3951 **Glycopeptides Derived from Individual Membrane Glycoproteins from Control and Rous Sarcoma Virus-transformed Hamster Fibroblasts.** (Eng) Tuszynski, G. P. (Wistar Inst. Anatomy and Biology, Philadelphia, PA, 19104); Baker, S. R.; Fuhrer, J. P.; Buck, C. A.; Warren, L. *J Biol Chem* 253(17): 6092-6099; 1978.

Membrane glycoproteins (gp's) from control (BHK_{21}/C_{13}) and Rous sarcoma virus-transformed (C_{13}/B_4) baby hamster kidney cells grown in medium containing (^{14}C- or D-3H-glucosamine were separated into two distinct classes by lithium diiodosalicylate (LiISal)-phenol extraction: a phenol-soluble fraction and an aqueous fraction. The phenol- and aqueous-soluble gp's differed in their sodium dodecyl sulfate-polyacrylamide gel profiles, polyacrylamide isoelectric focusing profiles, and glycopeptide distribution of Sephadex G-50. A number of aqueous- and phenol-soluble gp's from BHK_{21}/C_{13} and C_{13}/B_4 cells were purified to near homogeneity by polyacrylamide gel electrophoresis and gel electrofocusing. These gp's ranged in mol wt from 179,000 to 31,000 and had isoelectric points of 7.5-3.0. A comparison of 24 gp's from control and transformed cells showed that the pronase glycopeptides of 20 gp's from transformed cells gave rise to different Sephadex G-50 profiles than those from the homologous gp's from control cells. The exact chemical nature of these differences is not known, but they probably result from differences in sugar composition and/or oligosaccharide structure, which determine the size and shape of the glycopeptide. Such extensive alterations of membrane components could be the basis for the changes in cell behavior and growth properties that are known to accompany malignant transformation. (27 refs)

78-3952 **Immunogenicity of the Envelope Glycoprotein of Avian Sarcoma Virus.** (Eng) Halpern, M. S. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA, 19104); Friis, R. R. *Proc Natl Acad Sci USA* 75(4): 1962-1966; 1978.

The humoral response to the type- and group-specific determinants of viral envelope glycoprotein (VGP) was measured after sensitization of chick helper factor-positive [chf(+)] and chf(-) chickens to the VGP of exogenous avian sarcoma virus (ASV). The expression of type E-specific glycoprotein (gpE) in chf(+) but not chf(-) chickens is not restricted to a certain stage of embryogenesis, but persists in postembryonic chickens during the development and maintenance of immunocompetence. Sensitization of chf(+) and chf(-) chickens resulted in the induction of detectable antibody reactivity to determinants of the VGP that were type-specific as well as group-specific. Because group-specific determinants were present on gpE, these results link immunity to exogenous (VGP) in chf(+) chickens with autoreactivity. It is hypothesized that the VGP is a thymus-dependent antigen and that helper T cells in chf(+) chickens are functionally unreactive to the group-specific determinants of the VGP because the endogenous gpE in chf(+) chickens renders specific T cells, (but not B cells) unresponsive. With chf(+) chickens, triggering of B cells reactive to group-specific determinants would occur by a carrier effect in which T cells are primed to foreign determinants associated with exogenous VGP, such as the type-specific determinants. The available amount of T cell help may determine the strength of the response to group-specific determinants. (12 refs)

78-3953 **Tumor-specific and Tumor-associated Membrane Antigens of Rous Sarcoma Virus Transformed Hamster Fibroblasts.** (Eng) Comoglio, P. M. (Dept. Human Anatomy, Sch. Medicine, Univ. Turin, Turin, Italy); Bertini, M.; Prat, M. *Int J Cancer* 22(1): 55-62; 1978.

The antigenic composition of hamster fibroblasts transformed in vitro by Rous sarcoma virus (RSV) mutants either defective for the synthesis of gp85 (*env-*) or temperature-sensitive for the ability to transform the host cell (*ts*) is reported. Xenogeneic antisera obtained by immunizing rabbits with the RSV-transformed fibroblasts recognized, in the ^{51}Cr-release cytotoxicity assay, at least two tumor-membrane antigens: (1) an antigen also common to hamster cells transformed by unrelated DNA and RNA oncogenic viruses (polyoma virus, mouse sarcoma virus, simian virus 40); and (2) a virus-induced cell-surface antigen (VCSA) that is virus-specific and is also expressed by rat, quail, and chicken cells transformed by RSV. By comparing the anti-VCSA serum with antisera directed against purified gp85, group-specific proteins, reverse transcriptase, or detergent-lysed virus particles, it was shown that VCSA is not a known virion structural protein. VCSA expression did not correlate with viral replication, since it was not detected in chick embryo fibroblasts productively infected with the transformation-defective Rous-associated virus 1, which shares virus structural genes with RSV. However, in hamster cells transformed by the RSV *ts* mutant, VCSA expression at the cell surface correlated with the expression of the transforming gene. It is concluded that VCSA is under the control of the *sarc* gene and that in this respect, it seems to be the mammalian counterpart of the avian tumor-specific surface antigen. (38 refs)

78-3954 **Virus-like Particles Associated with the Mitochondria of Ethidium Bromide Treated Transformed Cells.** (Eng) Soslau, G. (Dept. Biological Chemistry, Hahnemann Medical Coll. and Hosp., Philadelphia, PA, 19102). *J Gen Virol* 40(part 1): 245-251; 1978.

Electron microscopy and enzyme studies demonstrated the production of mitochondria (M)-associated viruslike particles in baby hamster kidney cells transformed by the Bryan strain of Rous sarcoma virus (RSV) when they were treated with ethidium bromide (EB). These cells, line C_{13}/B_4, are normally nonproducers. However, when C_{13}/B_4 cells were grown in the presence of 2 μg/ml EB for 4 days, electron microscopy showed that their matrix was less dense than that of untreated cells and that viruslike particles were associated with the M. These particles were never observed in the M of untreated cells. M-associated virus particles have also been observed in an EB-resistant cell line ($B_4EB\text{-}R_2$, developed from C_{13}/B_4 cells) that has been cultured and cloned in the presence of 2 μg/ml EB for > 2 yr. The number of viruslike particles was enhanced when the $B_4EB\text{-}R_2$ cells were treated with 8 μg/ml EB for 4 days. Enzyme studies assaying for an RNA-dependent DNA polymerase [reverse transcriptase (RT)]-type activity were performed to support the electron microscope findings. Both C_{13}/B_4 and $B_4EB\text{-}R_2$ cells were positive for the presence of an RT-like enzyme associated with the M. The enzyme activity was not related to a terminal transferase, it had the characteristics of the virus RT with respect to template preference, and it was found at the buoyant density of the presumptive virus before and after treatment with Triton X-100. These results confirmed that a virus-like particle is induced by EB in both cell lines. It is not clear whether the particles are derived from integrated RSV information or they represent an endogenous species. (31 refs)

78-3955 **Simultaneous Production of Mouse Endogenous Virus and Rous Sarcoma Virus by Schmidt-Ruppin Virus Infected Mouse Cells.** (Eng) Kotler, M. (Lab. Molecular Virology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Lerer, I.; Ben Moyal, Z.; Spira, G. *J Gen Virol* 40(part 1): 141-150; 1978.

This study shows that injection of Schmidt-Ruppin Rous sarcoma virus subgroup D (SRV-D)-infected chick cells into C3H/f newborn mice gives rise to mouse tumor cells capable of expressing antigens of both avian sarcoma and mouse leukemia viruses and of producing C-type particles. Sc injections of 1 x 10^6 chick embryo fibroblasts transformed by SRV-D into the mice gave rise to tumors at the injection site after approx 60 days in about 40% of the mice. These tumors were transplantable in C3H/f mice and, after 18 passages, were able to induce tumors in the wing web of adult Leghorn chickens. Tumor cells from the 18th passage were also used to establish a cell line (C3HSR) in tissue culture. These cells released C-type virus particles that produced foci and were able to propagate in chick cells. The C3HSR cells were tested in complement fixation studies performed with antisera against Moloney murine leukemia virus (MuLV) p30 antigen, feline leukemia virus, and SRV-D. C3HSR cell extracts gave positive results with all three antisera, indicating that these cells express both avian and mammalian virus antigens. Examination of three subclones of C3HSR cells by immunofluorescence showed that both avian and mammalian antigens are produced by the same cell. Mouse cells infected with virus released by C3HSR cells produced MuLV-like particles, as revealed by the reverse XC syncytial test and by immunofluorescence tests. (24 refs)

78-3956 **Brain Tumors Induced in Hamsters by Intracerebral Inoculation of SR-RSV-Infected Embryonic Brain Cells.** (Eng) Nakazato, Y. (First Dept. Pathology, Gunma Univ. Sch. Medicine, 3-39-22 Showa-machi, 371 Maebashi-shi, Gunma-ken, Japan); Ishida, Y. *Acta Pathol Jpn* 28(3): 445-457; 1978.

Brain tumors were induced in Syrian hamsters by the intracerebral inoculation of brain cells obtained from 12-day-old syngeneic hamsters and infected in vitro with the Schmidt-Ruppin strain of Rous sarcoma virus (SR-RSV). A total of 212 tumors developed in all 25 recipients 19-125 days after transplantation. On the basis of light and electron microscopy, they were classified into four main groups: astrocytoma (45.8%), pleomorphic glioma (50.0%), sarcoma (3.8%), and unclassified (0.4%). The morphological features of these tumors are described, and the advantages of this brain tumor model are discussed. (22 refs)

78-3957 **Polyethylene Glycol-mediated Infection with Avian Sarcoma Viruses. Brief Report.** (Eng) Rohde, W. (Institut fur Virologie, Fachbereich Humanmedizin, Justus Liebig-Universitat Giessen, Frankfurter Strasse 107, D-6300 Giessen, W. Germany); Pauli, G.; Henning, J.; Friis, R. R. *Arch Virol* 58(1): 55-59; 1978.

The use of polyethylene glycol-induced fusion of viral and cellular membranes to overcome genetically determined resistance to infection is reported. Using this method, avian sarcoma virus-transformed mammalian cells were produced; these cells will be useful for the study of Rous sarcoma virus-specific tumor antigens. (11 refs)

78-3958 **Different Types of Virus-specific RNA in Mammalian Cells Transformed by Rous Sarcoma Virus as Revealed by Molecular Hybridization Tests.** (Eng) Kisseljov, F. L. (Cancer Res. Center, Moscow, USSR); Tatosyan, A. G.; Svoboda, J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 271-273; 1978.

Rous sarcoma virus-specific RNA was analyzed in virogenic XC rat cells and nonvirogenic RVP-3 and RVA-4 mouse cells. No virus-specific RNA was found in the cytoplasm of RVA-4 cells. RVP-3 and XC cells differed from each other with respect to the presence of viral group-specific antigen. These cells contained virus-specific RNA's that were transcribed from different parts of the integrated viral genome. (26 refs)

78-3959 **Restriction Enzyme Sites on the Avian RNA Tumor Virus Genome.** (Eng) Taylor, J. M. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA, 19111); Hsu, T. W.; Lai, M. M. *J Virol* 26(2): 479-484; 1978.

Full-size single-stranded DNA transcripts of the avian RNA tumor virus genome were isolated from the products of the endogenous reaction of detergent-disrupted avian sarcoma virus (ASV) particles. These transcripts were converted with *Escherichia coli* DNA polymerase I and ^{32}P-labeled nucleoside triphosphate precursors, in the presence of oligodeoxyribonucleotide primers, into labeled double-stranded DNA. The latter DNA was used to map the sites of action of seven restriction enzymes (*Pvu* I, *Hpa* I, *Kpn* I, *Xba* I, *Eco*RI, *Hin* dIII, and *Xho*I) on ghe genome of three strains of ASV (Prague B, Prague C, and Bratislava 77). The site on the genome where transcription of complementary DNA ultimately stops has not been determined; there may be different results for in vitro and in vivo transcription. The immediate application of the restriction data obtained from this study is to characterize those ASV DNA species, both integrated and unintegrated, that can be detected in infected avian cells. (19 refs)

78-3960 **Kinetics of Synthesis, Structure and Purification of Avian Sarcoma Virus-specific DNA Made in the Cytoplasm of Acutely Infected Cells.** (Eng) Varmus, H. E. (Dept. Microbiology, Univ. California, San Francisco, CA, 94143); Heasley, S.; Kung, H. J.; Oppermann, H.; Smith, V. C.; Bishop, J. M.; Shank, P. R. *J Mol Biol* 120(1): 55-82; 1978.

A virus-specific DNA synthesized in the cytoplasm of quail tumor cells acutely infected with the Bratislava-77 strain of avian sarcoma virus was characterized by rate-zonal sedimentation and polyacrylamide gel electrophoresis in conjunction with molecular hybridization reagents specific for various regions of the viral genome. The rate of appearance and elongation of the viral strands was sufficiently slow and synchronous to permit a description of the order of principal events in the synthesis of the cytoplasmic linear duplex. The description is completely compatible with the events described for reactions in vitro. Moreover, a homogeneous species of short strands (approx 250-300 nucleotides) with the same polarity as the viral genome was identified that contains sequences derived from both the 3′ and 5′ termini of the viral genome. In addition, the (-) strand of viral DNA, which is complementary to the viral genome, was purified from the cytoplasm of infected cells, and radiolabeled (+) strands were synthesized in vitro with *Escherichia coli* DNA polymerase I, using extremely small quantities (approx 10 nanograms) of purified (-) strands as template. (97 refs)

78-3961 **Transmission of B77 Virus Double Mutant LA334 Markers by Transfection.** (Eng) Stetina, R. (Inst. Experimental Medicine, Czechoslovak Acad. Sciences, 515 83 Olesnice v Orlickych horach, Praha 6, Czechoslovakia); Mach, O.; Svoboda, J. *Intervirology* 10(5): 276-288; 1978.

The successful transfection of chicken fibroblasts with DNA isolated from LA334-transformed duck cells and determination of temperature-sensitive (*ts*) lesions in the *src* and *gag* genes in the progeny viruses are reported. LA334 is the double mutant of avian sarcoma virus B77: it has two independent *ts* lesions, in virus transformation and replication, that implicate mutations in the respective *src* and *gag* genes. The exposure of chick fibroblasts from Brown Leghorn embryos to 1.5 and 3.0 μg DNA/culture transfected 1/6 and 3/9 cultures, respectively. Thus, lowering the DNA concentration decreased the efficiency of transfection. Similar attempts to transfect duck embryo fibroblasts were not successful. The four viruses recovered after transfection were all ts for transformation. Two were fully ts for replication, as shown by analysis of virus replication, by characterization of virus particles produced at the nonpermissive temperature (41 C) using density gradient centrifugation, and by electron microscopy. The other two viruses were only partially ts for replication. It is suggested that both the *src* and *gag* regions of the avian sarcoma virus genome are transferred simultaneously during transfection, probably by a single integral provirus copy. (37 refs)

78-3962 **Inactivation of Turkey Herpesvirus by UV Light.** (Eng) D'Aprile, P. (Instituto Zooprofilattico Sperimentale delle Venezie, Via G. Orus, 2, 35100 Padua, Italy); Petek, M. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 247-248; 1978.

The UV inactivation rate of turkey herpesvirus (HVT) was compared with that of Pseudorabies virus (PSRV). The UV inactivation curves of HVT were one-component but those of PSRV were two-component, and the former was more resistant to UV light than the latter. The high resistance of HVT was partially due to host cell reactivation. Caffeine treatment of host cells reduced this reactivation. A multiple reactivation effect was noted for PSRV but not for HVT. (2 refs)

78-3963 **Characterization of a Nonproductive Cell Line, MKT-1, Established from a Marek's Disease Tumor of Kidney in a Chicken.** (Eng) Nonoyama, M. (Lab. Molecular Virology, Life Sciences, Inc., St. Petersburg, FL, 33710); Tanaka, A.; Silver, S.; Mitchen, J. R.; Smith, M.; Plasencia, R. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 193-194; 1978.

The status of Marek's disease virus (MDV) genomes in MKT chicken lymphoblast cells was determined. Most MDV DNA existed as nonintegrated plasmid DNA with the characteristics of a closed, circular molecule. Since similar observations were made in herpesvirus saimiri-transformed cells, this form of DNA must be common in the genomes of tumor-inducing herpesviruses. Transcription studies in MKT cells indicated that approx 20% of the viral genome is transcribed. A similar amount of Epstein-Barr virus DNA is transcribed in Raji cells. (6 refs)

78-3964 **Relationship of Intracisternal A-Particles from Mouse Myelomas to C-Type Virus.** (Eng) Weimann, B. J. (Basel Inst. Immunology, CH-4005 Basel 5, Switzerland); Schmidt, J.; Pragnell, I. B.; Takacs, B. J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.;

Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 240-241; 1978.

Intracisternal A particles were isolated from mouse myeloma cells, and the DNA polymerase that eluted at 0.30 M KCl was compared with that of the C-type Abelson murine leukemia virus (A-MuLV). Both enzymes had a mol wt of 80,000 daltons, had almost identical slopes of inhibition, responded to the same polynucleotides, and carried ribonuclease H. Purified RNA from the A particles formed stable hybrids with a DNA probe synthesized in vitro from A-MuLV. (2 refs)

78-3965 **The Use of Nucleic Acid Hybridization to Demonstrate Relatedness Between the Naturally Occurring and Laboratory Strains of Murine C-type Viruses.** (Eng) Chattopadhyay, S. K. (Pediatric Oncology Branch, NCI, Bethesda, MD, 20014); Lander, M. R.; Kramer, B. S.; Hartley, J. W.; Levine, A. S.; Rowe, W. P. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIth International Symposium on Comparative Research on Leukemia and Related Disease, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 448-451; 1978.

^{3}H-DNA probes were used in hybridization reactions to confirm the finding that naturally occurring murine leukemia viruses (MuLV) are either ecotropic, xenotropic, or amphotropic. The entire genomes of the xenotropic viruses NZB-IU-1 and BALB-IU-1 and the amphotropic strain 1504 were present in the DNA of all inbred strains studied. Each of these three classes is biochemically distinct; ie, 20% of their genomes are comprised of host-derived sequences. Half the unique sequences of certain naturally occurring MuLV may be present in the genomes of laboratory viral strains. (13 refs)

78-3966 **The Early Appearance and Subsequent Distribution of Murine Leukaemia Virus in NZB Embryos.** (Eng) East, J. (Imperial Cancer Res. Fund, Mill Hill, London, England); Tilly, R. J.; Tuffrey, M.; Harvey, J. J. *Int J Cancer* 22(4): 495-502; 1978.

The location and distribution of murine leukemia virus (MuLV) particles were examined by electron microscopy in New Zealand Black (NZB) mouse embryos from before implantation (at 4.5 days after mating) until the 14th day of embryonic life. AKR, BALB/c, and (BALB/c x NZB)F_1 eggs were included for comparison of preimplantation phases, and five combinations of blastocysts fused in vitro as six- to eight-cell eggs were also examined. Four variants of A-type particles were distinguished, and they were associated with cisternae of the endoplasmic reticulum. All four replicated at some stage of preimplantation development in NZB and AKR eggs as well as in fused AKR <--> AKR and BALB/c <--> BALB/c blastocysts, and three were found in 8- or 14-day NZB embryos. The smallest A-type variant also appeared in one-cell unfertilitized NZB eggs, indicating that its production did not depend on fertilization. Immature C-type virus was first seen in 5-day NZB implants. The spleen of 12-day NZB embryos was the first discrete organ in which virus could be visualized, and it contained both immature C-type particles and one of the A-type variants. No mature C-type virus was detected. Coincidentally, huge numbers of immature and mature C-type MuLV were found distending the uterine glands of pregnant NZB females carrying 5- or 7-day implants. This unrestrained virus replication was not related to implantation, because the same pattern was seen in the uterine glands of 7/8 age-matched virgin NZB females. The lack of infectivity of A-type particles coupled with their genetic transmission and their ubiquity in NZB embryos may indicate that they are xenotropic. (32 refs)

78-3967 **Genetic Relationship of Wild Mouse Amphotropic Virus to Murine Ecotropic and Xenotropic Viruses.** (Eng) Bryant, M. L. (Dept. Pathology, Univ. Southern California Sch. Medicine, Los Angeles, CA, 90033); Roy-Burman, P.; Gardner, M. B.; Pal, B. K. *Virology* 88(2): 389-393; 1978.

The general nucleic acid homology between amphotropic and ecotropic wild mouse (*Mus musculus*) leukemia viruses (MuLV) and between these viruses and the ecotropic and xenotropic MuLV's of inbred mice was determined. The degree of homology was ascertained by DNA-RNA hybridization using viral complementary DNA and polyadenosine-containing 70S viral RNA. The results indicated that the genomes of individual cloned amphotropic and ecotropic wild mouse virus isolates were closely related. Furthermore, these nucleotide sequences were closely related to, but distinguishable from, the genomes of prototype ecotropic (Rauscher, Gross-AKR) and xenotropic (AT-124, NZB, AKR, BALB/c) MuLV's of inbred mice. (24 refs)

78-3968 **Murine Leukemia Virus in Chemically-induced Sarcomas as Detected by XC Plaque Formation.** (Eng) Basombrio, M. A. (Academia Nacional de Medicina, Las Heras 3092, 1425 Buenos Aires, Argentina); Pasqualini, C. D. *Arch Geschwulstforsch* 48(4): 308-312; 1978.

Endogenous murine leukemia virus (MuLV) was detected in short-term in vitro cultures of primary autochthonous sarcomas induced by sc implantation of 320 μg of 3-methylcholanthrene (3-MC) in CBF_1 mice. In direct XC syncytium assays, 13/31 cultures induced >4 syncytium plaques per flask. In seven tumors, the plaques numbered >200 or were confluent. The titers found in individual tumors were definitely higher than those that could be expected solely from endogenous, naturally occurring oncornaviruses present in animals of the same age and sex. All 39 normal spleen cultures had <4 plaques; 31 cultures had no plaques at all. It is postulated that the increased titer of virus was a result rather than a cause of tumor induction by chemicals. The association of MuLV with 3-MC-induced tumors may be due to in vitro conditions, which may favor endogenous MuLV replication in sarcoma cells. (14 refs)

78-3969 **Efforts to Control Type C Virus Expression in Wild Mice.** (Eng) Gardner, M. B. (Dept. Pathology, Univ. Southern California Sch. Medicine, Los Angeles, CA, 90033); Klement, V.; Henderson, B. E.; Estes, J. D.; Dougherty, M.; Casagrande, J.; Huebner, R. J. *Prog Cancer Res Ther* 2: 391-407; 1977.

The possibility of genetic control of the indigenous murine leukemia virus (MuLV) in Lc wild mice was studied. LC mice were crossbred with the inbred laboratory mouse strain B10. In contrast to the LC parents, there was an almost complete absence of detectable spleen CF group-specific (gs) antigen in the reciprocal B10 x LC F_1 hybrids and in the reciprocal F_1 and F_2 B10 backcrosses. It was more prevalent in the F_2 generation and in the F_1 backcross to LC mice. Suppression of gs antigen expression was correlated with a suppression of infectious MuLV production, and in the F_1 hybrids, lymphoma was significantly reduced in incidence and delayed in onset, and paralysis was essentially eliminated. Of the five lymphomas in F_1 hybrids, only two appeared similar to the LC lymphomas in their pathologic and virologic features. A breast carcinoma, a lung adenoma, and an adrenal adenoma also occurred in three respective F_1 hybrids with LC mothers. Attempts to actively immunize LC mice after the neonatal period with their indigenous MuLV and to prevent lymphoma and paralysis were not successful, apparently because of relative tolerance to this virus. The Fv-1 allele from the B10 mice and an additional gene(s) appeared to be responsible for the genetic control of MuLV expression and the related diseases in the B10 x LC hybrids. (35 refs)

78-3970 **Relationship of Endogenous Murine Xenotropic Type C Virus Production to Spontaneous Transformation of Cultured Cells.** (Eng) Avery, R. J. (Dept. Biological Sciences, Univ. Warwick, Coventry CV4 7AL, England); Levy, J. A. *J Gen Virol* 39(3): 427-435; 1978.

An investigation was made of (1) the regulation of spontaneous production of endogenous xenotropic viruses by clones of New Zealand Black (NZB) mouse embryo cells and (2) the interaction between endogenous virus production and the ability of clones to transform spontaneously. All clones derived from the established NZB-Q cell line produced xenotropic virus but at different titers; the level of production varied between 10^2 and 10^4 infectious particles/ml. Virus production was constant over several passages. A series of subclones derived from each clone produced titers of virus identical to that of the parental clone. These data indicate that the NZB-Q line was composed of cells with varying degrees of virus expression that could be separated by cloning. Foci of cell alteration occurred spontaneously in some of the clonal lines after 6 to 10 passages. The amount of virus generated did not correlate with the spontaneous transformation of NZB, NIH Swiss, or BALB/c mouse cells. It is concluded that individual mouse cells differ in their intracellular regulation of xenotropic virus expression but that this control, once established, remains constant. The findings also indicate that spontaneous cellular transformation does not alter the intracellular control of virus production. (21 refs)

78-3971 **Oncogenicity of Friend-Virus-infected Cells: Determination of Origin of Spleen Colonies by the H-2 Antigens as Genetic Markers.** (Eng) Wendling, F. (Unite de Physiologie Cellulaire de l'INSERM--U. 22, Fondation Curie-Institut du Radium, Faculte des Sciences, Batiment 110, 91405 Orsay, France); Tambourin, P. E. *Int J Cancer* 22(4): 479-486; 1978.

The origin of spleen colonies in semiresistant hybrid mice inoculated with Friend leukemia virus (FLV)-infected cells was determined with the use of histocompatibility (H-2) antigens as genetic markers. The standard cytotoxicity test was used to characterize the H-2 antigens present on the surface of spleen cells from normal or FLV-infected leukemic parents or F_1 hybrid mice. Colonies obtained from the spleens of (C3H x C57BL/6)F_1 recipients (H-2k/H-2b) 10 days after iv graft of FLV-infected spleen cells of C3H origin (H-2k) were sensitive to anti-C57BL/6 antibodies. Colonies obtained from the spleens of (DBA/2 x C57BL/10)F_1 recipients (H-2d/H-2b) grafted with DBA/2 leukemic spleen cells (H-2d) were sensitive to both anti-H-2b and anti-H-2d antibodies. These results prove that the main cell population constituting a spleen colony originates from the recipient. It is concluded that spleen colonies do not result from the neoplastic proliferation of injected donor cells but rather from the multiplication of host cells transformed by Friend virus produced by the grafted cells. (37 refs)

78-3972 **Origin of Spleen Colonies Generated by Friend Virus-infected Cells in Mice.** (Eng) Steeves, R. A. (Dept. Genetics, Albert Einstein Coll. Medicine, Yeshiva Univ., 1300 Morris Park Ave., Bronx, NY, 10461); Bubbers, J. E.; Plata, F.; Lilly, F. *Cancer Res* 38(9): 2729-2733; 1978.

"Tumor colonies" develop in the spleen of unirradiated C57BL/6 x DBA/2 F_1 (BD2F_1) mice (*H-2b/H-2d*) 9 days after iv injection of spleen cells from N-tropic Friend virus(FV)-infected DBA/2 donors (*H-2d/H-2d*). To determine whether these spleen colonies resulted from donor or host cell proliferation, the histocompatibility antigen markers of cell populations from dissected colonies and intercolonial areas were compared. Cytotoxic tests with mouse antisera specific for *H-2Db* and *H-2Dd* antigens revealed the presence of both antigen specificities on cells from both sources, indicating that most cells were of host BD2F_1 origin. The presence of donor cells in spleen colonies could be detected, however, when the recipients were lethally irradiated (800 R) BD2F_1 mice, the colonies of which consisted exclusively of cells bearing *H-2Dd* and not *H-2Db* (recipient) antigens. Analogous results were obtained by cytotoxic analysis of spleen colonies in C57BL/6 x C3H/An F_1 mice injected with FV-infected C3H/An spleen cells. In addition, direct visualization of virus-induced antigens and *H-2* antigens by immunofluorescent staining confirmed the cytotoxicity data. The colony-forming efficiency of *H-2d* cells was found to vary only slightly among unirradiated recipients of different *H-2* haplotypes. These findings indicate that spleen colony formation by FV-infected leukemia cells was based on donor cell proliferation in irradiated hosts and on host cell proliferation in unirradiated hosts (for the strains used here). The spleen colony assay for Friend leukemia cells in unirradiated hosts is therefore an assay for infectious centers in which virus is apparently released from donor cells in quantities sufficient to infect *Fv-1*-restrictive host cells and to initiate their proliferation. (21 refs)

78-3973 **In Vitro Infection of Mouse Hemopoietic Cells by Friend Virus as Measured by a Spleen Colony Assay for Infectious Centers.** (Eng) Steinheider, G. (Dept. Genetics, Albert Einstein Coll. Medicine, Bronx, NY); Bubbers, J. E.; Steeves, R. A. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 291-294; 1978.

Spleen colony formation was studied in unirradiated or irradiated (850 rads) BDF_1 mice inoculated iv with leukemic spleen cells (5 x 10^4 cells per animal) from Friend virus (FV)-infected DBA/2 mice. Nine days later, the histocompatibility antigen (H-2) type of the colony cells was compared with that of donor and recipient mice. The results showed that spleen colony formation by FV leukemia cells was based on donor cell proliferation in irradiated hosts and on host cell proliferation in unirradiated hosts. Thus, the injected cells acted as infectious centers (IC) in the unirradiated animals, releasing sufficient virus to Fv-1-restrictive host cells. IC were also generated after in vitro infection with FV. To study the effect of the Fv-2-resistant (r) gene on in vitro infection, infected bone marrow cells from DBA/a and congenic D2.Fv-2r mice were injected into CDF_1 and (BALB/c x D2.Fv-2r) hybrids, respectively. The frequency of IC was tenfold higher in DBA/2 cells than in D2.Fv-2r cells. The Fv-2-resistant animals may have fewer target cells for FV infection or their bone-marrow cells may release less virus after infection. (4 refs)

78-3974 **Studies on the Interaction Between Chemical Carcinogens and Leukemia Viruses for the Promotion of Cellular Transformation and Mutagenesis.** (Eng) Mishra, N. K. (Microbiological Associates, Bethesda, MD, 20014); Premkumar-Reddy, E.; Aulakh, G. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 249-250; 1978.

The relationship between chemical carcinogens and leukemia viruses for the promotion of cellular transformation was investigated. When Fischer rat embryo cells were infected with Rauscher leukemia virus and exposed to several carcinogens, transformation was accelerated and ouabain-resistant colonies were induced. Carcinogen treatment did not lead to an additional accumulation of viral genomes in the host DNA. The interactions between chemical carcinogens and leukemia viruses are concluded to be complex and probably multistep events. (no refs)

78-3975 **Immune Reactivity of RBL-3, a Nonproducer Leukemia Induced by Rauscher Murine Leukemia Virus in C57BL/6 Mice.** (Eng) Chang, K. S. (Viral Oncogenesis Section, Lab. Cell Biology, NCI, NIH, Public Health Service, U.S. Dept. Health, Education, and Welfare, Bethesda, MD, 20014). *J Natl Cancer Inst* 61(1): 107-112; 1978.

The in vitro characteristics of RBL-3, a Rauscher murine leukemia virus (R-MuLV)-induced leukemia of C57BL/6 mice, were investigated along with its virus inducibility by 5-iodo-2′-deoxyuridine (IUdR) and its oncogenicity and immune reactivity. No infectious virus particles were found, but viral structural proteins were detected on the membrane of RBL-3 cells by membrane immunofluorescence when cells were treated with anti-gp69/71 or anti-p30 serum. An NB tropic virus indistinguishable from R-MuLV was recovered after treatment with IUdR (optimal concentration, 200 μg/ml). Dexamethasone treatment slightly boosted the production of activated virus but did not by itself activate the virus. RBL-3 cells had Fc receptors, but no Thy 1.2 antigen or surface immunoglobulin. In mice inoculated with RBL-3 ip, the sc tumor mass and the spleens and lymph nodes showed some characteristics of a reticulum cell neoplasm, but their undifferentiated appearance did not permit definite classification. RBL-3 cells exhibited the Friend-Moloney-Rauscher virus-specific transplantation antigen, as shown by their immunosensitivity and immunogenicity in tumor rejection experiments. Crude membrane and crude soluble antigen preparations obtained from RBL-3 membranes retained the capacity to induce tumor rejection. These results indicate that RBL-3 leukemia is a virus nonproducer that contains an R-MuLV genome that can be activated. (24 refs)

78-3976 **Cellular Membrane Receptors for Oncovirus Envelope Glycoprotein: Properties of the Binding Reaction and Influence of Different Reagents on the Substrate and the Receptors.** (Eng) Bishayee, S. (Indian Inst. Experimental Medicine, 4, Raja Subodh Mullick Road, Calcutta 700032, India); Strand, M.; August, J. T. *Arch Biochem Biophys* 189(1): 161-171; 1978.

The binding of the purified envelope glycoprotein (gp69/71) of Rauscher murine C-type oncovirus (RaV) to intact cells and cell membranes was characterized. The binding of radiolabeled gp69/71 was specific to cells permissive to RaV infection (NIH 3T3, BALB/c 3T3, SC-1 and Swiss 3T3FI). There was no binding to mink, rabbit cornea, and human muscle/skin cells, which are nonpermissive to this virus. Specificity was also demonstrated with respect to viral interference: NIH 3T3 cells infected with Ra, Gross, or BALB/c N-tropic virus; BALB/c cells infected with endogenous BALB/c B-tropic virus; and SC-1 cells infected with Ra or AKR virus failed to bind gp69/71. In contrast, NIH cells infected with Moloney sarcoma virus, BALB/c cells infected by Kirsten sarcoma virus, and SC-1 cells infected with BALB xenotropic virus bound the virus envelope gp. The data suggest that recognition of host cells occurs immediately upon absorption and does not require penetration of the cell or modification of the envelope gp. The interaction is reversible, the gp dissociated from the cells by incubation with EDTA shows no change in size, and a substantial fraction of the dissociated gp is capable of repeated binding to cells. Studies of the effect of chemical modification on the ptor binding is a function of the protein moiety, not the carbohydrate one. Treatment with chemicals acting on protein confirmations resulted in substantial loss of activity. In contrast, digestion of gp69/71 with neuraminidase alone or with β-galactosidase did not affect binding activity. The marked sensitivity of gp68/71 membrane binding sites to trypsin, chymotrypsin, and phospholipase C indicates that the receptor is probably a protein and that it may require lipid for its activity or for the integrity of the membrane. (45 refs)

78-3977 **Biosynthesis of Reverse Transcriptase from Rauscher Murine Leukemia Virus by Synthesis and Cleavage of a *gag-pol* Read-through Viral Precursor Polyprotein.** (Eng) Kopchick, J. J. (Dept. Biology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); Jamjoom, G. A.; Watson, K. F.; Arlinghaus, R. B. *Proc Natl Acad Sci USA* 75(4): 2016-2020; 1978.

The synthesis of reverse transcriptase (RT) from Rauscher murine leukemia virus (RLV) was studied in RLV-infected NIH Swiss mouse embryo fibroblasts (JLS-V16) and BALB/c spleen-thymus (JLS-V5) cell lines. RT was synthesized in the infected cells by way of a read-through polyprotein of 200,000 mol wt (Pr200-*gag-pol*), which was precipitated by antiserum to RT. PR200-*gag-pol* contains both p30 and RT peptide sequences. Intermediate RT-related precursors of 145,000 (Pr145-*pol*), 135,000 (Pr135-*pol*), and 125,000 (Pr125-*pol*) mol wt were specifically recognized by precipitation from infected cell extracts by antiserum to RT. These proteins shared methionine-containing tryptic peptide sequences with a virion polypeptide of 80,000 mol wt (p80-*pol*) precipitable by antiserum to RT. Purification of active RT enzyme from virions labeled with ^{3}H methionine showed that p80-*pol* was the major component. A polypeptide (Pr80-*pol*), similar in size to and having a nearly identical peptide map to that of mature viral p80-*pol*, was also precipitated from infected cells by antiserum to RT. Pulse-chase studies indicated that Pr80-*pol*, Pr125-*pol*, and Pr135-*pol* were stable polypeptides, but that Pr200-*gag-pol* and Pr145-*pol* were unstable precursors. Pulse-chase studies with cycloheximide showed that the processing of Pr200-*gag-pol* occurred for a short time (30 min) in the absence of protein synthesis. (15 refs)

78-3978 **Morphological Conversion of 'Immature' Rauscher Leukaemia Virus Cores to a 'Mature' Form after Addition of the P65-70 (*gag* Gene Product) Proteolytic Factor.** (Eng) Yoshinaka, Y. (Worcester Foundation of Experimental Biology, Inc., Shrewsbury, MA, 01545); Luftig, R. B. *J Gen Virol* 40(part 1): 151-160; 1978.

Studies showed that a partially purified proteolytic factor from Rauscher leukemia virus (RLV) specifically cleaves P65-70, the *gag* gene precursor polyprotein, to p30 and that this cleavage is accompanied by a change of the RLV core in the intact virion from an immature to a mature form. To analyze which substructures of the intact virion were necessary for this large-scale morphological conversion to occur, an attempt was made to duplicate the process in vitro. When the factor was added at increasing concentrations to immature core subparticles of RLV, there was an increasing amount of cleavage of both P65-70 and P40 (an intermediate cleavage product containing p30) to p30. Electron microscopy showed that this cleavage was correlated with the disappearance of immature cores and the appearance of mature cores. Apparently, the concentric coil of the immature core region opened up and was converted through a series of ill-defined stages into mature cores. The mature cores had a hexagonal profile, did not contain any outer micellar material, and were about 7 nanometers smaller than the immature forms. Intermediate structures that retained elements of both forms were also observed, suggesting that the in vitro conversion occurs on a 1:1 basis. The fact that the conversion could be duplicated in vitro eliminates the outer virion envelope and glycoprotein spikes as required functional elements for this process. (16 refs)

78-3979 **Binding of tRNA to Reverse Transcriptase of RNA Tumor Viruses.** (Eng) Panet, A. (Dept. Virology, Hebrew Univ., Hadassah Medical Sch., Jerusalem, Israel); Berliner, H. *J Virol* 26(2): 214-220; 1978.

The interaction of transfer RNA (tRNA) with the reverse transcriptase (RT) of Moloney murine leukemia virus (M-MuLV) and simian sarcoma virus (SiSV) was examined. The purified RT of the viruses sedimented in glycerol gradients as a globular protein with a mol wt of 70,000. After interaction with tRNA, however, the enzyme cosedimented with a protein of 150,000 molecular wt. The enzyme complexes were partly dissociated by degradation of the tRNA moiety by pancreatic RNase A. The RT released from virions of M-MuLV, SiSV, and avian myeloblastosis virus (AMV) by nonionic detergent migrated faster on glycerol gradients than the purified enzyme preparation. This was probably due to complex formation between part of the virion enzyme and the tRNA, which is endogenous in virions. However, addition of exogenous tRNA was needed to quantitatively complex all the virion RT of M-MuLV and SiSV. The RT of M-MuLV did not show tRNA species specificity in the binding reaction when glycerol gradients were used for assay. Several tRNA species of *Escherichia coli*, yeast, chicken, and rat origin were able to complex with the enzyme. AMV RT also bound tRNA species of *E. coli*, yeast, and chicken origin. These results demonstrate that the RT of mammalian RNA tumor viruses exists in two forms; the purified enzyme is a single polypeptide with a mol wt of 70,000 that is converted into a 150,000-mol wt complex after binding of tRNA. (25 refs)

78-3980 **Genomic Masking of Nondefective Recombinant Murine Leukemia Virus in Moloney Virus Stocks.** (Eng) Fischinger, P. J. (Lab. Viral Carcinogenesis, NCI, Bethesda, MD, 20014); Blevins, C. S.; Dunlop, N. M. *Science* 201(4354): 457-459; 1978.

HIX virus cloned from Moloney leukemia virus (M-MuLV) stock is a nondefective, leukemogenic, and amphotropic murine oncornavirus with a recombinant-type major glycoprotein (gp71). Newborn mice inoculated with HIX virus produced lymphomas that were indistinguishable from the original M-MuLV-induced disease. Although M-MuLV stocks generally contain little or no free amphotropic virus, dilution analysis of several virus stocks and the examination of virus progeny from individual foci revealed that HIX virus is present and functionally coated with ectropic Moloney virus envelopes. De novo generation of recombinant virus apparently is rare or does not occur in tissue culture. The presence of recombinant, nondefective, and leukemogenic HIX virus in M-MuLV stocks has a counterpart in MCF virus, which is related to AKR-MuLV. Because most mice have serum factors that inactivate recombinant viruses, genomic masking may be operative as a general survival mechanism for HIX and other analogous recombinant viruses. (16 refs)

78-3981 **Virus-specific RNA Synthesis in Interferon-treated Mouse Cells Productively Infected with Moloney Murine Leukemia Virus.** (Eng) Fan, H. (Tumor Virology Lab., Salk Inst., San Diego, CA, 92112); MacIsaac, P. *J Virol* 27(2): 449-452; 1978.

NIH-3T3 mouse cells productively infected with Moloney murine leukemia virus (M-MuLV clone 1 cells) were treated with interferon (IF: 30 units for 16-24 hr), and intracellular virus-specific RNA was determined by hydridization with complementary DNA. The steady-state concentration of virus-specific RNA was 1.7 times greater in IF-treated cells than in untreated cells. This suggests that virus-specific RNA synthesis continues after IF treatment and that the increased level of virus-specific RNA may result from the failure of virus particles containing virion RNA to bud from the cell surface. This suggestion was tested by determining the rate of virus-specific RNA synthesis (ie, the hybridization of pulse-labeled nuclear RNA) in treated and untreated cells. The fraction of labeled virus-specific RNA was approx equal in both cell groups. Thus, virus-specific RNA synthesis continued at approx normal rates in IF treated mouse cells chronically infected with M-MuLV. The data support the conclusion that the primary site of the action of IF on MuLV producer cells is at virus assembly. (18 refs)

78-3982 **Size Analysis and Relationship of Murine Leukemia Virus-specific mRNA's: Evidence for Transposition of Sequences During Synthesis and Processing of Subgenomic mRNA.** (Eng) Fan, H. (Tumor Virology Lab., Salk Inst., San Diego, CA, 92112); Verma, I. M. *J Virol* 26(2): 468-478; 1978.

Virus-specific messenger RNA (mRNA) from purified polyribosomes of mouse cells infected with Moloney murine leukemia virus (M-MuLV) was analyzed by electrophoresis in agarose gels, followed by hybridization of gel slices with M-MuLV-specific complementary DNA (cDNA). Two virus-specific mRNA's of 38S and 24S were detected. The 38S virus-specific mRNA, which is extremely similar if not identical in size to virion 38S RNA, contained the nucleotide sequences for all three M-MuLV structural genes. The 24S virus-specific mRNA was predominantly derived from the 3 half of the M-MuLV genome, since cDNA-*gag(pol)* (complementary to the 5′ half of the M-MuLV genome) could not efficiently anneal with this mRNA. Sequences complementary to cDNA synthesized from the extreme 5′ end of M-MuLV 38S RNA (cDNA 5′) were present in the 24S virus-specific mRNA, since cDNA 5′ (130 nucleotides) efficiently annealed with this mRNA. The annealing of cDNA 5′ was not due to repetition of 5′ terminal nucleotide sequences at the 3′ end of M-MuLV 38S RNA. The sequences in 24S virus-specific mRNA recognized by cDNA 5′ were not present in 3′ fragments of virion RNA that were of the same length. Therefore, it is suggested that RNA sequences from the extreme 5′ end of the M-MuLV genome may be transposed to sequences from the 3′ half of the M-MuLV 38S RNA during synthesis and processing of the 24S virus-specific mRNA. (36 refs)

78-3983 **Interstrain Variation of the Major Internal Structural Component (p30-gag) of Two Murine Oncornaviruses: Comparative Immunochemical, Biochemical, and Biophysical Analysis.** (Eng) Burnette, W. N. (Fred Hutchinson Cancer Res. Center, Seattle, WA, 98104); Mitchell, W. M. *J Virol* 26(2): 522-531; 1978.

The biophysical and biochemical properties of the major internal structural protein (p30-gag) of Moloney murine leukemia virus and endogenous Y-1 murine oncornavirus were compared. Although the two viral proteins share murine group-specific antigenic determinants, the Y-1 virus p30 appeared to have both a lower relative number of these determinants and a decreased affinity at the cross-reactive sites for Moloney virus p30 monospecific antibodies. Immunological analysis indicated the presence of unique antigenic sites on the Moloney virus p30 not shared by the analogous Y-1 virus molecule. The two polypeptides copurified and had similar isoelectric points (pH 6.2-6.3) and sedimentation coefficients (2.47S). However, equilibrium sedimentation yielded a significant mass difference, 28,300 daltons for the Moloney virus protein and 31,000 daltons for the Y-1 virus protein. Amino acids analysis indicated a concomitant increase in total residues for the Y-1 virus, p30, although a number of residues appeared to have been conserved between the two proteins. Conformational studies and hydrodynamic calculations indicated secondary and tertiary structural differences between the proteins. Two-dimensional mapping of the ^{125}-I-labeled tryptic peptides of each p30 suggested that considerable sequence heterogeneity is responsible for many of the biophysical, biochemical, and immunochemical differences between the two. (47 refs)

78-3984 **Viruses, Human Leukemias and Sarcomas.** (Eng) Boiron, M. (Lab. Experimental Hematology, Institut de Recherches sur les Leucemies, Hopital Saint-Louis, 75010 Paris, France). *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 3-6; 1978.

Complementary DNA (cDNA) probes were prepared using Moloney murine sarcoma virus(murine leukemia virus) [M-MSV(MuLV)] and simian sarcoma virus(simian sarcoma murine-associated virus) [SSV(SSAV)] and used to investigate human leukemias. In the first experiment, bone marrow cells, buffy coats, and continuous human cell lines from 46 leukemia cases and 10 controls were examined using the murine probe. Hybrids were identified in 22 of the leukemia cases and in none of the controls. Positive cases were found in all the different categories of leukemia, with the exception of chronic myeloid leukemia. Although hybridization rates were highly variable, some samples had hybridization levels similar to that of M-MSV(MuLV) RNA. In a second experiment, 12 leukemias and 4 controls were studied using both probes. There was a certain correlation between the two probes with respect to the positivity or negativity of the results, but this correlation was far from absolute. Some cases that were negative with the simian probe were positive with the murine probe and vice versa. The human RNA sequences detected by the two probes were shown to be unrelated. These results indicate that MSV- and SSV-like sequences are found in human DNA in addition to baboon endogenous virus (BEV)-like sequences. These findings, taken together with previous ones, suggest that all human tissues contain an endogenous provirus distantly related to BEV and that in certain leukemia patients, two independent sets of oncor navirus-related sequences (one related to murin oncornaviruses and another related to BEV) are present i the DNA. Sequences related to SSV-gibbon ape leukemi virus may be present in the cytoplasm of leukemic cells, bu they are not found in the DNA. (no refs)

78-3985 **The Effect of Mouse Interferon on the Transformation of NIH/3T3 Mouse Cells by Murin Sarcoma Virus.** (Eng) Morris, A. G. (Dept. Biologica Sciences, Univ. Warwick, Coventry, CV4 7AL, England) Clegg, C. *Virology* 88(2): 400-402; 1978.

The effect of interferon (IF) on the transformation of mous NIH/3T3 cells by the Kirsten strain murine sarcoma viru (MSV) was studied at various low multiplicities of infection Two assays of MSV transformation independent of virus replication were used: focus formation under conditions i which foci developed from cells infected with MSV alone an colony formation in agar suspension medium. Pretreatmen of cells with purified IF (10^4 units/ml) inhibited transformation upon infection with MSV by about 90%. Reconstructio experiments indicated that IF treatment did not block focu or colony formation by cells already stably transformed b MSV. If inhibited transformation per se, and not virus replication and spread. These findings indicate that there is a IF-sensitive step early in transformation. (11 refs)

78-3986 **Nucleases and Adenosine 3′,5′-Cyclic Monophosphate Phosphodiesterase Activities in Murine Sarcoma Virus (Moloney)-infected Mice.** (Eng) Tsou K. C. (Harrison Dept. Surgical Res., Sch. Medicine, Univ Pennsylvania, 1515 Ravdin Inst., 3100 Spruce St., Philadelphia, PA, 19101); Herberman, R. B. *J Natl Cancer Ins* 61(4): 1077-1083; 1978.

To provide information on the role of nucleases in oncogeni virus infection, the activities of 3′-nucleotide phosphodiesterase (3′-NPDase), 5′-nucleotide phosphodiesterase (5′ NPDase), acid deoxyribonuclease (DNase II), and 3′,5′ cyclic AMP phosphodiesterase (cAMPDase) in spleen extracts of murine sarcoma virus-infected C57BL/6 inbre mice were studied. Mice were inoculated im in the thigh region with 0.05 ml of a 1:20 dilution of the virus extract (x 10^6 focus-forming units/ml). This treatment produced tumors at the inoculation site in all recipients. At the peak o tumor growth and of the cell-mediated cytotoxic (CMC) response against tumor-associated antigens, 3′-NPDase, 5′ NPDase, and DNase II all showed depressed activities in th spleen, whereas the activity of cAMPDase increased at th peak of the CMC response and remained elevated thereafter Serum enzyme activities of the infected mice were also determined, and only 3′-NPDase in serum correlated well with th CMC response. Inasmuch as the correlation of tumor growt with the CMC response was established in this system, further study of tumors with variance between CMC and growt is necessary to determine if serum 3′-NPDase is a useful biochemical marker for the CMC response in vivo. (62 refs)

78-3987 **New Type B Retrovirus Isolates Associated with Kinetochores and Centrioles of the Host Cell.** (Eng) Heine, U. I. (Lab. Viral Carcinogenesis, NCI, Bethesda, MD, 20014); Todaro, G. J. *J Gen Virol* 39(1): 41-52; 1978.

Two new virus isolates, M432 and M832, obtained from the Southeast Asian mouse were characterized with respect to their ultrastructural composition and intracellular assembly. These viruses can be differentiated morphologically from each other by the structure of their virus envelopes, both in ultrathin sections and in negatively stained virus preparations. The mature virions resemble in certain respects the B-type murine retroviruses. The new isolates, however, have an intracellular precursor, an A-type particle, closely associated with the mitotic apparatus. The intracellular transport of the A-type particles to the cell surface, where they are released by budding, is closely associated with the microtubule system of the cell. The data also give evidence, for the first time, that virions belonging to the family of Retroviridae can be related to centriole-associated virus precursors. Since centrioles are known to be self-replicating organelles, it is speculated that this association could represent a possible mechanism for the vertical transmission of these viruses. (21 refs)

78-3988 **Presence of a Mouse Mammary Tumor Virus-related Antigen in Human Breast Carcinoma Cells and Its Absence from Normal Mammary Epithelial Cells.** (Eng) Yang, N. S. (Dept. Biology, Michigan Cancer Foundation, 110 E. Warren Ave., Detroit, MI, 48201); McGrath, C. M.; Furmanski, P. *J Natl Cancer Inst* 61(5): 1205-1208; 1978.

The human breast cancer cell line MCF-7 and normal human mammary epithelial cells were assayed for the presence of murine mammary tumor virus (MuMTV)-related antigen(s). Antigen(s) related to the major external glycoprotein (gp52) of MuMTV was found by immunofluorescence assays in the MCF-7 cells and in MuMTV producer cells, but not in any of the normal human mammary epithelial cells. Some of the primary cultures of cells from normal donors were treated with progesterone (10^{-8} M) and/or dexamethasone (10^{-5} M) prior to immunoassay. These hormones, which have been shown to stimulate MuMTV expression markedly in mouse tumor cells and to enhance the production of MuMTV-related antigens in MCF-7 cells, did not change the negative response of the normal human breast cells to anti-MuMTV sera. Even though normal human breast cells may contain virus particles, these particles are apparently not related to MuMTV. (25 refs)

78-3989 **Type B and Type C RNA Tumor Virus Replication in Single Cells. Electron Microscopy with Tannic Acid.** (Eng) Schidlovsky, G. (Medical Dept., Brookhaven Natl. Lab., Upton, NY, 11973); Shibley, G. P.; Benton, C. V.; Elser, J. E. *J Natl Cancer Inst* 61(1): 91-95; 1978.

Cells of a murine mammary tumor cell line (Mm5mt/c_1) were treated with tannic acid during specimen preparation, and thin sections were examined by electron microscopy. Simultaneous replication of murine mammary tumor virus (MuMTV; B-type) and murine leukemia virus (MuLV; C-type) was demonstrated along continuous stretches of the plasma membrane of single cells. The tannic acid treatment permitted improved discrimination between the B- and C-type virus in the thin sections. The B-type surface spikes were shown to be a homogeneous band of intermediate density and constant width on the bud surfaces, but the C-type buds had relatively smooth, spike-free envelopes. Both B- and C-type buds may contain morphologically identical horseshoe-shaped nucleoids, and, therefore, discrimination between the types must be made by ascertaining the presence or absence of characteristic spikes on the surfaces of the virus buds. The results show that complete replication of MuMTV and MuLV particles can occur simultaneously in the same host cell. (25 refs)

78-3990 **Mouse Mammary Tumor Virus and Murine Leukemia Virus Cell Surface Antigens on Virus Producer and Nonproducer Mammary Epithelial Tumor Cells.** (Eng) Schochetman, G. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD, 21501); Fine, D. L.; Massey, R. J. *Virology* 88(2): 379-383; 1978.

Mouse mammary tumor virus (MMTV)- and murine leukemia virus (MuLV)-specific cell-surface antigens (CSA) on virus producer and nonproducer mammary epithelial tumor cells were studied. The techniques used included lactoperoxidase-catalyzed iodination of cell surface proteins followed by radioimmune precipitation with monospecific antisera to the major MMTV (gp52, gp36, p27, and p10) and MuLV (gp70 and p30) proteins. The incorporation of iodinated CSA into extracellular virus was determined by analyzing labeled proteins in purified virus. Both gp52 and gp70 were present on the surface of cells producing only MMTV. Furthermore, gp52 was the only labeled protein in extracellular MMTV produced by these cells. On cells producing MMTV and MuLV, both gp52 and gp70 were present on the cell surface, and they were the only labeled proteins present in their respective extracellular viruses. This indicates that gp70 and gp52 were present on mutually exclusive cellular viral budding sites. In addition, MuLV anti-p30 serum precipitated two iodinated proteins with mol wts of 85,000 and 95,000 daltons, analogous to the Gross CSA. Since labeled gp52 and gp70 were also present on the surface of cells producing no virus but producing large amounts of MMTV glycoproteins and nonglycoproteins, they represent true CSA. These results show that the precursor to the MMTV glycoproteins (gPr75-MMTV *env*) is cleaved prior to the appearance of gp52 on the cell surface. (28 refs)

78-3991 **Immunological Characterization of the Low-Molecular-Weight DNA Binding Protein of Mouse Mammary Tumor Virus.** (Eng) Arthur, L. O. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD); Long, C. W.; Smith, G. H.; Fine, D. L. *Int J Cancer* 22(4): 433-440; 1978.

The purification of p14, the low-mol wt DNA-binding protein of mouse mammary tumor virus (MMTV), and its immunological properties are discussed. This protein was purified to homogeneity by guanidine-agarose chromatography followed by affinity chromatography on single-stranded DNA sepharose. Analysis of DNA-binding fractions by sodium dodecyl sulfate-polyacrylamide gel electrophoresis revealed a polypeptide of 14,000 daltons. Immunoprecipitation assays showed that MMTV p14 shared no cross-reactivity with gp52, gp36, or p10, antigens associated with the MMTV envelope, or with p27, which is found in the virion core. MMTV p14 did not cross-react with the low-mol-wt DNA-binding proteins of murine leukemia virus (MuLV). Cross-reactivity was observed between p14 and intracytoplasmic A particles, supporting the concept that A particles are morphological precursors to MMTV cores. Shared antigenic determinants were also demonstrated between intracytoplasmic A particles and MMTV p27, p20, and p10. p14 did not show cross-reactivity with the low-mol-wt DNA-binding proteins of C- or D-type viruses of higher mammals (Mason-Pfizer monkey virus, squirrel monkey retrovirus, baboon leukemia virus, AKR MuLV, and equine infectious anemic virus) in competitive radioimmunoassays. These results indicate that MMTV p14 is immunologically distinct from other MMTV proteins as well as from the DNA-binding proteins of other retroviruses. (29 refs)

78-3992 **Prevalence of Murine Mammary Tumor Virus Antibody and Antigens in Normal and Tumor-bearing Feral Mice.** (Eng) Fine, D. L. (Viral Oncology Program, NCI Frederick Cancer Res. Center, P.O. Box B, Frederick, MD, 21501); Arthur, L. O.; Gardner, M. B. *J Natl Cancer Inst* 61(2): 485-491; 1978.

Normal and tumor-bearing feral mice (*Mus musculus*) from three southern California areas, Asian *Mus* species, and *M. musculus* subspecies were studied to determine whether they possess antibody (Ab) to murine mammary tumor virus (MuMTV) or MuMTV-related antigens, particularly MuMTV gp52. Mice from Lake Casitas (LC) and La Puente (LP), California, have a high spontaneous lymphoma incidence, mice from Bouquet Canyon (BC) a low incidence. Radioimmunoassay showed that approx 20% of LC, LP, and BC mice expressed gp52 in specific tissues and that the organ distribution, predominantly in submaxillary glands and seminal vesicles (in addition to LC mammary tumors), was similar to that observed in inbred strains and did not differ with trapping location. In a radioimmune precipitation assay using ^{125}I-labeled MuMTV, only 6% of LC, LP, and BP mice contained precipitating Ab to MuMTV. Although moderate to high levels of gp52 were expressed in mammary tumor tissues of 3/7 LC and 3/3 (B10 x LC)F$_1$ mice, these animals showed no detectable MuMTV-precipitating Ab. Neither MuMTV Ab nor gp52 was detected in 10 lymphoma-bearing and 5 hepatoma-bearing LC mice. Low levels of MuMTV Ab and gp52 were detected in three *M. musculus* subspecies and in the Asian species *M. cervicolar*. Compared with normal and tumor-bearing inbred mice of high (C3H/HeN) and low (C3H/HeNf) mammary tumor strains, normal and tumor-bearing feral mice expressed low frequencies of MuMTV Ab and gp52. (39 refs)

78-3993 **Mapping of Linear and Circular Forms of Mouse Mammary Tumor Virus DNA with Restriction Endonucleases: Evidence for a Large Specific Deletion Occurring at High Frequency During Circularization.** (Eng) Shank, P. R. (Dept. Microbiology, Univ. California, San Francisco, CA, 94143); Cohen, J. C.; Varmus, H. E.; Yamamoto, K. R.; Ringold, G. M. *Proc Natl Acad Sci USA* 75(5): 2112-2116; 1978.

Linear and circular forms of unintegrated viral DNA were isolated from rat hepatoma cells infected with the GR strain of mouse mammary tumor virus. A DNA transfer procedure was then used to prepare restriction endonuclease fragment maps of these viral DNA forms. According to these maps: (1) the major species of viral DNA is a linear molecule (mol wt 5.9 x 10^6 daltons); located in the cytoplasm; (2) the nuclei contain covalently closed circular viral DNA of two distinct sizes (5.1 x 10^6 and 5.9 x 10^6 daltons) in addition to linear molecules (5.9 x 10^6 daltons); (3) the linear molecule has specific termini; (4) there is extensive homology between regions at or near termini of the linear molecule; (5) the predominant form of circular DNA lacks the 1.2 kilobase pairs present in the larger circular molecule and the linear molecule; (6) sequences deleted from most of the circular DNA molecules are located at the ends of the linear DNA that are joined during circularization. (24 refs)

78-3994 **Lysine tRNA Is the Predominant tRNA in Murine Mammary Tumor Virus.** (Eng) Waters, L. C. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830). *Biochem Biophys Res Commun* 81(3): 822-827; 1978.

The transfer RNA's (tRNA's) of mouse mammary tumor virus (MMTV) isolated from AKR mouse fibroblasts grown in vitro and from the milk of RIII mice were characterized. In both systems, lysine tRNA was the major tRNA in both the "free" 4S RNA and the "70S-associated" 4S RNA. The amount of lysine tRNA associated with the 70S RNA of MMTV was essentially the same as that reported for proline tRNA in AKR murine leukemia virus. (15 refs)

8-3995 **Type C Virus Replication and Expression in Human Rodent Hybrid Cells.** (Eng) Gazdar, A. . (NCI, Bethesda, MD, 20014); Edgell, C. J.; Oie, H. K.; .ussell, E. K.; Brown, S.; Lalley, P.; Minna, J. D. *In: Ad-ances in Comparative Leukemia Research 1977. Proceedings f the VIIIth International Symposium on Comparative Re-earch on Leukemia and Related Diseases, held in Amster-'am, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Iilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Iolland Biomedical Press): 503 pp.; 238-239; 1978.

Iuman cells were infected with baboon M7, feline xenotrop-:, and woolly monkey nontransforming component viruses, ised with nonpermissive mouse cells, and studied for virus eplication. The mouse genome suppressed the production of ll three viruses in the hybrid cells. The M7 virogene was not ompletely eliminated as human chromosomes were lost. 'urther studies indicated that the mouse cells restricted virus utput but not virus entry or integration. It appeared that uman chromosome 19 and possibly, chromosome 3 are es-ential for M7 replication. (no refs)

78-3996 **Macromolecular Synthesis in Cells Infected by Frog Virus 3. IX. Two Temporal Classes of Early Viral RNA.** (Eng) Willis, D. B. (Div. Virology, St. Jude Children's Hosp., P. O. Box 318, Memphis, TN, 38101); Granoff, A. *Virology* 86(2): 443-453; 1978.

By selectively inhibiting functional viral protein synthesis with cycloheximide (CX) or fluorophenylalanine (FPA) and analyzing the resultant viral RNA's by polyacrylamide gel electrophoresis and hybridization, early frog virus 3 (FV3) RNA could be subdivided into two sequentially synthesized classes. The polyacrylamide gel pattern of viral RNA synthesized 6 hr postinfection (PI) in FPA-treated cells was identical to that of the RNA synthesized at 1 hr PI in the absence of drug. RNA synthesized at 6 hr PI in the presence of FPA hybridized to 36% of the viral genome, but RNA synthesized 6 hr PI in its absence contained many additional species and hybridized to 50% of the genome. Therefore, the FPA RNA's appeared to represent the same sequences as the early viral RNA. In contrast, viral RNA's synthesized when protein synthesis was inhibited by cycloheximide (CX) did not contain all of the species present at 1 hr PI or in FPA RNA. The CX RNA's which hybridized to only 16% of the genome, comprised a subset of the FPA RNA's. One of the viral RNA species was synthesized at a much higher rate in CX-exposed cells than during infection in the presence or absence of FPA. When the addition of CX was delayed until 1 or 2 hr PI, all the remaining early viral RNA species were synthesized, and the rate of synthesis of the overproduced RNA decreased. Therefore, FV3 early RNA synthesis can be divided into two temporal classes: "immediate early", which occurs in the absence of protein synthesis CX and "delayed early", which occurs under conditions of restricted protein synthesis (FPA). The data also suggest that at least one species of immediate early viral RNA was subject to negative transcriptional control. (19 refs)

78-3997 **A Portion of the Feline Leukaemia Virus Genome Is Not Endogenous in Cat Cells.** (Eng) Okabe, H. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD, 21501); duBuy, J.; Gilden, R. V.; Gardner, M. B. *Int J Cancer* 22(1): 70-78; 1978.

An attempt was made to distinguish the feline leukemia virus (FeLV)-related sequences in normal cat cell DNA from those of FeLV-infected cellular DNA. Total ^{3}H-complementary DNA (^{3}H-cDNA) transcripts, prepared by viral endogenous reverse transcriptase reactions, were first annealed with excess DNA from normal cat liver (CL) under relaxed conditions. The portion remaining single-stranded after exhaustive hybridization was isolated and further purified by reannealing with the homologous viral 70S RNA. These cDNA probes, termed ^{3}H-cDNA(-CL), were used in hybridization kinetic analyses with cellular DNA from various sources. This probe could clearly distinguish virus-infected cells from noninfected cells by both hybridization extent and melting profiles of the resulting hybrids. It was found that (1) a portion of the genome of FeLV is not endogenous in normal cat cell DNA, and (2) this portion is found in cells and tissues that contain the virus. The same approach using the recycled ^{3}H-cDNA(-CL) of RD114 virus, another endogenous feline virus, failed to show any difference between a virus-infected cell line and normal cells, including both virus-inducible and noninducible lines. This finding suggests that the procedures are specific for FeLV. (33 refs)

78-3998 **Abrogation of Lymphocyte Blastogenesis by a Feline Leukaemia Virus Protein.** (Eng) Mathes, L. E. (Dept. Veterinary Pathobiology, Ohio State Univ., 1925 Coffey Road, Columbus, OH, 43210); Olsen, R. G.; Hebebrand, L. C.; Hoover, E. A.; Schaller, J. P. *Nature* 274(5672): 687-689; 1978.

The cause of the lymphocyte unresponsiveness (decline in cell-mediated immunity with no decline in circulating T cells) accompanying feline leukemia virus (FeLV) infection in cats was investigated. Purified p15 and p27 (the 15,000- and 27,000-mol wt structural proteins of FeLV) were tested for their inhibitory effect on the concanavalin A-lymphocyte blastogenesis (Con A-LBT) response of lymphocytes from normal, specific-pathogen-free cats. Addition of purified p15 reduced the Con A-LBT response by 45%-92% of that obtained with control cultures free of p15. p15 was inhibitory at concentrations ≥0.2 μg/well. Addition of p27 had no effect. Further studies indicated that addition of p15 had no effect on lymphocyte viability, it was not toxic to Con A-stimulated cells, and it was not a competitive inhibitor of Con A binding. These findings suggest that p15 is the mediator of FeLV-associated lymphocyte unresponsiveness. (20 refs)

78-3999 **Influence of Thymectomy on the Susceptibility of Cats to Feline Leukemia Virus and Lymphosarcoma.** (Eng) Hoover, E. A. (Dept. Veterinary Pathobiology, Ohio State Univ., Columbus, OH, 43210); Krakowka, S.; Cockerell, G. L.; Mathes, L. E.; Olsen, R. G. *Am J Vet Res* 39(6): 993-995; 1978.

Of 12 cats thymectomized at age 5 wk, 6 were inoculated at age 8 wk and 6 at age 4 mo with the Rickard strain of feline leukemia virus (FeLV-R), which produces a high incidence of thymic lymphosarcoma (LSA). The cats received 1.0 ml of an inoculum containing 10^3 focus-forming units of FeLV infectivity. Two groups of age-matched nonthymectomized cats were inoculated with the same FeLV-R stock. Thymectomy prior to FeLV infection had no influence on the induction of viremia or LSA incidence. In the FeLV-inoculated nonthymectomized group, two cats developed thymic LSA. In the thymectomized group, three cats developed LSA in the intestine, mesenteric lymph nodes, or bone marrow, but the malignant lymphoblasts had surface markers characteristic of feline T lymphocytes. It is concluded that the presence of the thymus per se is not required for infection and oncogenesis by FeLV and that feline T lymphocytes may be transformed after peripheralization to other tissues. (28 refs)

78-4000 **Differential Growth and Transmission in Cats of Feline Leukaemia Viruses of Subgroups A and B.** (Eng) Jarrett, O. (Dept. Veterinary Pathology, Veterinary Sch., Univ. Glasgow, Bearsden, Glasgow G61 1QH, Scotland); Russell, P. H. *Int J Cancer* 21(4): 466-472; 1978.

Cats were infected with feline leukemia virus (FeLV) of subgroups A (FeLV-A) or B (FeLV-B), and the course of infection was followed. After FeLV-A infection, virus was quickly recovered from the blood and oropharynx of most animals. Following infection with FeLV-B, only a small proportion of cats developed a viremia and then only after a long interval. There was no evidence that FeLV-B was transmitted by contact. In cats infected with mixtures of FeLV-A and -B (FeLV-AB), each virus operated independently. In a high proportion of the cats, but not in all cats, FeLV-A was recovered from the plasma first and FeLV-B appeared later. There was evidence of interaction, however, in that the proportion of cats that were viremic with FeLV-B was greater following FeLV-AB infection than after infection with FeLV-B alone. FeLV-B was transmitted by contact from cats excreting FeLV-AB. These results help to explain the apparent dependence of FeLV-B on FeLV-A under natural conditions and the frequency of occurrence of subgroups A and B in FeLV isolates. It is suggested that the differences between FeLV-A and FeLV-B may be caused by each virus infecting a different cell population. (19 refs)

78-4001 **Immunological Nature of the Inhibition of BLV-induced Early Polykaryocytosis by Bovine Sera.** (Eng) Astier, T. (Groupe d'Etude de la Leucose Bovine, Institut National de la Sante et de la Recherche Medicale, Unite de Recherches de Radiobiologie Experimentale, de Cancerologie U-117, 229 cours de l'Argonne, 33076 Bordeaux, France); Mamoun, R.; Guillemain, B.; Parodi, A. L.; Duplan, J. F. *Int J Cancer* 22(1): 47-54; 1978.

A quantitative in vitro assay, the early polykaryocytosis inhibition (EPI) test, was developed for the serological detection of bovine leukemia virus (BLV) infection in cattle. The EPI assay is based on the inhibition by bovine sera of syncytia formation in mixed cultures of BLV-infected fetal lamb kidney cells and mouse sarcoma virus-infected feline cells. These investigations were concerned specifically with characterizing the EP-inhibiting factor. The immunoglobulin (Ig) nature of the factor was demonstrated by its high mol wt, its sensitivity to digestion with proteinase-K but not RNase or DNase, and by studies ruling out the participation of serum complement or interferon. Removal of Ig's from positive serum fractions completely destroyed the EP-inhibiting ability. Serum fractionation by gel filtration and affinity chromatography indicated that EP-inhibiting activity is mediated only by Ig's of the IgG class. These antibodies were classified as IgG1 by ion-exchange chromatography. Although the fundamental mechanism of EP has not been clarified completely, the EPI technique, because of its specificity for BLV and its high sensitivity, can be adapted to large-scale seroepidemiological surveys in cattle. (20 refs)

78-4002 **Analysis of Human Diploid Cell Cultures for the Presence of Oncornavirus Determinants.** (Rus) Stepanova, L. G. (No affiliation given); Andzhaparidze, O. G.; Avakova, A. N.; Liozner, A. L.; Mel'nikova, N. L.; Korchak, O. B.; Lotte, V. D.; Shukhmina, N. R.; Rozina, E. E. *Vopr Virusol* (2): 170-176; 1978.

The expression of primate leukemia virus (PLV) was assayed in various strains of human diploid cell cultures (human embryo lung cells: WI-38, and strains L-58 and L-63). The assays included determination of reverse transcriptase activity, molecular hybridization, evaluation of infectivity in a KC test, immunodiffusion, immunofluorescence, and evaluation of tumorigenicity in Syrian hamsters. During passage in the second to third growth phases, all three diploid cell cultures retained the morphological and karyological characteristics of normal fibroblasts. The cultures were found to be free of oncornavirus particles or virus-specific antigens. Transitory tumorigenicity was recorded only in the 44th passage of strain L-63. L-58 strain cells were tumorigenic in hamsters at passages 27 and 43; subsequent passages were not tumorigenic. (15 refs)

78-4003 **Hepatitis B Surface Antigen and Primary Liver Cancer.** (Eng) Van Den Heever, A. (Dept. Internal Medicine, H. F. Verwoerd Hosp., Privale Bag X169, Pretoria, 0001, Republic S. Africa); Pretorius, F. J.; Falkson, G.; Simson, I. W. *S Afr Med J* 54(9): 359-361; 1978.

The incidence of hepatitis B surface antigen (HBsAg) was determined in the sera of 92 black patients with primary liver cancer (PLC) and 92 matched controls with no history of liver disease. Sera from 31 PLC patients and 8 controls were positive for HBsAg; this difference was significant ($p < 0.01$). Associated liver disease was diagnosed in 56 PLC patients. Of these patients, HBsAg was found in the sera of 8/36 patients with chronic Budd-Chiari syndrome, 6/9 with post-collapse cirrhosis, 1/9 with siderosis, and 0/2 with fibrosis. It is suggested that the pathogenesis of PLC has the following cycle: hepatitis B infection, cirrhosis, PLC. These findings combined with those in the literature suggest that the role of hepatitis B infection in the pathogenesis of PLC varies in different geographical areas. (23 refs)

78-4004 **Virus-specific Markers and Virus-like Particles in Cell Lines of Tumors Produced by CELO Virus in Syrian Golden Hamsters.** (Eng) Asch, B. B. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX, 77030); McCormick, K. J.; Stenback, W. A.; Trentin, J. J. *J Natl Cancer Inst* 61(1): 163-171; 1978.

Five cell lines were established from chicken embryo lethal orphan (CELO) virus-induced hamster tumors and examined for the presence of CELO virus-specific markers and hamster viruslike particles (VLP). Two of the cell lines were derived from primary hepatocellular carcinomas (HCC) arising in CELO virus-inoculated LSH/Lak inbred hamsters and three from sarcomas that developed in CELO virus-inoculated outbred hamsters. Cell lines from two of the sarcomas (COT and CMT) and one HCC (CEHEP) produced CELO virus-specific tumor (T) antigen. The antigen was not detected in cells of the third sarcoma line (RCT) until they had undergone >34 passages in vitro. Although 5%-10% of cells in the second HCC line (CILT/2) contained T antigen during early passages, it was not detected after the fifth subculture. Cells of both HCC lines possessed CELO virus tumor-specific transplantation antigen. All five cell lines also contained hamster R-type particles, and both HCC lines had C-type and intracytoplasmic A-type particles. The percentage of carcinoma cells producing R-type particles increased during cultivation in vitro, whereas the number of cells with A-type particles decreased. Treatment with dibutyryl cyclic AMP and theophylline enhanced the number of cells producing C-type particles in one HCC line and R-type particles in two of the sarcoma lines. This study provides no evidence that indigenous VLP of the hamster are more than passengers in CELO virus-induced tumors. CELO virus appear to be the primary etiologic agent. However, the host cell may exert a regulatory function on the expression of CELO virus-related markers. (49 refs)

78-4005 **Application of the Immuno-peroxidase Method to the Study of Tumor Viruses. 1. DNA Tumor Viruses.** (Eng) Imamura, M. (Dept. Pathology, Sapporo Medical Coll., Sapporo, Japan); Toh, K.; Ueno, H. *Tumor Res (Sapporo)* 12: 14-28; 1977.

The use of immunoperoxidase methods to study the ultrastructural localization of the cell-surface antigens of DNA tumor viruses is described. Although satisfactory and reproducible results were obtained, several technical problems remain to be settled, mainly dealing with cell fixation. (40 refs)

78-4006 **Membrane Alterations Associated with "Transformation" by BUdR in BUdR-Dependent Cells. Fluorescence Polarization Studies with a Lipid Probe.** (Eng) Rosenthal, S. L. (Div. Human Genetics, Children's Hosp. Medical Center and Dept., Microbiology and Molecular Genetics, Harvard Medical Sch., Boston, MA, 02115); Parola, A. H.; Blout, E. R.; Davidson, R. L. *Exp Cell Res* 112(2): 419-429; 1978.

Steady-state and nanosecond fluorescence polarization (FP) analyses were used to study the membrane fluidity of a bromodeoxyuridine (BUdR)-dependent cell line (B4) derived from a malignant Syrian hamster melanoma line. In the presence of BUdR, B4 cells resemble transformed cells. For comparison, the cells were also grown in the absence of BUdR (as 'untransformed' cells). Membrane fluidity was estimated from FP measurements of membrane lipids labeled with the fluorescent probe 1,6-diphenyl-1,3,5-hexatriene, a technique used previously to show that FP values differ between normal and malignant lymphocytes and between untransformed and virally transformed 3T3 cells. The steady-state FP values of the membrane lipids of B4 + BUdR cells were larger than those of the lipids of B4-BUdR cells; ie, the membranes of the 'transformed' B4 cells were less fluid than those of the 'untransformed' B4 cells. The change in FP values was found to be fully reversible. Nanosecond rotational correlation time experiments confirmed and extended these results. In view of the findings and those of previous studies, it is suggested that the membranes of the B4 + BUdR cells resemble those of virally transformed 3T3 cells and the membranes of B4-BUdR cells resemble those of untransformed 3T3 cells. Line B4 offers a unique opportunity for elucidating the metabolic alterations involved in malignant transformation. (23 refs)

78-4007 **Natural Transmission of Gibbon Leukemia Virus.** (Eng) Kawakami, T. G. (Comparative Oncology Lab., Univ. California, Davis, CA, 95616); Sun, L.; McDowell, T. S. *J Natl Cancer Inst* 61(4): 1113-1115; 1978.

The possible natural mode of transmission of gibbon ape leukemia virus (GaLV) was investigated. Transmission of infectious virus was based on the detection of humoral antibody, presence of infectious virus, or on molecular hybridization. It was found that GaLV can infect prenatal gibbons through in utero infection or postnatal gibbons through contact transmission. The transmission of infectious virus was from viremic gibbons and not from uninfected or antibody-positive animals. The two modes of transmission could be distinguished by the amount of proviral DNA integrated into the muscle tissue of viremic gibbons. Muscle of gibbons infected postnatally had little or no proviral DNA, whereas gibbons infected prenatally had a large quantity of proviral DNA. Examination of cellular DNA for provirus in 'nontarget' organs may be useful in establishing the natural mode of transmission of exogenous oncornaviruses. (18 refs)

78-4008 **The Major Core Protein of Mammalian Type C Viruses: Structural Comparisons of a Conserved Gene Product.** (Eng) Gautsch, J. W. (Scripps Clinic and Res. Foundation, La Jolla, CA, 92037); Elder, J. H.; Jensen, F. C.; Lerner, R. A. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 237; 1978.

The major core proteins of > 40 mammalian C-type viruses were mapped and (MuLV) p30's;, the p27's of the woolly monkey-gibbon ape viruses and a xenotropic mouse virus; and the p27's from baboon, cat, and rat C-type viruses formed three distinct structural groups. In addition, several structural subtypes of MuLV p30's were noted. The close relationship between the p27's of RD114 and M-7 viruses and the p27's of feline leukemia viruses and rat viruses suggests that they are of recent common origin. (no refs)

78-4009 **Natural Distribution of Squirrel Monkey Retrovirus Proviral Sequences in Primate DNAs.** (Eng) Schochetman, G. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD, 21501); Fine, D. L. *J Gen Virol* 40(part 1): 257-261; 1978.

The cellular DNA of various primates was examined for the presence of virus genes related to those present in squirrel monkey retrovirus (SMRV) by nucleic acid hybridization. ^{3}H-SMRV 70S RNA hybridized extensively to squirrel monkey DNA, whereas no significant hybridization above background was observed between SMRV and the DNA of New World monkeys (woolly monkey, capuchin, owl), Old World monkeys (rhesus, African green), apes (gibbon, chimpanzee), and humans (A204 human rhabdomyosarcoma cell line, uninfected or infected with Mason-Pfizer monkey virus). The hybridization conditions used would only detect highly conserved (perfectly matched) sequences. The extent of hybridization to the DNA'S of liver, kidney, and brain from two different squirrel monkeys ranged from 52% to 76%, and it correlated with the number of proviral sequences, as measured by the $C_0t1/2$ values. These values indicated that SMRV provirus was present in multiple copies (5-10) in these tissues. In contrast, a max of 30% hybridization was obtained with the DNA of SMRV-infected A204 cells, a result of the low number (1-2 copies) of proviral sequences present in these cells. These results demonstrate that SMRV is a new endogenous, vertically transmitted virus of squirrel monkeys and the first isolated from a New World primate. (13 refs)

78-4010 **Comparison of Mason-Pfizer Monkey Virus and Squirrel Monkey (*Saimiri sciureus*) Retrovirus by Immunoelectron Microscopy.** (Eng) Smith, G. C. (Southwest Foundation Res. and Education, P.O. Box 28147, San Antonio, TX, 78284); Heberling, R. L.; Kalter, S. S. *J Natl Cancer Inst* 61(2): 411-413; 1978.

Immunoelectron microscopy indicated that Mason-Pfizer monkey virus and squirrel monkey retrovirus (both candidate D-type oncornaviruses) do not share common surface antigens. In addition, there was no indication of cross-reactivity with baboon endogenous virus antiserum and the surface antigens of the two viruses. (16 refs)

78-4011 **Comparative Studies on Non-Type C Primate Retraviruses: Squirrel Monkey Virus (SMRV) from New World Monkeys and Mason-Pfizer Virus (MPMV) from Old World Monkeys.** (Eng) Fine, D. L. (Viral Oncology Program, NCI Frederick Cancer Res. Center, P.O. Box B, Frederick, MD, 21501); Schochetman, G.; Arthur, L. O.; Gilden, R. V. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 242-244; 1978.

Studies of the relationship between squirrel monkey virus (SMRV) from New World monkeys and Mason-Pfizer virus (MPMV) from Old World monkeys indicated that SMRV is antigenically distinct from various mammalian retraviruses and that it is present in multiple copies in the DNA of squirrel monkeys. According to hybridization experiments, SMRV is endogenous to squirrel monkeys and shares little sequence homology with MPMV. (5 refs)

78-4012 **Immunological and Structural Relationships Between Langur Virus and Other Primate Type-D Retroviruses.** (Eng) Colcher, D. (Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD, 20014); Teramoto, Y. A.; Schlom, J. *Virology* 88(2): 384-388; 1978.

The major structural proteins of three D-type retroviruses, Mason-Pfizer monkey virus (MPMV) from rhesus monkeys, squirrel monkey retrovirus (SMRV), and langur retrovirus (LV), were compared using sodium dodecyl sulfate-polyacrylamide slab and tube gel electrophoresis and competitive radioimmunoassays (RIA's). Squirrel monkeys are a New World species, but the rhesus and langur monkeys are Old World species. The virion-associated polypeptides of LV and MPMV had indistinguishable electrophoretic mobilities in polyacrylamide gels. Homologous RIA's for the major 27,000-dalton structural protein (p27) of LV and for the p27 of MPMV also could not distinguish between these two viruses. LV and MPMV could be differentiated, however, by either of the RIA's developed for their 10,000- to 12,000-dalton proteins. The major structural protein of SMRV was more immunoreactive with LV than with MPMV. An interspecies RIA was also developed using antiserum to LV to precipitate ^{125}I-labeled MPMV p27. This assay was more effective than either of the homologous LV or MPMV RIA's for the recognition of SMRV, and it may be useful in detecting and characterizing new primate retrovirus isolates. (15 refs)

78-4013 **Biological and Biochemical Properties of a Human Oncornavirus System (PMF).** (Eng) Bierwolf, D. (Zentralinstitut fur Krebsforschung, Akademie der Wissenschaften der DDR, 1115 Berlin-Buch, E. Germany); Wunderlich, V.; Micheel, B.; Graffi, A. *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 38-47; 1978.

Virus particles that appeared in a human embryonic cell line obtained from the embryo of a woman who had generalized carcinoma of the cervix uteri were characterized. Morphological experiments indicated that the particles were considered a D-type oncornavirus (designated PMF virus) with a structure very similar to that of Mason-Pfizer monkey virus. Of several antisera tested, only antiserum to Mason-Pfizer monkey virus produced immunoprecipitation lines with the PMF virus or virus-containing cell extracts. Thus, these two types of viruses are closely related. Biochemical investigations indicated that both exogenous and endogenous reverse transcriptase (RT) activity is present in the virus particles. This RT required the presence of divalent cations and was preferentially activated by Mg++ rather than by Mn++. According to preliminary data obtained by gel electrophoresis, the virus contains the polypeptides p10, p16, p30, gp40, p49, and gp65. The data suggest that PMF virus is of human origin. (10 refs)

78-4014 **Type D Retroviruses: Occurrence of Natural Antibodies to Mason-Pfizer Monkey Virus in Rhesus Monkeys.** (Eng) Fine, D. L. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD, 21501); Devare, S. G.; Arthur, L. O.; Charman, H. P.; Stephenson, J. R. *Virology* 86(2): 567-571; 1978.

Sera of rhesus monkeys maintained in colonies at three separate geographic regions were found to possess naturally occurring antibody to the Mason-Pfizer monkey virus (MPMV) major structural protein, p26. The specificity of this reactivity was established by absorption with purified viral protein. Animals from two of the colonies were housed in association with MPMV-infected monkeys, but no such prior exposure to virus-infected animals could be traced in the case of the third colony. These results, in combination with previous inability to detect full complements of MPMV genetic sequences in the cellular DNA of normal rhesus monkeys, provide evidence for the horizontal transmission of MPMV. Alternatively, the present findings could reflect a humoral immune response to expression of an endogenous D-type retrovirus highly related to MPMV. (25 refs)

78-4015 **Simian Mason-Pfizer Virus-induced Chronic Infection in Primate Cell Cultures.** (Rus) Nosik, N. N. (D. I. Ivanovskii Inst. Virology, Moscow, USSR); Fain, D. L.; Priori, E. S.; Artur, L.; Gonda, M. S.; Plouman, D. T.; Ershov, F. I. *Vopr Virusol* (2): 165-169; 1978.

The expression of simian Mason-Pfizer virus (MPV) was studied in a *Macaca rhesus* prepuce cell culture (Rhfs), a transplantable culture of monkey kidney cells (BSC-1), and a primary culture of trypsinized human embryo kidney cell (HEK). Chronic infection with MPV was established in the Rhsf culture during the third passage. Characteristic features of chronic infection were the presence of virus-specific gs antigen in the perinuclear zone, polynuclear cells, and relatively high levels of RNA-dependent DNA-polymerase activity. Although the BSC-1 cells also showed enhanced DNA synthesis, they lacked polynuclear cells. The HEK culture was the least susceptible to the oncornavirus. There were no signs of transformation in any of the three cultures. (10 refs)

78-4016 **Structures of Polyoma Virus: On the Histone Component and Virion Core.** (Eng) Frost, E. (Montreal Cancer Inst., Notre Dame Hosp., 1560 Sherbrooke St. E., Montreal, Quebec H2Z 4K8, Canada); Bourgaux, P. *J Gen Virol* 39(1): 103-111; 1978.

Purified polyoma virus was able to take up an amount of calf thymus histone equivalent to 10%-50% of its normal histone content under conditions permitting the binding of considerably lesser amounts of several other proteins (myoglobin, ovalbumin, and bovine serum albumin). Some of the bound histone could not be released by procedures routinely used for virus purification. Some of the histone present in purified polyoma virus could be selectively released by detergent treatment without breakdown of virus particles. These observations suggest that a significant proportion of the histone of purified polyoma virus is either located on the surface of the virion or it can readily enter or leave the capsid. (35 refs)

78-4017 **Chromatographic Separation of the Polyoma Virus Proteins and Renaturation of the Isolated VP$_1$ Major Capsid Protein.** (Eng) Brady, J. N. (Div. Biology, Kansas State Univ., Manhattan, KS, 66506); Consigli, R. A. *J Virol* 27(2): 436-442; 1978.

Chromatography of 6 M guanidine hydrochloride-denatured polyoma virion proteins through Sepharose CL-6B beads allowed separation of the dimer, VP$_1$, VP$_2$, VP$_3$, and the histone proteins VP$_{4\text{-}7}$ in highly purified form. Renaturation of VP$_1$ resulted in the formation of subunits that were morphologically, biophysically, and immunologically similar to native virion capsomeres. (20 refs)

78-4018 **BK Virus DNA: Cleavage Map and Sequence Analysis.** (Eng) Yang, R. C. (Dept. Biochemistry, Cornell Univ., Ithaca, NY, 14853); Wu, R. *Proc Natl Acad Sci USA* 75(5): 2150-2154; 1978.

A detailed physical map of BK virus (MM strain) DNA was constructed with respect to the cleavage sites of 11 different restriction enzymes. The enzymes cut the DNA at 61 specific sites, the locations of which were determined. Preliminary nucleotide sequence analysis was performed in the region from 0.70 to 0.75 map units on the viral DNA. An 80% homology was found between 0.714-0.744 map units on BK virus DNA and 0.722-0.752 map units on simian virus 40 (SV40) DNA. This region of SV40 DNA codes for the synthesis of the leader sequence of late messenger RNA. It is likely that only part of this sequence constitutes specific recognition signals and is absolutely required. (27 refs.)

78-4019 **Failure to Demonstrate Papovavirus Tumor Antigen in Human Cerebral Neoplasms.** (Eng) Greenlee, J. E. (Dept. Neurology, Johns Hopkins Univ. Sch. Medicine, Baltimore, MD, 21205); Becker, L. E.; Narayan, O.; Johnson, R. T. *Ann Neurol* 3(6): 479-481; 1978.

Cell cultures established from 80 brain tumors (astrocytoma, meningioma, glioblastoma multiforme, medulloblastoma, ependymoma, and others) were examined by the fluorescent antibody technique for the presence of the intranuclear tumor (T) antigen common to cells transformed by simian virus 40 (SV40) or BK or JC viruses. Brilliant intranuclear fluorescence comparable to that seen in cells cultured from papovavirus-induced hamster cerebral tumors was not seen in any of the human brain tumor cells. A small number of cells from one meningioma showed faint nuclear fluorescence at early passages but not on subsequent passages. These results provide no support for the association of these primate papovaviruses with human cerebral neoplasms. (28 refs)

78-4020 **Brain Tumors in Owl Monkeys Inoculated With a Human Polyomavirus (JC Virus).** (Eng) London, W. T. (Infectious Diseases Branch, Natl. Inst. Neurological and Communicative Disorders and Stroke, Bethesda, MD, 20014); Houff, S. A.; Madden, D. L.; Fuccillo, D. A.; Gravell, M.; Wallen, W. C.; Palmer, A. E.; Sever, J. L.; Padgett, B. L.; Walker, D. L.; ZuRhein, G. M.; Ohashi, T. *Science* 201(4362): 1246-1249; 1978.

Adult feral owl monkeys (*Aotus trivirgatus*) were inoculated intracerebrally, sc, and iv with human JC (14 animals) or BK virus (4) or with simian virus 40 (SV40; 4 monkeys). Six JCV-inoculated monkeys received immunosuppressive doses of cyclophosphamide and prednisone for 10 wk before and 10 wk after JCV inoculation. One mo after inoculation, all JCV monkeys had antibody titers to JCV of 80-2,560 and BKV monkeys had antibody titers to BKV of 160-10,240, as determined by hemagglutination-inhibition. Antibody titers to SV40, as determined by plaque-reduction neutralization were 80-$\geq$160 in SV40 monkeys. Antibodies against tumor (T) antigen appeared in the serum of all virus-inoculated animals after 3-5 mo, and they were still detectable after 3 yr. Two of four surviving JCV monkeys developed brain tumors, one 16 mo and one 25 mo after inoculation. Neither monkey had been immunosuppressed. A Grade 3 to Grade 4 astrocytoma (resembling a human glioblastoma multiforme) was found in the left cerebral hemisphere and brainstem of one monkey. The second monkey developed a malignant tumor in the left cerebral hemisphere that contained both glia and neuronal cell types. Impression smears of the second tumor showed numerous cells containing intranuclear T antigen; virion antigens were not detected. Tumor cells cultured in vitro also contained T antigen, but they were negative for virus antigen. Infectious virus was not isolated from tumor extracts. These results suggest that the oncogenicity of JCV extends to nonhuman primates. (30 refs)

78-4021 **Effect of Inactivated Sendai Virus on Adsorption and Penetration of Polyoma Virus.** (Eng) Artamonova, V. A. (N. F. Gamaleia Inst. Epidemiology and Microbiology, Moscow, USSR); Shevliagin, V. I. *Vopr Virusol* (2): 187-192; 1978.

The effect of Sendai virus (SV) on the adsorption, elution, and penetration of oncogenic polyoma virus (PV) was studied in primary cultures of murine, chick, and human embryo fibroblast cultures. Within 3 hr of adsorption of ^{3}H-thymidine-labeled PV, the SV-treated permissive murine fibroblast culture contained approx 60% of radioactivity in the nuclear fraction. In the nonpermissive SV-treated chick and human fibroblast cultures, 46%-57% of the radioactivity was detected in the granular fraction but only 14% in the nuclear fraction. SV enhanced the penetration of PV only in the permissive culture. (8 refs)

78-4022 **Viral Sequences Related to a Human Skin Papillomavirus in Genital Warts.** (Eng) Orth, G. (Unite' de Recherches sur l'Etiologie Virale des Cancers Humains, U140 INSERM, LA147 CNRS, Institut Gustave-Roussy, 94800 Villejuif, France); Favre, M.; Jablonska, S.; Brylak, K.; Croissant, O. *Nature* 275(5678): 334-336; 1978.

Total DNA from the genital warts (condylomata acuminata) of seven patients as well as purified form I DNA preparations were hybridized with ^{3}H-labeled RNA's complementary (cRNA's) to four types of human papillomavirus (HPV-1, -2, -3, and -4) to determine whether the genital wart DNA had sequences homologous to HPV DNA. No significant hybridization was obtained with any of the genital wart DNA's (total or form I) and HPV-1, HPV-2, or HPV-3 cRNA's. However, all seven total DNA preparations as well as purified form I DNA from two patients showed significant levels (3.5%-8.8%) of hybridization with the HPV-4 cRNA probe. Thus, viral sequences related to HPV-4 are present in the DNA of the warts studied, but it is not known to what extent the sequences are related. (22 refs)

78-4023 **A Search for an Association Between Simian Virus 40 and Human Meningiomas.** (Eng) Cikes, M. (Ludwig Inst. Cancer Res., 1066 Epalinges, Switzerland); Beth, E.; Giraldo, G.; Acheson, N.; Hirt, B. *In: Mechanisms of Carcinogenesis. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4 (Book B), 698 pp.; 1009-1011; 1977.

Twenty-four human meningiomas were examined for the presence of simian virus 40 (SV40) tumor (T) antigen. All tumors were negative by indirect immunofluorescence. In addition, RNA from the six tumors tested failed to hybridize with SV40 DNA, and extracts of 16 tumors were negative in complement fixation tests. Finally, no antibodies against the T antigens of SV40, JC, or BK viruses were detected in the sera of all 16 patients analyzed. (9 refs)

78-4024 **The Structure of Genes, Intergenic Sequences, and mRNA from SV40 Virus.** (Eng) Thimmappaya, B. (Dept. Human Genetics, Yale Univ. Sch. Medicine, New Haven, CT, 06510); Reddy, V. B.; Dhar, R.; Celma, M.; Subramanian, K. N.; Zain, B. S.; Pan, J.; Weissman, S. M. *Cold Spring Harbor Symp Quant Biol* 42(part 2): 449-456; 1978.

Analysis of the nucleotide sequence of simian virus 40 (SV40) DNA showed that the late proteins VP2 and VP3 are coded for by overlapping messages translated in the same reading frame. The initiator codon for VP3 may be a methionine codon within the gene for VP2. The codons for the carboxy-terminal amino acids of VP2 and VP3 were read in a second frame as the codons for the amino-termini amino acids of VP1. The largest continuous stretch of triplets in SV40 DNA that could be transcribed into sense codons included $<75\%$ of the early region, and it could only code for a protein of mol wt $<70{,}000$. This contrasted with the reported mol wt of the SV40 'A' protein of 70,000-94,000. Almost one-third of the SV40 DNA did not code simply for known proteins. The remaining DNA included sequences complementary to the untranslated 3′ ends of early and late messenger RNA (mRNA), sequences at the origin of DNA replication, reiterated sequences, and sequences that could potentially code for small polypeptides. One such sequence lies between the origin of replication and the initiator codon for VP2. The transcript of much of this DNA was abundantly represented in cytoplasmic polyadenylated RNA, but there appeared to be a short gap at 0.765 fractional genome length where little or no unmodified transcript was present in the cytoplasm. The sequence preceding 0.765 appeared to be covalently joined to the RNA sequence of transcripts of DNA beginning from 0.95 and onward. This composite RNA was the 16S mRNA that has been reported to direct the synthesis of VP1. (26 refs)

78-4025 **Polysomes and Polysomal mRNAs in SV40-infected CV-1 Cells. In Vivo Observations.** (Eng) Ho-Terry, L. (Dept. Bacteriology, Univ. Coll. Hosp. Medical Sch., University St., London WC1E 6JJ, England); Terry, G. M.; Cohen, A. *Biochim Biophys Acta* 519(2): 390-397; 1978.

The association of late simian virus 40 (SV40)-specific messenger RNA's (mRNA's) with polysomes in virus-infected (100 plaque-forming units/cell) African green monkey kidney CV-1 cells was examined. A comparison of polysomes in mock- and SV40-infected cells was made by labeling the cells with ^{3}H-uridine prior to their infection. Sucrose gradient centrifugation indicated that the polysomes from the virus-infected cells were heavier and UV absorbance indicated that they contained more ribosomes than the polysomes of mock-infected cells. Approx 40%-45% of the mRNA's on the polysomes of the infected cells were virus-specific. The mRNA's coding for the virion polypeptides VPI, II, and III synthesized per unit time was determined by measuring the radioactivity incorporated by them per minute. The ratio of VPI to VPII + III was 3.38:2.16. Since the 16S mRNA codes for VPI and the 19S mRNA codes for the other two polypeptides, the amount of these mRNA's associated with the polysomes was also determined. The 16S and 19S mRNA's were present in a ratio of 1.88:1.00, similar to the ratio obtained with the polypeptides. Thus, the relative amounts of virus-specific polypeptides synthesized in virus-infected cells is determined by the relative amounts of 16S and 19S mRNA's coding for them. (16 refs)

78-4026 **Cytogenetic Studies of a Simian Virus 40 Temperature-sensitive Mutant-transformed Chinese Hamster Cell Clone at the Permissive and Nonpermissive Temperature.** (Eng) Lehman, J. M. (Dept. Pathology, Univ. Colorado Medical Center, 4200 E. Ninth Ave., Denver, CO, 80262); Robinson, C. C. *J Natl Cancer Inst* 61(4): 1171-1174; 1978.

A clone of Chinese hamster embryo cells transformed by tsA58, the temperature-sensitive mutant of simian virus 40, was analyzed for chromosome abnormalities at the permissive temperature (37 C) and nonpermissive temperature (40.5 C). The cells of this clone, A58/1, were grown at 37 C for 1 day; then one bottle was shifted to 40.5 C and subcultured (2 passages) for 1 wk. The cells were harvested for cytogenetic studies after a 2 hr treatment with colcemid (0.05 μg/ml) and processed for trypsin-Giemsa banding. The metaphases from cells at both temperatures were pseudodiploid, with numerous chromosome changes primarily affecting chromosomes 1 and 2. Other chromosomes (6, 11, and the X) were also frequently involved. A marker chromosome, LM, was present in 35% of the cells at 40.5 C. Chromosome changes have been associated with malignant transformation, making the SV40 tsA-transformed cells an interesting model to determine the relationship of modulation (temperature shift) of the transformed phenotype to chromosome changes. (12 refs)

78-4027 **Transcription, Processing and Structure of Chromatin in SV40-transformed Cell.** (Eng) Chae, C. B. (Dept. Biochemistry, Univ. North Carolina, Chapel Hill, NC, 27514); Wong, T. K.; Gadski, R. A. *Biochem Biophys Res Commun* 83(4): 1518-1524; 1978.

The transcription, processing, and structure of chromatin in simian virus 40 (SV40)-transformed 3T3 cells (SVB209) cells, which contain one copy of the SV40 genome, were studied. Nuclear RNA from SVB209 cells was hybridized with a trace amount of the separated strands of tritium-labeled SV40 DNA. When the nuclear RNA was hybridized with the E strand, the entire length of the viral DNA strand was transcribed. However, only 40% of the L strand was transcribed in the SVB209 cells. Examination of the RNA extracted from the cytoplasm showed that the E strand was derived from 55% of the SV40 DNA. Therefore, although the entire length of the E strand of SV40 DNA is transcribed, only half the length of the RNA is transported into the cytoplasm. More than 90% of the SV40 DNA in the nuclei of SVB209 cells was completely digested by DNase I. Hybridization experiments indicated that this result was not due to the generation of very short DNA fragments by DNase I. It is most likely a result of structural modification of the chromatin. The sensitivity of >90% of the SV40 DNA in the SVB209 nuclei is in good agreement with transcription of the entire length of the E strand of this DNA in the cell. It appears, however, that both strands of the SV40 DNA in the transformed cell are sensitive to DNase I. (24 refs)

78-4028 **Nucleotide Sequence of the Simian Virus 40 Small-t Gene.** (Eng) Volckaert, G. (Lab. Molecular Biology, State Univ. Ghent, Ledeganckstraat 35, B 9000 Ghent, Belgium); Van de Voorde, A.; Fiers, W. *Proc Natl Acad Sci USA* 75(5): 2160-2164; 1978.

The nucleotide sequence of simian virus 40 (SV40) DNA between map positions 0.649 and 0.533 is presented. This segment is approx half of the restriction fragment *Hin*d A, it is
located near the beginning of the early region, and it is tra
scribed counterclockwise. A potential initiating ATG codo
is located 13 nucleotides from the *Hin*d C-*Hin*d A junctio
in the strand with the same polarity as early messenger RN
From this signal on, an open reading frame is present th
would allow the synthesis of a polypeptide of 174 amino aci
until a TAA termination codon is reached at nucleotide 6
(map position 0.547). DNA sequence analysis indicated th
this polypeptide corresponds almost exactly to small (t) t
mor antigen. Correlation of the deduced amino acid sequen
with the NH_2-terminal sequences of SV40 small t and lar
T antigens suggests that both proteins are initiated at t
ATG triplet. Because the DNA region between 0.547 a
0.534 is blocked for translation in all three reading fram
by multiple termination codons, it is concluded that the lar
T antigen must be coded for by two noncontiguous DN
segments: the segment from 0.65 to approx 0.60, which bo
antigens share, and another segment starting at some poi
after position 0.534 and continuing counterclockwise until
terminates at map position 0.174. Small t antigen is methi
nine-rich and has a remarkably high number of cysteine re
dues clustered mainly in its COOH-terminal half. It is ri
in both basic and acidic residues, with the former being sligh
ly in excess. (37 refs)

78-4029 **Large and Small Tumor Antigens from Simi
Virus 40 Have Identical Amino Termini Ma
ping at 0.65 Map Units.** (Eng) Paucha, E. (Translation La
Imperial Cancer Res. Fund, Lincoln's Inn Fields, Lond
WC2A 3PX, England); Mellor, A.; Harvey, R.; Smith, A.
Hewick, R. M.; Waterfield, M. D. *Proc Natl Acad Sci U*
75(5): 2165-2169; 1978.

Large and small tumor (T) antigens of simian virus 40 (SV4
were synthesized in vitro with L-cell extracts that had be
treated to prevent amino-terminal acetylation of nascent pr
teins to determine whether both antigens share the sa
amino-terminal amino acid sequence. The partial amino-t
minal amino acid sequences of both forms of T antigen we
identical. Methionine residues were located at positions 1 a
14, a lysine residue at position 3, and leucine residues at po
tions 5, 11, 13, 16, 17, and 19. These amino acid sequen
data match perfectly the amino acid sequence predicted fro
a sequence of nucleotides in the E strand of SV40 DNA th
begins near the junction between *Hin*dII/III fragments A a
C, at about 0.65 map unit. This finding strongly suggests th
the sequence coding for the amino terminus of both protei
is located at this position. The data are consistent with
model for the synthesis of both forms of T antigen that pr
dicts that (1) small T antigen is coded for by a sequence
nucleotides from the 5′ end of the early region and (2) lar
T antigen is coded for by nucleotide sequences from two no
contiguous regions of SV40 DNA. (31 refs)

78-4030 **Contact-inhibited Revertant Cell Lines Isolat
from SV40-transformed Cells. VIII. Membra**

Protein and Glycoprotein Composition. (Eng) Vessey, A. R. (Dept. Immunology, Cleveland Clinic Foundation, Cleveland, OH, 44106); Culp, L. A. *Virology* 86(2): 556-561; 1978.

The compositions of enriched surface membrane preparations from BALB/c 3T3 simian virus 40 (SV40)-transformed 3T3 (SVT2), and concanavalin A-selected revertant variant cells were investigated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. The electrophoretic profiles of the membrane proteins from the three cell lines were similar. However, ^{14}C-leucine-labeled or Coomassie blue-stained preparations from revertant cells lacked a protein band of 110,000 mol wt (band 15) that was present in 3T3 and SVT2 membranes and that was replaced in revertant membranes by two new bands (15a and 15b) migrating on either side of band 15. Lactoperoxidase-catalyzed iodination studies indicated that the relative proportion of band 5 [presumptive LETS (large external transformation-sensitive) glycoprotein] was decreased and that of band 24 (mol wt, 65,000) was increased in 3T3 cells, compared with the other cell lines. Revertant membranes had increased proportions of sialic acid residues on several glycoproteins. The LETS glycoprotein from all three cell types was found to be sialylated. Two different compartmentalized pools of cell surface-associated LETS were identified. These results are discussed in relation to the method of selection of revertant cells and to possible changes induced in cell surface architecture by transformation and reversion. (20 refs)

78-4031 **Detection of Antigenically Active Mutant HGPRT after Mutagenesis with Simian Virus 40.** (Eng) Strauss, M. (Dept. Cell Genetics, Div. Bioregulation, Central Inst. Molecular Biology, Acad. Sciences GDR, DDR-1115 Berlin-Buch, E. Germany); Theile, M.; Eckert, R.; Geissler, E. *Mutat Res* 51(2): 297-300; 1978.

An antigenically active mutant hypoxanthine-guanosine phosphoribosyltransferase (HGPRT) was detected in Chinese hamster cells following infection with simian virus 40 (SV40). Inhibition assays with V79-4 wild-type cell extracts demonstrated that 50 μl of the tenfold diluted antibody soln were required to precipitate the max amount of HGPRT protein from 50 μg of cell-extract protein. The titration curve of the amount of antibody against the residual enzymatic activity in the supernatant soln demonstrated a region of linear dependence. In this region, the addition of 5 μl of the antibody soln led to a residual enzymatic activity of 70%, but 10 μl antibody soln caused enzymatic activity of only 36%. The amount of cross-reacting material (CRM) or the antigenicity of mutant HGPRT enzymes from virus-induced and spontaneous mutants was determined quantitatively. For each individual clone, the enzymatic activity was between 4,350 and 2,350 disintegrations/min. The CRM values for the six clones ranged from 60% for clone V5 to 100% for clone V150. Therefore, all the virus-induced and spontaneous mutants examined were CRM-positive. Most of the clones seemed to synthesize normal amounts of HGPRT enzyme protein (80%-100%). Thus, the HGPRT enzyme proteins must be caused by point mutations. (12 refs)

78-4032 **Higher Order Structure of Simian Virus 40 Chromatin. Histone H1 Is Responsible for the Condensation of Intranuclear Viral DNA-Protein Complexes.** (Eng) Muller, U. (Dept. Microbiology, Univ. Heidelberg, Im Neuenheimer Feld 230, D-69 Heidelberg, W. Germany); Zentgraf, H.; Eicken, I.; Keller, W. *Science* 201(4354): 406-415; 1978.

Viral DNA-protein complexes isolated from the nuclei of cultured cells infected with simian virus 40 (SV40) were used to study the conditions required for the formation and maintenance of the higher order chromatin structure. At moderate ionic strength, SV40 nucleoprotein complexes contain histone H1 as well as the nucleosomal histones, and they have a compact conformation made up of globular subunits with an av diameter of 190 A. At high ionic strength, histone H1 is released and the structure unfolds into chains averaging 24 nucleosomes in length. Such H1-depleted nucleoprotein complexes can be brought back to a compact state by the addition of purified histone H1 in vitro. Transcriptionally active SV40 chromatin undergoes the same structural transitions. The higher order structure of viral chromatin may be analogous to the compact state of cellular chromatin fibers observed at physiological ionic strength. It appears that the higher order organization of chromatin does not interfere with DNA replication and transcription. (57 refs)

78-4033 **Role of the Simian Virus 40 Gene *A* Product in Regulation of DNA Synthesis in Transformed Cells.** (Eng) Butel, J. S. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX, 77030); Soule, H. R. *J Virol* 26(3): 584-594; 1978.

The kinetics of induction of DNA synthesis were determined for hamster cell lines transformed by temperature-sensitive (*ts*A) mutants of simian virus 40 (SV40). The hamster transformants were shifted to the permissive temperature (33 C) after a 48-hr serum arrest at the nonpermissive temperature (40.5 C). DNA synthesis was initiated in the *ts*A transformants by 8 hr after shiftdown and was max by 12 hr, as determined by incorporation of ^{3}H-thymidine (^{3}H-TdR). The presence or absence of fetal bovine serum at the time of temperature shift had no effect on the kinetics of initiation of DNA synthesis. Analysis of thymidine triphosphate (TTP) in *ts*A transformants revealed simi incorporation of ^{3}H-TdR into TTP at both permissive and nonpermissive temperatures. Autoradiography revealed that by 12 hr after a shift to the permissive temperature, approx 50% of the cells had labeled nuclei after a 60-min pulse with ^{3}H-TdR, indicating that most of the cells were actively synthesizing DNA. By 8-12 hr after a shiftup of confluent *ts*A transformants to the nonpermissive temperature, the number of labeled nuclei was reduced to 16%, regardless of serum concentration. These results indicate that the SV40 gene *A* product directly or indirectly regulates cellular DNA synthesis in transformed cells. (58 refs)

78-4034 **Differences in Behavior and Prognosis Between Leukemic and Lymphomatous Forms of a Transplantable Hamster Lymphocytic Neoplasm Induced by Simian Virus 40.** (Eng) Diamandopoulos, G. T. (Dept. Pathology, Harvard Medical Sch., 25 Shattuck St., Boston, MA, 02115). *Proc Natl Acad Sci USA* 75(4): 2011-2015; 1978.

A lymphocytic leukemia, induced in a Syrian golden hamster by iv injection of simian virus 40, was adapted to grow in the allogeneic host either as leukemia or as lymphoma. The leukemia was produced by transplanting sc neoplastic lymphocytes that had circulated through and/or proliferated in lymph nodes and spleen, and it was characterized by dissemination with systemic manifestations and poor prognosis. The lymphoma was produced by transplanting sc neoplastic lymphocytes that had proliferated at sc sites of cell implantation, and it was characterized by localization and favorable prognosis. The tissue environment that the neoplastic lymphocytes encountered during circulation and/or proliferation must have regulated their subsequent behavior in the intact host. Since the leukemic and lymphomatous forms of this animal model resemble very closely the analogous human lymphocytic neoplasms, the model can be used to study factors responsible for the differences in the behavior of these neoplasms and to determine how these differences may influence prognosis and response to therapy. (27 refs)

78-4035 **Altered Metabolism of Heparan Sulfate in Simian Virus 40 Transformed Cloned Mouse Cells.** (Eng) Winterbourne, D. J. (Macromolecular Biology Section, NCI, NIH, Bethesda, MD, 20014); Mora, P. T. *J Biol Chem* 253(14): 5109-5120; 1978.

Comparisons were made between glycoconjugates (GC's) from a family of closely related cell lines derived from AL/N mouse embryos: a parent clone and three daughter subclones, two of which expressed simian virus 40 (SV40) tumor (T) antigen. Papain-digested macromolecules from the parent, labeled with ^{3}H-glucosamine, and from one of the subclones, labeled with ^{14}C-glucosamine, were compared simultaneously by diethylaminoethylcellulose chromatography. Three culture compartments (the medium, the cell-surface trypsinate, and the cells) from the paired cell lines were combined at the earliest time during the harvesting of the cells. The results demonstrated a close relationship among alterations in the metabolism of heparan sulfate, the expression of SV40-specific T antigen, and the ability of the cells to grow to high densities in monolayer culture, but not in suspension in viscous medium. Apart from the altered elution of HS produced by the T-antigen-positive subclones, the salt concentration at which any given GC eluted from the ion-exchange columns was highly reproducible and similar for each cell line. This allowed preliminary identification of the GC's and assured that the GC's of the individual compartments in each experiment were not subject to unrecognized differences in the isolation procedure. The T-antigen-positive subclones had essentially the same tumorigenicity as the parent clone, which had relatively low tumorigenicity in syngeneic AL/N and nude mice. (49 refs)

78-4036 **Mapping of *H-2* Genes Associated with T Cell-mediated Cytotoxic Responses to SV40-Tumour-associated Specific Antigens.** (Eng) Pfizenmaier, K. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA, 19104); Trinchieri, G.; Solter, D.; Knowles, B. B. *Nature* 274(5672): 691-693; 1978.

The primary cytotoxic responses to simian virus 40 (SV40) tumor-associated specific antigens (TASA) were investigated in B10 and BALB/c mice, and the genes associated with these responses were mapped. The mice were immunized with SV40, and the antigenic specificity of the cytotoxic T lymphocytes (CTL) was determined using SV40- and adenovirus 5 (Ad5)-transformed cells as targets in the ^{51}Cr-release assay. Max cytotoxic activity was obtained in both strains 7 days after in vivo immunization followed by a further 3-day in vitro culture period. CTL could be demonstrated in B10 mice when tested directly 7 days after immunization, and this response was enhanced by in vitro culture. CTL could be demonstrated in BALB/c mice only after the in vitro culture. Target cell lysis was SV40-TASA-specific and syngeneically restricted, as neither Ad5- nor allogeneic SV40-transformed cells were lysed. The findings suggest that suppressor mechanisms may account for the eclipse in immunity in mice bearing syngeneic SV40-induced tumors. Mapping experiments using lymph node cells from various strains of SV40-immunized mice showed that the cytotoxic response to SV40-TASA was associated with the *Kb, Kk, Db,* and *Dd* but not with the *Kd* and *Dk* genes of the major histocompatibility (*H-2*) complex. There may also be a possible *Ir* gene influence on the T cell response to SV40-TASA associated with *H-2Dd*. (20 refs)

78-4037 **Synthesis of the SV40 Viral Polypeptide Vp1 During Infection.** (Eng) Kasamatsu, H. (Molecular Biology Inst., Univ. California, 405 Hilgrad Ave., Los Angeles, CA, 90024); Flory, P. J. *Virology* 86(2): 344-353; 1978.

The synthesis of simian virus 40 (SV40) polypeptides in permissively infected CV-1 cells was studied by a sensitive method in which ^{125}I-labeled antibodies are used to identify and quantitate polypeptides immobilized in gels. The synthesis of Vp1 begins about 24 hr postinfection, with the majority of this polypeptide being found in the nucleus. Smaller quantities of several Vp1-related polypeptides were observed in highly purified virions. Two of these species, with apparent mol wts of about 117,000 and 100,000, were also found in infected cells. They appeared long after Vp1 synthesis had begun, at about 54 hr postinfection in the nuclei and at about 95 hr postinfection in the cytoplasm. (26 refs)

78-4038 **1 Biosynthesis, Immunological Specificity, and Intracellular Distribution of the Simian Virus 40-specific Protein Induced by the Nondefective Hybrid Ad2+ND$_1$.** (Eng) Jay, G. (Pediatric Oncology Branch, NCI, NIH, Bethesda, MD, 20014); Jay, F. T.; Friedman, R. M.; Levine, A. S. *J Virol* 26(2): 411-419; 1978.

A study was made of the synthesis, immunological specificity, and intracellular distribution of a 28,000 (28K)-mol wt protein induced by Ad2+ND$_1$, a nondefective hybrid virus containing a segment of the early region of simian virus 40 (SV40) DNA covalently inserted into the human adenovirus 2 genome. Radioimmunoprecipitation using sera from hamsters bearing SV40-specific tumors indicated that the Ad2+ND$_1$-induced 28K protein is SV40-specific. This protein was synthesized as a 30K-mol wt precursor, and it was detectable only when infected cells were pulse-labeled in the presence of a protease inhibitor. Fractionation of labeled cell extracts revealed that 80% of the 28K protein was in the plasma membrane fraction and that 20% was associated with the outer nuclear membrane. Blockage of proteolytic cleavage did not alter the topographic distribution of the 28K protein; however, the amount of the 30K precursor in the outer nuclear membrane increased fourfold while that in the plasma membrane decreased proportionately. This suggests that the 28K protein is transferred from the outer nuclear membrane to the plasma membrane after posttranslational cleavage of the 30K precursor polypeptide. The data support the proposal that the 28K protein contains the determinants for SV40 U antigen and that it is responsible for SV40 enhancement of adenovirus growth in simian cells. (28 refs)

78-4039 **Studies on the Occurrence of High Molecular Weight Adenovirus Type 2 DNA in Productively Infected Cells.** (Eng) Fanning, E. (Inst. Genetics Fachbereich Biologie, Univ. Konstanz, D-7750 Konstanz, W. Germany); Schick, J.; Doerfler, W. *Virology* 88(1): 186-190; 1978.

The conditions under which high-mol-wt adenovirus type 2 (Ad2) DNA is formed and the factors that determine its quantity and quality were studied. High-mol-wt viral DNA (> 100S and 50S-90S) occurred in Ad2-infected KB, HeLa, and HEK cells and, to a lesser extent, in WI-38, XP4, XP25, BHK21, and CHO cells. The proportion of Ad2 DNA sequences in the 50S-90S DNA varied from one cell line to another, and it declined markedly with increasing cell density. The proportion of newly synthesized DNA in the > 100S, 50S-90S, and < 20S size classes was dependent on the multiplicity of Ad2 infection. In KB cells infected with UV-inactivated Ad2, synthesis of 34S viral DNA, but not high-mol-wt DNA, was reduced or nearly abolished. The data suggest that the synthesis of high-mol-wt viral DNA does not depend on the concurrent synthesis of 34S viral DNA, but that it does require active cell growth, possibly cellular DNA replication. (20 refs)

78-4040 **Adenovirus DNA-directed Transcription of 5.5S RNA In Vitro.** (Eng) Wu, G. J. (Dept. Microbiology, Emory Univ. Sch. Medicine, Atlanta, GA, 30322). *Proc Natl Acad Sci USA* 75(5): 2175-2179; 1978.

A mammalian cell-free and membrane-free system developed from human KB cells was used to investigate the synthesis and regulation of a 5.5S RNA that is produced in adenovirus type 2-infected KB or HeLa cells. Cell-free RNA synthesis was dependent upon exogenous templates, ribonucleoside triphosphates, and the cell-free postmitochondrial supernatant of human KB cells. The synthesis of 5.5S RNA was inhibited only by high levels of α-amanitin (≥ 100 μg/ml); therefore, it was performed by RNA polymerase III. The rate of synthesis was linear for at least 2 hr, indicating reinitiation. The 5.5S RNA synthesized in vitro was similar to the corresponding in vivo RNA in size, sequence, and coding region on adenovirus type 2 DNA. (35 refs)

78-4041 **Synthesis of Transfer Ribonucleic Acid in KB Cells Infected with Adenovirus Type 2.** (Eng) Sehulster, L. M. (Dept. Pathology, CMDNJ-Rutgers Medical Sch., Piscataway, NJ); Varricchio, F.; Raska, K. *J Gen Virol* 40(part 1): 183-194; 1978.

New techniques for separating low-mol-wt (LMW) RNA allowed examination of the synthesis of LMW RNA and changes in transfer RNA (tRNA) synthesis during infection of KB cells with adenovirus type 2. Cells were labeled postinfection (PI), with ^{3}H-uridine or ^{32}P. An increasing amount of virus-coded LMW RNA was detected from 8 hr PI onward. The rate of synthesis of tRNA was unchanged 0-16 hr PI, but it decreased thereafter. From 24 to 48 hr PI, the specific activity of tRNA was about 60% of the control value. The specific activity of cellular 5S RNA increased from 12 hr PI. Two-dimensional gel electrophoresis resolved the tRNA into 42-47 spots, so that changes in tRNA synthesis in individual spots could be seen. From 8 to 12 hr PI, the relative rate of incorporation into RNA increased in seven spots and decreased in eight. At 12-16 hr PI, incorporation into RNA increased in six spots, especially in spot 9 (sevenfold). Simultaneously, incorporation in six spots decreased. At 24-34 hr PI, RNA synthesis increased in eight spots and decreased in eight. Incorporation into RNA in two spots was virtually shut off. The material that migrated in spot 9 contained tRNA. It is hypothesized that increased amounts of a specific tRNA species are required for optimal synthesis of virus DNA in infected cells and that the marked increased RNA synthesis in spot 9 reflects this requirement. Assuming that reproducible changes of incorporation into specific RNA spots reflect changed rates of synthesis of specific individual tRNA species (and not modifications of preexisting tRNA after virus infection), the results show that changes, transient or permanent, in the utilization of certain tRNA's resulting from adenovirus infection can be reflected in the tRNA synthesis rates. However, some of the changes may be due to alterations in the relative mobility of tRNA's, reflecting changes in their primary structure. (38 refs)

78-4042 **Infectivity and Oncogenicity of DNA from Simian Adenovirus SA7 (C8) and Infectivity of DNA from Human Adenovirus Type 6.** (Rus) Vikhnovich, E. M. (D. I. Ivanovskii Inst. Virology, Moscow, USSR); Dreizin, R. S.; Zolotarskaia, E. E.; Chaplygina, N. M.; Ponomareva, T. I.; Gol'dberg, E. Z. *Vopr Virusol* (2): 176-179; 1978.

The infectivity of the DNA from human adenovirus type 6 and the infectivity and oncogenicity of the DNA from simian adenovirus SA7 (C8) was studied in newborn hamsters. Inoculation of the hamsters with isolated DNA from SA7 (5-10 μg, sc) induced tumors in 1/2, 1/9, and 2/9 animals (in three different experiments). The induced tumors contained tumor (T) antigen, and sera derived from the tumor-bearers contained complement-binding antibodies against T antigen. Isolation of the virus from a green monkey kidney cell culture infected with the DNA of SA7 and from a KV cell culture and a human embryo kidney cell culture infected with the DNA of adenovirus type 6 indicated the infectivity of the isolated viral DNA. (9 refs)

78-4043 **Localization of Simian Adenovirus 7 (SA 7) Transcription and Replication in Lytic Infection. An Ultracytochemical and Autoradiographical Study.** (Eng) Moyne, G. (Institut de Recherches Scientifiques sur le Cancer, B.P. no. 8, F-94800 Villejuif, France); Pichard, E.; Bernhard, W. *J Gen Virol* 40(part 1): 77-92; 1978.

High-resolution cytochemistry and autoradiography were used to determine the precise localization of DNA transcription and replication in the nucleus of cells (kidney cells of *Cercopithecus aethiops*) infected with simian adenovirus 7. The majority of studies were made with cells taken 24 hr postinfection, when most cells contained virus particles. The morphology of the nucleus was essentially similar to that of cells infected by other adenoviruses, except that the nucleolus was distinct from the main inclusion body. The nucleolus incorporated uridine much more actively than both the inclusion body and cellular chromatin. Conventional staining distinguished dense and clear fibrillar inclusions in addition to virus particles. Use of a DNA-specific stain allowed the direct visualization of newly formed DNA-containing structures before the appearance of other nuclear alterations. Up to four degrees of DNA packing in the infected cell nucleus were demonstrated: chromatin, types A and B fibrils, and the nucleoids of the virus particles. The A and B fibrils were equated with the clear and dense fibrillar inclusions, respectively. The B fibrils probably represented virus DNA produced in excess in adenovirus-infected cells, the A fibrils the actively transcribing and replicating form of DNA. These findings were confirmed by ribonucleoprotein (RNP) preferential staining and enzymic extractions. The use of serial sections showed that the DNA and RNP staining techniques were complementary. Correlation of these findings with high-resolution autoradiography revealed that virus DNA replication and transcription are carried out in the clear fibrillar inclusions. The functional role of host cell chromatin and of the nucleolus is discussed. (33 refs)

78-4044 **Immunohistochemistry of Local Immunoglobulin Production in Nasopharyngeal Carcinoma.** (Eng) Ho, H. C. (Medical Health Dept., Queen Elizabeth Hosp., Kowloon, Hong Kong); Kwan, H. C.; Ng, M. H. *Br J Cancer* 37(4): 514-519; 1978.

Biopsy specimens from 36 nasopharyngeal carcinoma (NPC) patients were studied histochemically for antibody production by the immunoperoxidase method. Electron micrographs of stained biopsy sections revealed an accumulation of plasma cells, particularly the IgA type, in the connective tissue surrounding nests of tumor cells. The extent of accumulation varied in the different biopsy specimens, which included 35 poorly differentiated, nonkeratinizing squamous or undifferentiated carcinomas and 1 moderately differentiated squamous carcinoma. IgA+ plasma cells were also observed in the submucosal stroma of a choanal polyp, but they were found infrequently in the uninvolved mucosa of a patient with oropharyngeal carcinoma. There was no correlation between the occurrence of IgA+ plasma cells and titers of serum IgA antibodies to Epstein-Barr virus (EBV) capsid antigen. The results suggest that the nasopharynx is the site of local antibody production in response to antigenic stimulation by NPC cells. The local antibody response might also involve the production of antibodies to EBV antigens. (16 refs)

78-4045 **Transforming Activity of Epstein-Barr Virus Obtained by Superinfection of Raji Cells.** (Eng) Yajima, Y. (Dept. Microbiology, Rush-Presbyterian-St. Lukes's Medical Center, Chicago, IL, 60612); Marczynska, B.; Nonoyama, M. *Proc Natl Acad Sci USA* 75(4): 2008-2010; 1978.

Human cord blood lymphocytes were transformed by Epstein-Barr virus (EBV; SI Raji virus) obtained by superinfection of Raji cells with EBV recovered from P3HR1 cells (HR1 virus). A human lymphoblastoid cell line, HLB, established by this transformation contained 22 EVB genomes per cell and EBV-associated nuclear antigen; a few cells contained early or viral capsid antigen complexes. Chromosome analysis revealed that HLB cells were diploid, with normal female karyotypes. Replication of EBV DNA and inhibition of host cell DNA synthesis were observed in HLB cells after superinfection with HR1 virus. Two possibilities are considered for the origin of SI Raji virus. (1) SI Raji virus DNA is the replicational result of parental HR1 virus DNA. HR1 virus DNA is modified or partially deleted during replication of virus DNA, resulting in production of defective virus with transforming activity, (2) SI Raji virus DNA is derived from resident EBV DNA in Raji cells. The regulatory mechanism controlling the expression of EBV genomes in Raji cells is destroyed by HR1 infection, leading to the replication of latent EBV DNA. (11 refs)

78-4046 **Relationship Between Epstein-Barr Virus (EBV)-Production and the Loss of the EBV Receptor/Complement Receptor Complex in a Series of Sublines Derived from the Same Original Burkitt's Lymphoma.** (Eng) Klein, G. (Dept. Tumor Biology, Karolinska Institutet, S-10401, Stockholm 60, Sweden); Yefenov, E.; Falk, K.; Westman, A. *Int J Cancer* 21(5): 552-560; 1978.

Virus production, Epstein-Barr virus (EBV, P3HR-1 substrain) superinfectability, iododeoxyuridine (IUdR) inducibility, and expression of EBV and complement (C3) receptors were assessed in (1) two independently maintained Jijoye Burkitt's lymphoma lines; (2) the P3HR-1 clone that releases a nontransforming, cytopathic, and growth-inhibiting viral mutant; and (3) nonproducer P3HR-1 sublines. Both Jijoye lines were superinfectable and inducible, and they carried EBV and C3 receptors. The virus-producing P3HR-1 cells and their nonproducer sublines lacked EBV and C3 receptors and they could not be superinfected, but they were inducible with IUdR. Of two long-passaged nonproducer sublines that reexpressed EBV and C3 receptors, the one with the higher expression also manifested P3HR-1 viral superinfectability. These findings support the hypothesis that the EBV/C3 receptor negativity of P3HR-1 and its sublines is due to negative selection by the growth inhibitory, cytopathic P3HR-1 virus variant. The closely linked disappearance and reappearance of the EBV and C3 receptors suggests that they are identical or closely linked membrane constituents. (30 refs)

78-4047 **Recovery of Transforming EBV from Non-producer Cells after Superinfection with Non-transforming P3HR-1 EBV.** (Eng) Fresen, K. O. (Institut Virologie, Zentrum Hygiene, Universitat Freiburg, Hermann-Herder-Strasse 11, 7800 Freiburg, W. Germany); Cho, M. S.; zur Hausen, H. *Int J Cancer* 22(4): 378-383; 1978.

The viral progeny released after superinfection of nonproducer lymphoblastoid cell lines with Epstein-Barr virus (EBV) were characterized and compared with the input virus. Treatment of Raji (a Burkitt's lymphoma-derived cell line) or NC37 (derived from the peripheral blood of an EBV-seroreactive donor) cells with iododeoxyuridine (IUdR) or the tumor promoter 12-O-tetradecanoylphorbol-13-acetate resulted in induction of early antigen (EA) only. After superinfection by nontransforming P3HR-1 EBV, however, 5.5% of Raji cells and 6.0% of NC37 cells showed viral capsid antigen (VCA) synthesis. Viral particles were seen in the supernatants of these cells 5 days after superinfection. Infection of cord blood lymphocytes with supernatants from P3HR-1 EBV-superinfected Raji or NC37 cells led to transformation and resulted in the establishment of EB nuclear antigen-positive permanently growing lymphoblastoid cell lines. Infection of P3HR-1 EBV-converted BJA-B cells (an EBV-genome-negative human B-lymphoma cell line) led to EA and VCA induction and the release of nontransforming viral particles. When permanently growing lymphoblastoid cell lines established after infection of human cord blood lymphocytes with supernatants of P3HR-1-superinfected Raji or NC37 cell lines or with transforming B95/8-EBV were compared, only one cell line, NC37-RI, spontaneously expressed EA (8.5% of the cells) produced VCA (6%), and had transforming properties. These results suggest that viral genomes persisting within Raji and NC37 cells are defective and become complemented by the superinfecting P3HR-1 virus. (30 refs)

78-4048 **Epstein-Barr Virus in Somatic Cell Hybrids Between Mouse Cells and Human Nasopharyngeal Carcinoma Cells.** (Eng) Steplewski, Z. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA, 19104); Koprowski, H.; Andersson-Anvret, M.; Klein, G. *J Cell Physiol* 97(1): 1-8; 1978.

Somatic cell hybrids were produced between human nasopharyngeal carcinoma (NPC) cells and mouse cells to determine whether the expression of Epstein-Barr nuclear antigen (EBNA) in the hybrid cells is related to the retention of a specific human chromosome(s) derived from NPC patients. NPC cells were taken from biopsy specimens or from NPC tumors that had been maintained in nude mice. Three mouse cell lines were used: two deficient in thymidine kinase and one deficient in hypoxanthine phosphoribosyltransferase. Fusion of NPC and mouse cells resulted in 13 independent hybrid lines, 10 of which contained cells with EBNA-positive nuclei. In some clones derived from these lines, 90% of the EBNA-positive cells were maintained for > 6 mo; in others, the number of EBNA-positive cells decreased to 10%-20% during this time. Although human chromosomes 12 and 21 (or parts of them) appeared to be preferentially retained in the EBNA-positive hybrid cells, subcloning indicated that neither of these chromosomes segregated with EBNA. Thus, it was impossible to correlate the presence of EB virus (EBV) DNA and the expression of EBNA with a specific human chromosome(s). It is suggested that the production of EBNA is associated with a human chromosome fragment carrying EBV DNA sequences that cannot be detected by karyotype analysis or isozyme assays. (29 refs)

78-4049 **Studies on the Association of the Epstein-Barr Virus Genome and Human Chromosomes.** (Eng) Glaser, R. (Dept. Medical Microbiology, Ohio State Univ. Coll. Medicine, Columbus, OH, 43210); Nonoyama, M.; Hampar, B.; Croce, C. M. *J Cell Physiol* 96(1): 319-326; 1978.

Cloned and subcloned mouse fibroblast/Burkitt's lymphoma hybrid cells(Cl1D/HR-1) were used in attempts to identify a specific human chromosome(s) that is associated with the Epstein-Barr virus (EBV) genome. Ten clones of CL1D/Hr-1 cells were assayed for the spontaneous expression of early antigen (EA) and virus capsid antigen (VCA) by indirect immunofluorescence, but no EA- or VCA-positive cells were found. Attempts to induce EA and VCA with iododeoxyuridine were unsuccessful. Clone M44 and four of its subclones were assayed for EBV DNA and the presence of human chromosomes. Subclone 5 [EB nuclear antigen (EBNA)-negative] and subclone 4 (2%-3% EBNA-positive) had no detectable levels of EBV DNA. Clone M44 and subclones 2 and 24 ($\geq$ 90% EBNA-positive) contained 0.4-0.6 EBV genome equivalent/cell. Examination of Giemsa-banded metaphase chromosomes and isozyme analysis were used to determine the presence of human chromosomes. Five intact human chromosomes (7, 1, 12, 15, 17) were found in at least 10% of the M44 cells, but in none of the four subclone cells. A human marker chromosome derived from human chromsome 14 was present in all four subclones, and all four expressed human nucleoside phosphorylase activity. It is concluded that the EBV genome is not associated with any specific human chromosome in the cell line studied. (19 refs)

78-4050 **Reversible Inhibition by Phosphonoacetic Acid of Human B Lymphocyte Transformation by Epstein-Barr Virus.** (Eng) Thorley-Lawson, D. A. (Sidney Farber Cancer Inst, Harvard Medical Sch., Boston, MA, 02115); Strominger, J. L. *Virology* 86(2): 423-431; 1978.

Adult peripheral blood B lymphocytes, purified by immunoabsorbent chromatography, were used to study the kinetics of DNA synthesis due to Epstein-Barr virus (EBV) infection. The kinetics showed two phases: an early phase, lasting 3-4 days, the rate and absolute level of which depended on the multiplicity of infection, and a later phase, representing normal exponential growth, the level but not rate of which depended on multiplicity of infection. The time of induction of DNA synthesis (24 hr) agreed well with published times for the appearance of EBV nuclear antigen (EBNA). Absorption and penetration of the cells by EBV required 1-2 hr. The induction of DNA synthesis proceeded normally during the first phase in the presence of phosphonoacetic acid (PAA: 200 μg/ml). Thereafter, DNA synthesis remained at a plateau. Blast-transformed EBNA-positive cells could be isolated from these cultures. Removal of PAA at any time up to 28 days postinfection resulted in resumed proliferation and outgrowth in the presence of PAA, these cells expressed four phenotypic properties of transformation by EBV: (1) induction of DNA synthesis, (2) EBNA expression, (3) blast transformation, and (4) immortalization (survival for at least 28 days). However, they did not proliferate. It is, therefore, proposed that EBV infection in the presence of PAA may be termed abortive transformation. (21 refs)

78-4051 **Transformation of Lymphocytes by Herpesviruses: A Molecular Model for Transformation of Viral Genomes by the Host Restriction Enzymes and Recombination of Transforming Genes with the Chromosomal DNA.** (Eng) Becker, Y. (Lab. Molecular Virology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 226-227; 1978.

A model for the transformation of lymphocytes by herpesviruses is presented. A figure eight-shaped herpesvirus DNA molecule is cleaved at the X junction, resulting in a circular molecule that is incapable of selfreplication. However, if cleavage results in the liberation of fragments containing the S component of the viral DNA, these fragments can be incorporated into the host DNA. The host cell is induced to produce a restriction enzyme that cleaves the circular viral DNA, and transcription and replication occur. (9 refs)

78-4052 **Delay of Tumor Growth in Hamsters Treated with Lynestrenol and Effect of *Staphylococcus aureus* Cowan A.** (Eng) Wybran, J. (Dept. Immunology, Saint-Pierre Univ. Hosp., 322 rue Haute, 1000 Brussels, Belgium); Thiry, L. *J Natl Cancer Inst* 61(1): 173-176; 1978.

The influence of lynestrenol, a synthetic progesteronelike substance, and of *Staphylococcus aureus* Cowan A on the growth of tumors induced by malignant cells transformed by herpes simplex virus type 1 (HSV-1) was studied in Syrian golden hamsters. The hamsters received ip injections of 1, 0.1, or 0.01 mg/kg lynestrenol and/or 0.1 ml of a 10% suspension of killed *S. aureus* from the day of birth. Some hamsters were inoculated sc 1 day after the start of therapy with 2 x 10^5 viable transformed cells and some were inoculated with 2 x 10^6 viable cells when they were 6 wk old. In most instances, a dose of 1 mg/kg lynestrenol delayed tumor growth for 1 wk at day 18. *S. aureus* alone did not delay tumor appearance. Survivals were best among animals receiving lynestrenol + *S. aureus*. Survivors showed an increased resistance to challenge with infected cells. Hamsters previously given injections of 10^7 killed transformed cells and then challenged with 2 x 10^5 viable cells showed a facilitation of tumor growth. This facilitation was inhibited by lynestrenol alone or in combination with *S. aureus*. It is suggested that these effects are related to an immunopotentiating property of lynestrenol. (11 refs)

78-4053 **The Transfer and Stable Integration of the HSV Thymidine Kinase Gene into Mouse Cells.** (Eng) Pellicer, A. (Inst. Cancer Res., Columbia Univ., 701 W. 168th St., New York, NY, 10032); Wigler, M.; Axel, R.; Silverstein, S. *Cell* 14(1): 133-141; 1978.

Treatment of mutant cells (Ltk-) deficient in thymidine kinase with Bam I reduction endonuclease-cleaved herpes simplex virus type 1 (HSV-1) DNA results in the appearance of numerous surviving colonies with a stable tk+ phenotype. Through a series of electrophoretic fractionations association with transfection assays, a 3.4 kilobar fragment was isolated that contained the thymidine kinase gene (*tk*) and that transfected *tk* activity to Ltk- cells. The distribution of *tk* sequences in the DNA of several transformed clones following stable gene transfer was examined in a series of complementary experiments involving reassociation kinetics in soln and annealings with *tk* DNA to restriction-cleaved cellular DNA following electrophoresis and transfer to filters. In all clones examined, the *tk* gene was present in all cells at a frequency of one copy per chromosomal complement. The *tk* gene was integrated stably in the DNA of the transformants, but integration was not site-specific as it occurred at different loci in the DNA of all transformants. The existence of a single active *tk* gene in tk+ transformants will facilitate analysis of the sequence organization of tk- mutant cells and provide a useful model system for studies of the transfer of cellular genes. (16 refs)

78-4054 **Oncogenic Properties of Herpesviruses.** (Eng) Munk, K. (Institut fur Virusforschung, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, 6900 Heidelberg, W. Germany); Flugel, R. M.; Braun, R.; Darai, G. *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 22-29; 1978.

The oncogenicity of the herpes simplex virus (HSV) strains HSV-1 Thea, HSV-2 Muller, and HSV-2-HG-52 and of cytomegalovirus (CMV) were investigated. When rat embryo fibroblasts (REF) were infected with HSV, transformed cells had a two- to threefold higher plating efficiency than normal cells. New cell colonies showed two different morphologies, epitheliallike or spindlelike. Clones were isolated from these colonies and established in tissue culture. The modal chromosome number of the transformed cells was >42, the modal number of primary rat cells. The spindlelike transformed cells grew in soft agar, but the epitheliallike cells did not. In newborn Sprague-Dawley rats, the transformed cells with the spindlelike morphology were highly tumorigenic, producing spindle cell sarcomas that grew rapidly and metastasized to the lung. Immunofluorescence revealed the presence of HSV antigens in the cytoplasm of the spindlelike cells but not in that of the epitheliallike cells. The spindlelike cells were resistant to superinfection with HSV types 1 and 2, and the superinfectability of the epitheliallike cells was reduced considerably, compared with REF cells. Further studies with human CMV demonstrated infection of hamster embryo fibroblasts, and persistent infection of human embryo lung fibroblasts (nonpermissive and permissive cell systems, respectively). Human and tree shrew cells were also transformed by CMV; both cell types exhibited CMV-specific immunofluorescence and were resistant to superinfection with CMV. (42 refs)

78-4055 **Latent Herpes Cervicitis and Mixed Cervical Carcinoma.** (Eng) Lauchlan, S. C. (Dept. Pathology, Women and Infants Hosp. Rhode Island, 50 Maude St., Providence, RI, 02908). *Am J Obstet Gynecol* 131(1): 109-110; 1978.

A 25-yr-old woman with an adenosquamous carcinoma of the cervix was found to have herpesviruslike intranuclear inclusions in several paracervical neurons. Although positive virologic identification of the inclusions was not made, their appearance was consistent with that of herpesvirus hominis, probably type 2. (2 refs)

78-4056 **Conditions Required for Induction of Murine p30 by Herpes Simplex Virus.** (Eng) Isom, H. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA, 17033); Colberg, A.; Reed, C.; Rapp, F. *Int J Cancer* 22(1): 22-27; 1978.

An attempt was made to confirm previous findings that infection of NIH Swiss mouse cells (N cellA cell10) with herpes simplex virus type 2 (HSV-2), strain 333, induces the expression of murine leukemia virus (MuLV) p30 antigen, as measured by indirect immunofluorescence (IIF). An attempt was also made to quantitate the IIF results by competition radioimmunoassay (RIA) and to determine the conditions of virus infection required for p30 induction. Uninfected N clA cl10 cells contain 1.2-2.5 nanograms of p30/mg protein; this amount of antigen could be detected by RIA, but not by IIF. Induction of p30 by HSV-2 was multiplicity-dependent, and it was optimal 8 hr postinfection when cells at 50%-70% confluence were infected at a multiplicity of infection (MOI) of 5-8 plaque-forming units/cell. At an MOI of 2.5, 70%-80% of the cells expressed HSV antigens, but none of the cells expressed p30; at an MOI of 5.0, 70%-80% of the cells expressed HSV antigens, and 55% of the cells expressed p30. Inhibition of virus DNA synthesis alone or both virus and cell DNA synthesis had no effect on p30 induction. Attempts to quantitate the IIF results by RIA were not successful: the HSV-2-induced increase in p30 was not detectable by RIA. It is likely that HSV-2 infection causes the p30 to become sufficiently labile so that the protein is degraded or altered during the preparation of competing antigen and it cannot be measured by RIA. (12 refs)

78-4057 The Phosphorylation of Histones in Herpes Virus Infected Vervet-Monkey Kidney Cells (CV-1). (Eng) Letnansky, K. (Inst. Cancer Res., Univ. Vienna, Vienna, Austria); Dostal, V.; Reiss-Gutfreund, R. J. *Oesterr Z Onkol* 4(2-3): 39-41; 1977.

The phosphorylation of histones in African green monkey kidney cells (CV-1) infected with herpes simplex virus type 2 (HSV-2, strain MS, 5 plaque-forming units/cell) was investigated. The incorporation of phosphate into histone fractions was diminished during the early periods after virus infection. This result was consistent with the observation that DNA replication is repressed during this period. However, 14-15 hr postinfection, high amounts of phosphate (twice control values) were incorporated into histones of the F1 group, fraction F2a1, and, particularly, fraction F2a2. It is not known whether these histones were of cellular or viral origin. At the same time, large amounts of DNA were produced; as demonstrated by separation experiments in cesium chloride gradients, this DNA was primarily of viral origin. It is suggested that the stimulated phosphorylation of these histone fractions is a prerequisite for the enhanced synthesis of DNA and, possibly, of RNA. (13 refs)

78-4058 Alkaline DNase Activity in Cells Infected with a Temperature-sensitive Mutant of Herpes Simplex Virus Type 2. (Eng) Francke, B. (Tumor Virology Lab., Salk Inst. Biological Studies, San Diego, CA, 92112); Moss, H.; Timbury, M. C.; Hay, J. *J Virol* 26(2): 209-213; 1978.

Experiments were conducted to determine whether the temperature-sensitive mutant *ts*13 of herpes simplex virus type 2 (HSV-2) represents the structural gene for viral alkaline nuclease. Baby hamster kidney cells (BHK-21) infected with *ts*13 at a nonpermissive temperature lacked alkaline nuclease activity, which was induced by the mutant at a permissive temperature and by wild-type virus at either temperature. For *ts*13, enzyme activity could be induced by a temperature shift to permissive conditions, but not in the presence of cycloheximide. After a shift from permissive to nonpermissive conditions in the presence of cycloheximide, the activity was stable in wild-type, but not in mutant-infected, cells. After extensive purification, the wild-type nuclease was four times more heat stable in the presence of substrate than was the mutant enzyme. These results indicate that two temperature-sensitive functions can be defined in *ts*13, one related to the virus particle and likely to be a structural polypeptide and the other the viral nuclease activity. (9 refs)

See also:
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78-3675, 78-3676, 78-3677, 78-3678.
*(Chem.): 78-3745, 78-3831.
*(Phys.): 78-3914, 78-3936.
*(Immun.): 78-4060, 78-4061, 78-4062, 78-4063
78-4065, 78-4066, 78-4074, 78-4076
78-4084.
*(Path.): 78-4091, 78-4092, 78-4107, 78-4133.
*(Epid.-Biom.): 78-4153, 78-4170.

78-4059 **Antitumor Immunity after Surgical Removal of 3-Methylcholanthrene-induced Murine Tumors: Correlation Between Resistance and Delayed Hypersensitivity Reaction.** (Eng) Abe, S. (Faculty Pharmaceutical Sciences, Univ. Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113, Japan); Yamazaki, M.; Mizuno, D. *Gann* 69(3): 323-329; 1978.

The relationship between tumor resistance and the antitumor delayed hypersensitivity reaction (ATDHR) after tumor resection was studied with the use of the syngeneic 3-methylcholanthrene-induced murine tumor, MCA-DDD. MCA-DDD was inoculated sc into the tail of DDD mice and, 4 wk later, the tumor-bearing tail was resected. The tumor resistance of the mice was then determined from the diameters of the tumor that developed 14 days after sc challenge of the mice with tumor cells in the flank. The mice showed a specific resistance to the tumor until 30 days after tumor resection. In parallel with tests on tumor resistance, the ATDHR of the mice after tumor resection was examined by the footpad test. The tumor antigen fraction was a cell-free extract of sonicated MCA-DDD tumor cells. The ATDHR of the mice appeared soon after tumor resection, reached a max on day 7, and then decreased slowly until day 29. Mice that acquired tumor resistance showed a rapid increase in ATDHR after challenge with fresh tumor cells. The ATDHR was high on day 4 after challenge, and its level correlated well with the tumor resistance of the mice. (14 refs)

78-4060 **Modulation of Virus-induced Tumor Antigens (MLr) on Murine Leukemia Cells.** (Eng) Feltkamp, C. A. (Div. Cell Biology, Antoni van Leeuwenhoekhuis, Netherlands Cancer Inst., Sarphatistraat 108, Amsterdam, Netherlands); Wilschut, I. J.; Van Blitterswijk, W. J.; Hilgers, J.; Sonnenberg, A. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 345-348; 1978.

The antibody-induced modulation of tumor antigens was studied in ascitic murine leukemia cells (GRSL), which possess mammary tumor virus (MTV)-induced cell surface antigens. Incubation of GRSL cells with anti-MTV serum at 37 C resulted in the loss of MTV antigens, as measured by the complement-dependent cytotoxicity assay. Within 15 min, the percentage of dead cells was significantly lower than that in control cultures. This early phase of modulation was paralleled by the patch formation of immune complexes. Max modulation (after a 120-min incubation) was accompanied by the shedding of part of the patches from the cell surface. The loss of antigenicity was specific for MTV cell-surface antigens, and it was not due to endocytosis or capping of immune complexes. (3 refs)

78-4061 **Involvement of the Immune System in the Spontaneous Regression of Friend Virus Induced Erythroleukemia.** (Eng) Hines, D. L. (Michigan Cancer Foundation, Detroit, MI, 48201); Dietz, M.; Marcelletti, J.; Rich, M. A.; Furmanski, P. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 233; 1978.

Studies of the role of the immune system in the spontaneous regression of erythroleukemia induced by a variant of Friend virus, RFV, suggest that the immune response to RFV-specific antigens is a major component of regression and that it is involved in maintaining the regressed state. Redevelopment of leukemia, which occurs in 50%-70% of regressed mice, may be due to loss of an effective immune response with time. RFV or RFV-leukemic cells may be antigenically different from nonregressing strains of Friend virus. (no refs)

78-4062 **Epstein-Barr Virus Infections in the X-Linked Recessive Lymphoproliferative Syndrome.** (Eng) Purtilo, D. T. (Dept. Pathology, Univ. Massachusetts Medical Sch., 55 Lake Ave., North, Worcester, MA, 01505); Bhawan, J.; Hutt, L. M.; DeNicola, L.; Szymanski, I.; Yang, J. P.; Boto, W.; Maier, R.; Thorley-Lawson, D. *Lancet* 1(8068): 798-801; 1978.

The hypothesis that immunodeficiency to Epstein-Barr virus (EBV) can lead to variable phenotypic expressions of the X-linked recessive lymphoproliferative syndrome was investigated in three brothers with the syndrome. All three had the same defective lymphoproliferative-control locus on the X chromosome but showed different phenotypic expression: brother A (16-yr-old) had hypogammaglobulinemia (HGG), fatal infectious mononucleosis (IM), and immunoblastic sarcoma without EBV antibodies; brother B (15-yr-old) had HGG and subclinical EBV infection with EBV antibodies; after treatment, he has been symptom-free for 8 mo since EBV infection; brother C (18-yr-old) had histiocytic lymphoma; he survives recurrence-free 6 yr postresection. HGG and measles pneumonitis had preceded EBV infection in brothers A and B. The diverse phenotypic expressions probably resulted from the varied immune response to EBV. However, the difference could also be explained in part by environmental factors: C was separated from his two brothers since the age of 3. A and B had shared the same bedroom and, probably, the same microorganisms. Xg blood-grouping of the RBC of A and B revealed that A was (a+), but B was (a-). Both T-cell activity and the capacity to form EBV antibodies were defective in A. The apparent recombination of the X chromosome of B, however, equipped him with immune defenses that prevented IM from appearing. T-cell proliferation in a lymph node of B vs T-cell depletion with B-cell replacement in the lymphoid tissues of A supports this view. In conclusion, EBV can produce diverse lymphoproliferative disorders and can be potentially oncogenic in immunodeficient patients. (24 refs)

78-4063 **The Molecular Nature of the Gross Cell-Surface Antigen (GCSA).** (Eng) Snyder, H. W. (Memorial Sloan-Kettering Cancer Center, New York, NY); Stockert, E.; Fleissner, E. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 353-354; 1977.

Gross virus-induced leukemia E(male)G2 cells were examined for the presence of surface molecules carrying Gross cell-surface antigen (GCSA). The leukemia cells expressed glycosylated murine leukemia virus core (gag) polyproteins on their surface. These polyproteins bore the antigenic determinants of GCSA. (6 refs)

78-4064 **Cellular Immunity to Solubilized Tumor Antigens of a Methylcholanthrene-induced Sarcoma with a Migration Inhibition Assay.** (Eng) Padarathsingh, M. L. (Dept. Immunology, Biomedical Res. Div., Litton Bionetics, Inc., Kensington, MD, 20795); Dean, J. H.; McCoy, J. L.; Lewis, D. D.; Northing, J. W.; Natori, T.; Law, L. W. *J Immunol* 120(6): 1981-1985; 1978.

BALB/c mice immunized with Nonidet P-40 (NP-40) crude solubilized (CS) extracts of a syngeneic methylcholanthrene-induced BALB/c sarcoma (Meth A) were challenged with viable Meth A cells to determine the ability of the solubilized preparations to induce transplantation rejection. Animals resisting such challenge were then used in agarose microdroplet macrophage migration inhibition (MMI) and tumor cell neutralization (Winn) assays to evaluate the antigenic specificity of these CS extracts. Spleen cells from animals that rejected Meth A after immunization with the NP-40-solubilized preparations effectively neutralized the tumor-producing capacity of Meth A tumor cells, as determined in Winn assays. The MMI assays were quite sensitive; migration inhibition of peritoneal exudate (PE) cells from immunized mice was detected with extract concentrations as low as picogram quantities. Meth A expressed no antigenic cross-reactivity with similarly prepared extracts of an unrelated simian virus 40-induced sarcoma (mKSA) or with a mineral oil-induced plasmacytoma (ADJ-PC5) of BALB/c mice. Inhibition of PE cell migration was mediated by culture supernatants (presumably migration inhibition factor) generated from a mixture of immune spleen cells and mitomycin C-treated Meth A cells, as assayed in an indirect MMI test. Although extracts from strongly antigenic murine tumor systems may permit strong MMI reactivity, this observation may not necessarily hold for human systems. (30 refs)

78-4065 **Suppressed Murine Mammary Tumor Virus (MuMTV) Expression in RIII Female Mice Treated Neonatally with Goat Antiserum to MuMTV.** (Eng) Frensdorff, A. (Dept. Microbiology, Faculty Life Science, Dr. George S. Wise Centre Life Sciences, Tel Aviv Univ., Tel Aviv, Israel); Wald, A. *J Natl Cancer Inst* 61(2): 437-439; 1978.

Newborn inbred R3/Imr mice were passively immunized with goat antiserum to murine mammary tumor virus (α MuMTV) according to two schedules, and the influence of α-MuMTV on MuMTV expression was studied. Schedule A mice received 10 im injections of whole goat No. 2 antiserum to MuMTV (α-MuMTV$_2$) or normal goat serum (NGS) in increasing dosage to a total of 3.75 ml serum/mouse over 21 days. Schedule B mice received 10 im injections of the globulin fraction of α-MuMTV$_2$ or NGS, each in amounts equivalent to 500 μl of original serum to a total of 5.0 ml serum equivalent/mouse over 21 days. At 1 mo of age, females were mated with untreated R$_3$ males, and milk samples were collected from mothers on days 7-10 postpartum upon the 3rd, 6th and 9th pregnancies. The milk was tested for MuMTV antigen by double diffusion in agar gels and by radial immunodiffusion. Milk from mice given NGS and from untreated mice contained MuMTV antigens at the third lactation, but a considerable part of third lactation milk from all immunized mice was negative. In addition, 4/12 mice born at third parity to mothers on Schedule A and 6/12 born to mothers on Schedule B were negative with respect to MuMTV antigens. At the fourth parity, all mice on Schedule A (12) were positive for MuMTV antigens, but 4/8 on Schedule B were still negative. The 14 females that remained antigen-negative at the sixth lactation bore no tumors, even though 10 were $>$9.5 mo old, whereas spontaneous mammary tumors had arisen in 100% of corresponding controls, between 4.5 and 9 mo of age. Early administration of goat antiserum to MuMTV to newborn R3 mice apparently leads to delay and even suppression of MuMTV expression. (12 refs)

78-4066 **Suppression of Leukemogenesis in Hairless Mice by Anti-Type C Viral Immune Gamma Globulins.** (Eng) Pottathil, R. (Jackson Lab., Bar Harbor, ME, 04609); Meier, H.; Huebner, R. J. *Naturwissenschaften* 65(8): 443-444; 1978.

The suppression of leukemogenesis by passive neonatal immunization against ecotropic murine leukemia virus (MuLV) was investigated in the hairless mouse, HRS/J--*hr/hr*. Haired and hairless littermates were given a total of 0.45 ml anti-radiation leukemia virus goat IgG. This preparation had a neutralizing antibody titer of 1:800 to 1:1,600. Each mouse received, sc, 0.05 ml on days 0, 4, and 7; 0.1 ml on day 10; and 0.2 ml on day 14. MuLV titers were assayed at 1, 3, and 6 mo, and the mice were observed until moribund, at which time they were killed and evaluated histopathologically. Immunized haired and hairless mice showed a significant reduction in MuLV titers compared with control mice. IgG-treated hairless mice had a very low rate of leukemia occurrence (2/14 at 16 mo), compared with untreated mutants (8/13 at 16 mo). There was no appreciable difference in leukemia incidence between treated and untreated normal (haired) mice. The reduction in leukemia incidence of treated hairless mice in conjunction with lowered ecotropic virus titers tends to verify the existence of a close relationship between high endogenous ecotropic MuLV expression and leukemia susceptibility. These results confirm the applicability of antibody therapy to virus-associated malignancies. (17 refs)

78-4067 **Paraproteinemia in Normal Family Members of Eight Cases with Primary Intestinal Lymphomas in Iraq.** (Eng) Alsabti, E. A. (Philharmonic Residence, Flat N4, Liverpool L8 7SD, England). *Oncology* 35(2): 68-72; 1978.

The immunoglobulin patterns of six men and two women (8-28 yr old) with primary intestinal lymphoma were investigated. Immunoelectrophoresis of the serum proteins from all patients showed the IgA heavy chain disease pattern. The clinical, laboratory, radiological, and histopathological data for these patients were suggestive of the malabsorption syndrome. Immunoelectrophoresis of serum from the 129 normal family members of the 8 patients revealed the IgA heavy chain pattern in 23 persons from four of these families. This finding suggests that a hereditary factor may be involved in the production of alpha-chain diseases and that these 23 persons should be closely monitored for the development of intestinal lymphoma. (30 refs)

78-4068 **A Possible Role of Prostaglandins in the Inhibition of Natural and Antibody-dependent Cell-mediated Cytotoxicity Against Tumor Cells.** (Eng) Droller, M. J. (Dept. Immunology, Wenner-Gren Inst., Univ. Stockholm, Stockholm, Sweden); Schneider, M. U.; Perlmann, P. *Cell Immunol* 39(1): 165-177; 1978.

The effects of prostaglandins E_1 and E_2 on natural and antibody-dependent lymphocyte cytotoxicity against the T_{24} and HCV_{29} tumor lines were examined to determine whether prostaglandin production by the tumor cells could alter the cellular immune response. The T_{24} line was derived from a transitional cell tumor of the bladder; line HCV_{29}, obtained from normal bladder epithelium of a patient successfully treated for bladder carcinoma by radiotherapy, had become transformed upon passaging in tissue culture. The target cell lines were labeled with ^{51}Cr and incubated at 37 C for various times with lymphocytes prepared from the venous blood of normal donors. Antiserum to T_{24} and varying concentrations of prostaglandin E_1 or E_2 were added to the samples prior to incubation. In some experiments, lymphocytes or labeled target cells were preincubated with prostaglandins and then washed prior to their addition to the assay tubes. Cytotoxicity was determined by measuring the release of ^{51}Cr from the target cells after incubation. Both prostaglandins inhibited natural and antibody-dependent lymphocyte cytotoxicity against the target cells. The effect on lymphocytes appeared to be direct, and it was amplified by the presence of theophylline in the medium. Inhibition could be effected early in lymphocyte/target cell interaction, and only a short exposure of lymphocytes to prostaglandins was required for the effect to be manifested. The production of prostaglandins by tumor cells may constitute a mechanism by which the tumor cells subvert the effect of a cellular immune response directed against them. (26 refs)

78-4069 **Differentiation Linked Expression of p28,33 (Ia-like) Structures on Human Leukaemic Cells.** (Eng) Janossy, G. (Membrane Immunology Lab., Imperial Cancer Res. Fund, London, England); Goldstone, A. H.; Capellaro, D.; Greaves, M. F.; Kulenkampff, J.; Pippard, M.; Welsh, K. *Br J Haematol* 37(3): 391-402; 1977.

A differentiation-linked expression of Ia-like (B-cell-associated) structures was detected by heterologous antisera against p28,33 antigen on human leukemia cells. A double fluorochrome immunofluorescence test was used to distinguish Ia+ (p28,33+) leukemia cells from B lymphocytes, and leukemia cells with varying intensities of anti-p28,33 staining were separated. p28,33 was very strongly expressed on undifferentiated blast cells from Philadelphia chromosome (Ph^1)-positive lymphoid blast crisis and non-T, non-B acute lymphoid leukemia. In Ph^1-positive myeloid blast crisis and in acute myeloid leukemia, the p28,33 antigen was only moderately strongly expressed on myeloblasts and virtually absent on promyelocytes. The p28,33 antigen was also absent on the relatively more mature cells during the chronic phase of chronic myeloid leukemia. Nonleukemic samples (bone marrow samples from fetuses and from patients with a severe megaloblastic anemia or myeloid hyperplasia) also contained p28,33-positive immature blast cells. These observations suggest that Ia-like antigens on leukemia cells reflect the origin of the leukemias from hemopoietic stem cells, which are themselves Ia-positive. It is also suggested that Ia-like membrane structures are stem cell markers that may play an important role in the immunological responses of B cells and in the regulation of early hemopoietic cell differentiation. (29 refs)

78-4070 **Cell Surface Immunoglobulins of Bovine Leukemic Cells.** (Eng) Trainin, Z. (Dept. Immunology, Kimron Veterinary Inst., Bet Dagan, Israel); Ungar-Waron, H.; Meirom, R.; Glueckmann, A.; Gavish, M.; Katzav, S.; Haimovich, J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 253-254; 1978.

The cell surface immunoglobulins of peripheral blood lymphocytes (PBL) from normal and leukemic cattle were compared. IgM was present on PBL from 2/4 leukemic cows: 60%-80% of the PBL exhibited IgM, compared with 20% of normal cow PBL. Electrophoresis indicated that the μ-chain from the IgM of leukemic PBL migrated slower than that from the IgM of normal PBL. These μ-chains had mol wts of 95,000 and 85,000, respectively. Since lymphoid tumor cells do not exhibit membrane IgM, it is suggested that two clones of lymphoid cells are involved in bovine leukemia. (6 refs)

78-4071 **Kinetic Studies of Surface Markers on the Pathological Cells of Hairy Cell Leukaemia (HCL) and Chronic Lymphocytic Leukaemia (CLL).** (Eng) Burns, G. F. (Dept. Haematological Medicine, Univ. Cambridge, Cambridge, England); Cawley, J. C.; Barker, C. R.; Nash, A. A.; Rees, J. K.; Worman, G. P.; Hayhoe, F. G. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 251-252; 1978.

The expression of surface markers was studied on pathological B cells from six patients with hairy cell leukemia (HCL) and four patients with chronic lymphocytic leukemia (CLL). HCL and CLL cells each possessed distinct receptors for IgG and IgM. The IgM receptor was spontaneously shed and reexpressed in a cyclical fashion, with a peak-to-peak reexpression time of 6 hr in HCl and 12 hr in CLL. Surface membrane immunoglobulin markers were thought to be removed from the cell membrane as soon as they were expressed. (4 refs)

78-4072 **Pre-B-Cell Leukemia. A New Phenotype of Childhood Lymphoblastic Leukemia.** (Eng) Vogler, L. B. (224 Tumor Inst., Univ. Alabama in Birmingham, Birmingham, AL, 35294); Crist, W. M.; Bockman, D. E.; Pearl, E. R.; Lawton, A. R.; Cooper, M. D. *N Engl J Med* 298(16): 872-878; 1978.

Leukemic cells from children with acute lymphoblastic leukemia were examined for lymphocyte markers characteristically acquired at different stages of development. Large lymphoid cells containing small amounts of cytoplasmic IgM (cIgM) but undetectable surface immunoglobulin (sIg) have been recognized recently as precursors of B lymphocytes. In 4/22 consecutive patients (2.5-15 yr old), leukemic cells had the cIgM+.sIg-phenotype of pre-B cells. The cells of these patients shared common acute lymphoblastic leukemia antigens and B-cell alloantigens, but differed in expression of several developmental features characteristic of the B-cell lines. Pre-B-cell leukemias were readily responsive to chemotherapy. It is concluded that a distinct subpopulation of previously unclassified leukemias reflects oncogenic transformation at the earliest recognizable stage in differentiation along the B-cell axis. (38 refs)

78-4073 **Genetically Determined Immunodeficiency Diseases (GDID) and Malignancy: Report from the Immunodeficiency-Cancer Registry.** (Eng) Spector, B. D. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN, 55455); Perry, G. S.; Kersey, J. H. *Clin Immunol Immunopathol* 11(1): 12-29; 1978.

Cases reported to the Immunodeficiency-Cancer Registry (ICR) were updated, resulting in new data related to the association between immunodeficiency and malignancy. There were 267 patients with both genetically determined immunodeficiency diseases (GDID) and malignancies in the ICR. The 267 subjects fell into 13 different GDID categories. The predominant tumor histology was non-Hodgkin's lymphoma and lymphoid leukemia (158/267). There were an additional 42 mesenchymal tumors that arose in cells other than the lymphocytic-reticuloendothelial series, including Hodgkin's disease (22), and myelogenous leukemia (9). Of the 65 epithelial tumors, 11 affected the nervous system only. In the remaining 54 cases, the major sites were stomach (21 cancers), lung (6), genitals (6), and skin (4). Among the 11 cases in which the results of surface marker studies of malignant blast cells were reported to ICR, 7 had phenotypes that were B-like in origin, 2 were T-like, and 2 were non-B, non-T. The data revealed the following malignancy patterns: (1) if a child with a GDID develops a malignancy, it will most likely be a non-Hodgkin's lymphoma, whereas adults will develop non-Hodgkin's lymphomas and carcinomas in about equal proportions; (2) in most GDID's, the general malignancy type is lymphoreticular; and (3) the most common single histologic type of non-Hodgkin's lymphoma is histiocytic lymphoma. Although the case material in the registry is valid, the methods of reporting the cases may have resulted in certain biases, such as the over- or underreporting of tumors occurring in certain GDID's. (64 refs)

78-4074 **Human Malignant Lymphomas In Vitro.** (Eng) Sundstrom, C. (Wallenberg Lab., P.O. Box 562, S-751 22 Uppsala, Sweden); Nilsson, K. *Acta Pathol Microbiol Scand (A)* 86(2): 173-184; 1978.

A series of 55 biopsies from different types of malignant lymphomas were characterized in short-term culture experiments and during prolonged growth in vitro. Most of the lymphocytic (LC) lymphomas and half of the histiocytic (HC) lymphomas expressed surface immunoglobulin, either in monoclonal or polyclonal form, indicating B-lymphocyte derivation. No lysozyme production was noted in either type of lymphoma, giving further support to the notion that HC lymphomas are not truly HC. Production of β_2-microglobulin was higher in HC than in LC lymphoma and Hodgkin's disease, but it did not differ significantly from the production observed in nonneoplastic lymph node disorders. Incorporation of ^{3}H-thymidine varied greatly within each category of lymphoma; the highest mean labeling index was noted in HC lymphoma, possibly reflecting the generally more malignant course in these cases. Epstein-Barr virus-associated nuclear antigen was observed in one case of Hodgkin's disease. Attempts to establish permanent tumor cell lines were successful only from two explants of LC lymphoma and one pleural effusion from a HC lymphoma. The two cell lines derived from LC lymphomas both exhibited B-lymphocyte characteristics. The HC lymphoma line lacked lymphocyte markers, produced lysozyme, and was found to be rich in cytoplasmic esterases. These features are consistent with a true HC derivation of this line. Lymphoblastoid cell lines representing nonneoplastic EBV-carrying lymphocytes contaminating the biopsies were derived from 19 biopsies, 10 of which were from cases of Hodgkin's disease. The tumor lines were all EBV-genome-negative. (83 refs)

78-4075 **The Influence of Embryonal Bursectomy on Benzpyrene-induced Sarcoma of the Chicken.** (Eng) Niedorf, H. R. (Inst. Pathology, Univ. Munster, Westring 17, D-4400 Munster, W. Germany); Lusznat, A.; Hultsch, E.; Grundmann, E. *Z Krebsforsch* 91(3): 323-334; 1978.

The effect of embryonal bursectomy on the incidence, growth, and metastatic spread of benzo(a)pyrene (BP)-induced sarcomas was studied in specifically virus-free chicks. There were two groups of 160 chicks: Group B was bursectomized on day 3 of embryonic life by immersion of the eggs into testosterone propionate. Group C (controls) was not bursectomized. All chicks were inoculated with BP (10 mg im) 2 and 6 wk posthatching. At 18 wk posthatching, 78 Group B and 129 Group C chicks had survived, and tumors could be palpated in 4 of the former, 6 of the latter. From wk 20 onward, however, Group B chicks showed a lower tumor incidence than Group C. At 24 wk, tumor incidence was 32.1% and 49.6%, respectively ($p \leq 0.012$). Light and electron microscopy identified the tumors as malignant polymorphocellular rhabdomyosarcomas. At 27 wks, tumor incidence in Group B chicks (58.9%) was closer to that of Group C (65.1%), which shows that bursectomy prolonged tumor latency. Metastases were found in 10/46 Group B tumor-bearers and in 35/84 Group C tumor-bearers. They were most common in the liver (7 Group B chicks, 21 Group C) and lungs (5 Group B, 19 Group C). Many animals had metastases in more than one site. These findings are an illustration of the disturbed equilibrium between T- and B-cell responses. (50 refs)

78-4076 **Immunosuppressive Activity of Antibody Directed Against Endogenous C-type Virus Interferes with Early Events of the Immune Response.** (Eng) Schumann, G. (Res. Dept., Pharmaceuticals Div., CIBA-GEIGY Limited, R-1056.4.13, CH-4002 Basel, Switzerland); Moroni, C. *J Immunol* 120(6): 1913-1916; 1978.

The physiologic role of endogenous viruses in the immune system was examined by testing the effect of antiserum directed against BALB/c endogenous xenotropic C-type virus on the humoral immune response of mice. Both in vivo and in vitro, this serum suppressed the response to sheep RBC, an effect that could be absorbed out by purified BALB/c xenotropic C-type virus or Friend leukemia virus, but not by Rous sarcoma virus. The serum produced its max effect when administered with or prior to the antigen, but not 24 hr later. This suggests that the antibody acts on an early event in the immune response. The critical viral antigen was expressed before the spleen cells were experimentally stimulated by the antigen. The same immunosuppressive effect was observed in a variety of mouse strains, including high-leukemia-incidence AKR mice and virus-free 129/J mice; this indicated that the immunosuppressive effect is independent of endogenous virus. The binding of antibody to viral antigen on the cell membrane may lead to steric hindrance of immunologic receptor structures, or the viral antigen itself may play a receptorlike role. If endogenous viruses are actively involved in the triggering of lymphocytes to form a clone of dividing cells, it may be possible to find a link between this system and leukemias in which C-type viruses are known to play a role. (19 refs)

78-4077 **Incidence of Spontaneous Ileocecal Immunocytomas in Hybrids of LOU/C Rats and Rat Strains with Low Spontaneous Tumor Incidence: Brief Communication.** (Eng) Becker, A. (Experimental Immunology Unit, Univ. Louvain, Box 3056, 30 Clos Chapelle-aux-Champs, 1200 Brussels, Belgium); Bazin, H. *J Natl Cancer Inst* 60(6): 1505-1508; 1978.

The incidence of spontaneous ileocecal immunocytomas in LOU/C, LOU/M, AxC, and Okamoto (OKA) rats and the F_1 hybrids of LOU/C x (1) AxC, (2) AUG, and (3) OKA rats was determined. Male and female tumor incidence in the respective strains was 31.6% and 16.1%, 0.7% and 2.1%, 4.2% and 1.9%, 0% and 0%, 18.5% and 14.1%, 16.7% and 7.8%, and 0% and 0%. In LOU/C rats, the susceptibility to immunocytomas was under genetic control: these rats contained a dominant locus or loci of susceptibility that could induce immunocytoma in inbred AUG and inbred AxC rats. However, inbred OKA rats had at least one dominant locus of resistance that did not exist in the other strains. The class distribution of monoclonal immunoglobulins originating in male and female rats and in the various hybrids did not differ significantly. Most tumors that originated appeared to develop in the ileocecal lymph node. A total of 710/720 spontaneous tumors that were transplanted into histocompatible rats grew during the first passage. Almost all tumors that were successfully transplanted continued to be transplantable in histocompatible hosts. (23 refs)

78-4078 **An Athymic Nude Mutation in the Rat.** (Eng) Festing, M. F. (MRC Lab. Animals Centre, Woodmansterne Road, Carshalton, Surrey, England); May, D.; Connors, T. A.; Lovell, D.; Sparrow, S. *Nature* 274(5669): 365-366; 1978.

The morphology and biological behavior of homozygous nude rats that originated from an athymic hairless mutation in 1953 are reported. The thymus is replaced by brown fat tissue, although there is a small and highly abnormal thymus rudiment. Nude rats grow to only about 60% to 80% of the size of their heterozygous littermates, and the preweaning mortality of nude rats is higher than that of their heterozygous littermates. Homozygous nude rats of both sexes are fertile. Nude rats are able to accept xenografts of skin and tumors. The output of thoracic duct lymphocytes 24 hr after cannulation is reduced to 15% to 25% of control values, and T-cell function is depressed or absent. Nude rats are killed within 10-13 days by an infection of the larvae of the tapeworm *Taenia crassiceps* which does not normally kill immunologically competent rats. Small-scale attempts to grow tumors in these rats have had mixed success. A plasma cell tumor specific to BALB/c mice and a human colon tumor grew for a short time in two rats and then regressed. In contrast, the TLX5 lymphoma grew rapidly in 4/6 nude rats, although it largely remained localized. The nude rat is in many ways similar to the nude mouse and is of potential usefulness in biomedical research. (5 refs)

78-4079 **Immunity to Transplantable Carcinogen-induced Fibrosarcomas in B2/B2 Chickens.** (Eng) Palladino, M. A. (New York Univ., New York, NY, 10003). *Diss Abstr Int B* 39(4): 1700; 1978. (no refs)

78-4080 **Effect of Adult Thymectomy on Tumour Immunity in Mice.** (Eng) Takei, F. (Dept. Microbiology, Univ. British Columbia, Vancouver, British Columbia, Canada V6T 1W5); Levy, J. G.; Kilburn, D. G. *Br J Cancer* 37(5): 723-731; 1978.

The effect of adult thymectomy (ATx) on (1) the growth of the P815 mastocytoma in DBA/2J mice, (2) the ability of mice to generate cytotoxic cells specific for P815, and (3) the ability of tumor-bearing animals to generate suppressor cells specific for this tumor was studied. Spleen cells from adult mice that had been Tx 8wk previously demonstrated a severely impaired primary cytotoxic response to P815 tumor cells, whereas their cytotoxic responses to allogeneic cells (C57BL/6) and to non-H-2 antigens (BALB/c) and their ability to form a primary antibody response to sheep RBC were unimpaired. Suppressor T cells, specific for P815 cells, appeared after 4-8 days in the thymuses of animals inoculated with P815 cells. No differences in tumor growth between ATx animals and sham-operated controls were observed, and the Tx tumor-bearing animals and untreated controls had equal levels of specific suppressor cells in their lymph nodes. Severely thymocyte-deprived mice that had been Tx, irradiated, and reconstituted with either marrow or spleen cells 8 wk before tumor implantation succumbed more rapidly to metastatic tumor than did all control animals. The data suggest that tumor immunity and temporary tumor containment, at least with this tumor line, depend on the presence of an intact immune system. (20 refs)

78-4081 **Glucan-induced Enhancement in Host Resistance to Experimental Tumors.** (Eng) Di Luzio, N. R. (Dept. Physiology, Tulane Univ. Sch. Medicine, New Orleans, LA, 70112); Hoffman, E. O.; Cook, J. A.; Browder, W.; Mansell, P. W. *In: Control of Neoplasia by Modulation of the Immune System.* Chirigos, M. A., ed.; Raven Press, New York 1977. 2: 475-499; 1977.

The influence of the macrophage stimulant, glucan, and/or glucan-activated macrophages on the growth and dissemination of acute myelogenous tumor cells was studied in Long Evans rats, as was the effect of glucan on the growth of the mouse melanoma and adenocarcinoma tumor model. Iv glucan inhibited tumor growth and promoted survival when given to rats before or after tumor cell inoculation. Sc administration of glucan at a nontumor site had little effect on the primary tumor but reduced metastases. Glucan also reversed tumor growth and metastases in rats when the primary tumor was 2% to 3% of the body wt and when infiltration of the liver, lung, and spleen had occurred. Primary tumor growth was significantly reduced when tumor cells were transplanted sc with glucan-activated peritoneal macrophages in ratios of 10:1, 2:1, and 1:1. The most pronounced inhibition of liver, lung, and spleen metastasis occurred when the tumor: macrophage ratios were 2:1 or 1:1. Facilitation of tumor growth was never observed in glucan-treated animals. Glucan, a potent nonspecific immunostimulant, has distinct advantages over the currently employed bacterial agents and should be of significant value in evaluating various concepts of immunotherapy. Activated macrophages appear to be key cells in the regulation of the internal environment against malignant cell populations. (80 refs)

78-4082 **Effect of Chemical Carcinogen Treatment on the Immunogenicity of Mouse Mammary Tumors Arising from Hyperplastic Alveolar Nodule Outgrowth Lines.** (Eng) Ruppert, B. (Dept. Cell Biology, Baylor Coll. Medicine, Texas Medical Center, Houston, TX, 77030); Wei, W.; Medina, D.; Heppner, G. H. *J Natl Cancer Inst* 61(4): 1165-1169; 1978.

The effects of chemical carcinogen treatment of precursor hyperplastic alveolar nodules (HAN) on the immunogenicity of subsequent mammary tumors were examined in female BALB/cCrgl mice. Mice were treated before or after the appearance of precursor HAN with 3-methylcholanthrene or dimethylbenzanthracene (DMBA), each 0.5 mg. The results of in vitro immunogenicity assays were compared with those of in vivo transplantation resistance tests. This comparison demonstrated that the in vitro assays predicted qualitatively and quantitatively the results from the in vivo test. With both tests, immunogenicity was frequent with tumors derived from chemically induced HAN and infrequent with those from hormonally induced HAN. The specificity of the immunogenic reactivity appeared to be unique to tumors arising from the same nodule line and did not extend to other mammary tumors (in vivo) or to a DMBA-induced rhabdomyosarcoma (in vitro). In addition, the data suggested that the immunogenicity of a tumor depends on events occurring during the development of precursor HAN rather than during the development of subsequent mammary tumors. (18 refs)

78-4083 **Solubilization and Partial Characterization of a Tumor-associated Transplantation Antigen of the Guinea Pig L_2C Leukemia.** (Eng) Hu, C. P. (Lab. Immunology, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD, 20014); Konen, T. G.; Hoes, C.; Green, I. *J Immunol* 120(5): 1521-1525; 1978.

Different methods to solubilize a tumor-associated transplantation antigen (TATA) from guinea pig L_2C leukemia cells were evaluated. These methods were papain digestion, detergent (Triton X-100 or NP-40) solubilization, and 3M KCl extraction of L_2C cells. The latter method yielded the most immunogenic TATA of L_2C cells. Immunization of normal strain 2 guinea pigs with this extract in complete Freund's adjuvant gave complete protection against a subsequent challenge with tumor cells. Immunization with papain-digested material did not lead to protection; detergent-solubilized antigen gave only partial protection. Further fractionation of the KCl extract of L_2C cells by Sephadex G-200 chromatography suggested that the immunogenic activity was present in the fraction containing materials with estimated mol wts of $<20{,}000$ daltons. (33 refs)

78-4084 **Immune Responses to Weakly Immunogenic Virally Induced Tumors. II. Suppressive Effects of the In Vivo Carried Tumor YAC.** (Eng) Devens, B. (Lautenberg Center General and Tumor Immunology, Hebrew Univ. Hadassash Medical Sch., Jersualem, Israel); Galili, N.; Deutsch, O.; Naor, D.; Klein, E. *Eur J Immunol* 8(8): 573-578; 1978.

The failure of the in vivo tumor line YAC (a Moloney Virus-induced tumor in A mice) to stimulate immunologic memory in A or C57BL/6 mice, in contrast to the in vitro subline YAC-1, was investigated. A and C57BL/6 mice were primed with the syngeneic or allogeneic tumors YAC, YAC-1, or RBL5, a Rauscher virus-induced in vitro tumor subline in C57BL/6 mice that cross-reacts serologically with YAC and YAC-1. Two wk later, their spleen cells were cultivated alone or in the presence of a syngeneic or allogeneic sensitizer. Spleen cells from unprimed animals were sensitized with the same tumors, and their responses were compared with those of the primed spleen cells. The antitumor cytotoxic response (CTR) of the cultures was tested after 6 days by ^{51}Cr-release assay. Spleen cells from A and C57BL/6 mice immunized with YAC-1 or RBL5 generated significant syngeneic cytotoxicities after cultivation in vitro. Unprimed spleen cells from these mice did not produce CTR's to their syngeneic tumors. YAC, unlike the in vitro sublines, could not stimulate a priming effect. Spleen cells from YAC-injected A mice suppressed the in vitro syngeneic CTR of primed spleen cells from YAC-1-injected mice. Spleen cells from YAC-injected C57BL/6 mice suppressed the CTR of spleen cells from RBL5-primed C57BL/6 mice. Supressor cells from YAC-primed mice can thus abrogate the CTR of differentiated memory cells from YAC-1- or RBL5-primed mice. The possible explanation of this effect is that the suppressor cells recognized idiotypic markers of their receptors on the suppressed memory cells. (10 refs)

See also:
*(Rev.): 78-3651, 78-3652, 78-3653, 78-3654.
*(Chem.): 78-3732, 78-3745, 78-3749, 78-3905.
*(Phys.): 78-3920.
*(Viral): 78-3946, 78-3952, 78-3971, 78-3973, 78-3975, 78-3988, 78-3991, 78-3992, 78-3999, 78-4001, 78-4031, 78-4042, 78-4044.
*(Path.): 78-4085, 78-4090, 78-4092, 78-4107, 78-4111.
*(Epid.-Biom.): 78-4155.

PATHOGENESIS

78-4085 **Familial Occurrence of Chronic Lymphatic Leukemia: HLA-Antigen and Cytogenetic Studies.** (Ger) Fazekas, T. (I. Medizinische Klinik der Universitat Szeged, P. B. 469, H-6701 Szeged, Hungary); Bach, K.; Toth, S.; Bodor, F. *Wien Med Wochenschr* 128(9): 262-264; 1978.

The finding of chronic lymphatic leukemia (CLL) in a sister (born 1904) and two brothers (born 1902 and 1907) and, subsequently, in the son of the older brother is reported. Eight other sisters are unaffected (2 died in infancy of unknown causes). One of these was studied at age 55 yr, but there were no abnormal hematological findings. Three other children of the three original patients were also studied, but no relevant findings resulted. The sister died 9 yr after diagnosis of CLL. One of the brothers has been treated only for symptoms and the other with Leukeran and prednisolone. Both are doing well since diagnosis in 1972. Antigen typing of the older brother indicated human-leukocyte antigen (HLA)-A, W_{25}, B_{27}, BW_{35}. The affected son of this brother had HLA-A_1, B_5, BW_{35}. The other brother had HLA-A_1,B_5, BW_{35}. Analysis of 50 metaphases of the brother showed that both have the karyotype 46,XY. Literature on the familial incidence of leukemia is reviewed briefly. (69 refs)

78-4086 **Familial Erythroleukaemia: A Cytogenetic and Haematological Study.** (Eng) Davidson, R. J. (Haematology Unit, Dept. Pathology, Univ. Aberdeen, Foresterhill, Aberdeen AB9 2ZD, Scotland); Walker, W.; Watt, J. L.; Page, B. M. *Scand J Haematol* 20(4): 351-359; 1978.

Clinical and hematological findings in a brother and sister (aged 42 and 39 yr, respectively) with a familial myeloproliferative disorder (erythroleukemia) are presented. Their father is presumed to have died from a similar disease. The two siblings showed similar but not identical chromosomal abnormalities that were detected initially in the marrow and later in the peripheral blood. Serial studies revealed the emergence of an increasingly diverse pattern of chromosomal changes concomitant with the progression of the disease. The changes included missing B-group chromosomes, C-group additions, 47, XY and 48, XY clones in the brother, and 47, XX and 48, XX clones in the sister. Cytogenetic abnormalities were also found in 5/15 asymptomatic relatives. (23 refs.)

78-4087 **Pure RBC Aplasia Associated with Chronic Granulocytic Leukemia.** (Eng) Kitahara, M. (Div. Hematology-Oncology, Dept. Medicine, Univ. Utah Coll. Medicine, Salt Lake City, UT). *JAMA* 240(4): 376; 1978.

The rare association of pure RBC aplasia and chronic granulocytic leukemia (CGL) is reported. Philadelphia chromosome (Ph^1)-positive CGL was diagnosed in a 54-yr-old woman who presented with splenomegaly, anemia, and leukocytosis. Bone marrow examination showed myeloid hyperplasia, with a myeloid to erythroid (M:E) ratio of 6:1. Therapy with busulfan (BS: 4 mg/day for 5 wk) and allopurinol (300 mg/day) was started. Three mo later, the patient was severely anemic, with a hematocrit value of 15%. Bone marrow examination showed myeloid hyperplasia and adequate megakaryocytes, but an M:E ratio of 100:1. Bone-marrow culture was Ph^1-positive. The presence of plasma erythroblast antibodies could not be demonstrated, and the aplasia could not be reversed by immunosuppressive therapy. From the onset of aplasia to her death 3.5 yr later due to terminal transformation of CGL, the patient received $>$ 200 units of packed RBC. The administration of BS to CGL patients reduces the number of WBC but generally does not inhibit RBC production. The active granulopoiesis and megakaryopoiesis during the RBC plasia stage suggest that the stem cell compartment was fully capable of producing WBC and platelets. Therefore, a causative role for BS seems unlikely. In addition, the protracted courses of this patient provide evidence that pure RBC aplasia dose not necessarily herald the onset of blast transformation in CGL patients. (3 refs)

78-4088 **Peripheral Microthrombotic Purpura Associated with Acute Promyelocytic Leukemia in Pregnancy. A Light and Electron Microscopic Study.** (Eng) Beller, F. K. (Universitats-Frauenklinik, Westring 11, D-4400 Munster i. Westf., W. Germany); Wagner, H.; Buchner, T. *Klin Wochenschr* 56(12): 593-599; 1978.

A case of promyelocytic leukemia in a pregnant 37-yr-old woman with four children is described. The patient was hospitalized in the 28th week of pregnancy, the fetus having died 2 days previously. Peripheral microthromboses were present on the nose, fingers, and lower extremities. The patient was heparinized, which resulted in an increase in the plasma fibrinogen level, a reduction in fibrogen breakdown products, an increase in prothrombin time, and a significant paling and decrease in size of the microthrombotic lesions. Heparin treatment was stopped, labor was induced, and heparin was restarted for 3 days after delivery. The patient developed generalized cramps, became comatose, and died 9 days after delivery. Autopsy confirmed a diagnosis of acute promyelocytic leukemia with a generalized hemorrhagic diathesis and massive cerebral ventricular hemorrhage. The exacerbation of disseminated intravascular coagulation is explained by a hypercoagulable state in pregnancy in association with the as yet unknown etiology of "promyelocytic fibrinopathic leukemia." (24 refs)

78-4089 **Specificity of Acquired Clonal Chromosome Abnormalities in New Zealand Black Mice.** (Eng) Fialkow, P. J. (Div. Clinical Genetics, Dept. Obstetrics and Gynecology, Univ. Texas Health Science Center, Dallas, TX); Bryant, J. I.; Friedman, J. M. *Int J Cancer* 21(4): 505-510; 1978.

Chromosomally abnormal cells in the spleens of spontaneous lymphoreticular malignancies of New Zealand Black (NZB) mice were subjected to detailed cytogenetic analyses. Increased reticulum cell proliferation was noted upon histopathological examination of the spleens from all 10 mice studied. The numbers of extra chromosomes in the abnormal cells ranged from 2 to 13 beyond the normal diploid complement of 40. A clonal origin of the hyperdiploid cells was suggested by the fact that in each mouse the chromosome numbers in these cells clustered about a single node. This origin was confirmed by karyotypic analysis and by the occurrence of distinctive marker chromosomes. The karyotypic changes were highly nonrandom, usually involving chromosomes 15 (10/10 mice), 2 (8/10 mice), 16 or 17 (9/10 mice), and the sex chromosomes (6/10 mice). These results demonstrate the presence of an important, but as yet undefined, relationship in mice between abnormal lymphoreticular proliferation and polysomy for the 15th chromosome. They also suggest an important relationship between chromosomal alterations and tumorigenesis in NZB mice. (15 refs)

78-4090 **Hodgkin's Disease--A 'B' Neoplasm?** (Eng) Staples, W. G. (Dept. Pathology, Wellington Clinical Sch., Wellington, New Zealand); Visser, A. E. *S Afr Med J* 54(5): 186-188; 1978.

Lymph node tissue from two patients with Hodgkin's disease was studied by light microscopy using the immunoperoxidase technique with a panel of monospecific antisera. The epithelioid cells were strongly positive for muramidase, thus confirming their histiocytic nature. The Reed-Sternberg cells and mononuclear Hodgkin's cells were negative for muramidase, but showed variable positivity for gamma heavy chains and lambda and kappa light chains. The positivity with mu and alpha chains was very weak and often absent. The available evidence is conflicting and it is not yet possible to state whether Hodgkin's disease is a T- or a B-cell neoplasm. (18 refs)

78-4091 **Cultural, Morphological, Cell Membrane, Enzymatic, and Neoplastic Properties of Cell Lines Derived from a Hodgkin's Disease Lymph Node.** (Eng) Roberts, A. N. (Dept. Microbiology, Univ. Tennessee Center Health Sciences, Memphis, TN, 38163); Smith, K. L.; Dowell, B. L.; Hubbard, A. K. *Cancer Res* 38(9): 3033-3043; 1978.

A neoplastic cell line (designated HuT_{11}) was established in continuous culture from an involved lymph node of a patient with Stage IIA Hodgkin's disease (mixed cellularity type). The line is morphologically heterogeneous, consisting of mononucleate lymphoidlike cells, polygonal epithelioid cells, and mono-, bi-, and multinucleate giant cells. Four clones initiated from isolated binucleate giant cells were also established as continuous cell lines. The cloned lines are morphologically distinct and more homogeneous, although typical giant cells have consistently appeared throughout long-term culture. The HuT_{11} lines grew as monolayers in McCoy's Medium 5A supplemented with 10% fetal calf serum, with generation times of 12-14 hr and high saturation densities. Early and later passages of HuT_{11} cells were aneuploid, and all cell lines were successfully heterotransplanted in the hamster cheek pouch. Repeated indirect immunofluorescence (IF) examinations showed that each line is negative for Epstein-Barr virus nuclear antigen. Indirect IF tests in which monospecific immunoglobulins were used revealed positive membrane reactions for the γ(heavy) chain and κ(light) chain of human IgG in approx 20% of viable cells in each line; however, direct IF with anti-human IgG $F(ab')_2$ reagent failed to confirm these reactions. Rosette tests for B- and T-lymphocyte and macrophage membrane receptors were negative. All cell lines were strongly phagocytic for latex particles and neutral red dye. Cytochemical stains of the monolayers revealed abundant esterase, fluoride-resistant nonspecific esterase, acid phosphatase, and leucine aminopeptidase activities, but lysozyme assays were negative. Although some properties of the HuT_{11} lines have suggested a macrophage derivation, an undifferentiated lymphoid cell origin of the Hodgkin's neoplastic cell remains a possibility. (69 refs)

78-4092 **B-Cell Nature Malignant Lymphoma Probably Caused by EB-Virus Infection.** (Eng) Emura, I. (Dept. Pathology, Niigata Univ. Sch. Medicine, Niigata, Japan); Ohnishi, Y.; Oboshi, S. *Virchows Arch Pathol Anat Histol* 378(4): 321-328; 1978.

Herpes virus-type particles were found in the tumor tissue of a 58-yr-old man with a sarcomatous type of malignant reticulosis that appeared to be B-cell lymphoma. Electron microscopy of tumor cells removed from the pericardial fluid revealed herpes-type virus particles in the nuclei. Epstein-Barr virus (EBV)-associated nuclear antigen and virus capsid antigen were found in the tumor cells, the pericardial fluid, and in cultivated cells. Serological studies revealed high anti-toxoplasma antibody levels in the pericardial fluid and in the serum. Both EBV and toxoplasma infections are assumed to have played an important role in the pathogenesis of this tumor. (8 refs)

78-4093 **Malignant Lymphoma and Multicentric Colonic Carcinomas as a Complication of Long-standing Ulcerative Colitis.** (Ger) Otto, H. F. (Pathologisches Institut der Universitat, Martinistrasse 52, 2000 Hamburg 20, W. Germany); Gebbers, J. O.; Matthaes, P.; Muller-Wieland, K. *Inn Med* 5(5): 189-195; 1978.

A 40-yr-old man with a 14-yr history of ulcerative colitis underwent total proctocolectomy for a rectal adenocarcinoma. Histologic examination of the operative specimen revealed two Dukes A carcinomas of the rectum and splenic flexure, multiple adenomatous and villous precancerous lesions, and one lymphoblastic-type malignant lymphoma of the upper sigmoid colon. A review of the literature revealed 18 cases of malignant non-Hodgkin's lymphoma and 3 cases of Hodgkin's disease of the colon and rectum arising in patients with ulcerative colitis. (25 refs)

78-4094 **Longitudinal Studies of Chromosomal Abnormalities and Reticulum Cell Proliferation in New Zealand Black Mice.** (Eng) Friedman, J. M. (Medical Genetics Section, Medical Service, Veterans Admin. Hosp., Seattle, WA, 98108); Fialkow, P. J.; East, J.; Bryant, J. I.; Salo, A. C. *Int J Cancer* 22(4): 454-457; 1978.

Serial cytogenetic and histopathological examinations were performed on the spleens of 40 New Zealand Black (NZB) mice, an inbred strain susceptible to the development of clones of aneuploid (Anp) spleen cells and malignant reticulum cell proliferation, and 20 BALB/c control mice. A population of Anp cells was detected in direct spleen preparations from 10/36 NZB mice $\geq$6 mo old. Definite or possible reticulum cell sarcoma type B was diagnosed in 10/32 NZB mice $\geq$11 mo old but in only 1/34 younger mice. No neoplasms were found in control mice. In mice with both splenic Anp mosaicism and reticulum cell neoplasia, aneuploidy (Anpy) was found earlier than the neoplasia. Although there was no apparent association between splenic Anpy and neoplasia in single specimens obtained from NZB mice at various ages, there was a significant association between the occurrence of Anp mosaicism some time during life and the histological diagnosis of reticulum cell neoplasia in NZB mice $\geq$8 mo old. This finding is compatible with the hypothesis that Anp clones that arise in the spleens of aging NZBs are at least potentially neoplastic. The results indicate that inability to demonstrate chromosomal abnormalities in a direct preparation cannot be used as evidence against the presence of karyotypic alterations within the neoplastic cells themselves. (10 refs)

78-4095 **Neoplastic Behavior of Chromosomally Abnormal Clones in New Zealand Black Mice.** (Eng) Friedman, J. M. (Medical Genetics Section, Medical Service, Veterans Admin. Hosp., Seattle, WA, 98108); Fialkow, P. J.; Bryant, J.; Reddy, A. L.; Salo, A. C. *Int J Cancer* 22(4): 458-464; 1978.

The distribution of aneuploid (Anp) cells within the spleens and other organs of New Zealand Black (NZB) mice was determined and compared to that of neoplastic reticulum cells to define the relationship between neoplasms and chromosomal abnormalities. The results indicate that Anp cells arise focally and may remain so for > 1 mo. All 14 mice studied histopathologically had abnormal reticulum cell proliferation except one, a mouse with lymphocytic leukemia. Aneuploidy (Anpy) was found in the marrow, nodes, and spleen of this mouse. Four other mice had Anpy in nodes as well as in the spleen, but only 2/13 had Anpy in the marrow. Spleen cells were transplanted into newborn NZB recipients from 10 old NZB donors, 6 of which had obvious Anpy. In each instance, at least one recipient had splenic Anp mosaicism and/or reticulum-cell sarcoma before 7 mo of age. In every case in which spleen cell Anpy was found in a young recipient mouse, the abnormal population was apparently clonal and of donor origin. Spleen cells from the three donors without Anpy did not induce histological malignancy or splenic Anpy. As opposed to Anpy, no statistically significant association was found between histologically diagnosed reticulum cell sarcoma in a donor and the transplantability of its spleen cells. These and previous data strongly support the hypothesis that Anpy and neoplasia occur in the same population of spleen cels in NZB mice and suggest that karyotypic abnormalities may be important in the development of reticulum cell neoplasia in NZB mice. (11 refs)

78-4096 **Early Lymphatic Spread of Osteogenic and Soft-Tissue Sarcomas.** (Eng) Weingard, D. N. (Surgery Branch, NCI, Bethesda, MD, 20014); Rosenberg, S. A. *Surgery* 84(2): 231-240; 1978.

The records of all 374 patients (227 males and 147 females, aged 9 mo to 80 yr) with osteogenic and soft tissue sarcomas who had been referred to NCI from 1953 to 1977 were reviewed to determine the incidence of lymph node (LN) involvement. Of the 210 patients were definitive therapy at NCI, 143 underwent surgery than included regional LN groups. The LN's were enumerated in the pathology reports of 113 surgical patients. The 5-yr survival rate for the 113 was 32.7% as compared with 41.6% for the larger group of 210. Early LN involvement was found among 6/113 patients, although only 3 cases represented true metastases. Five of the six patients died within 25 mo of initial treatment. Seventeen patients underwent prophylactic LN dissection. Only one of these disections was positive for metastasis, and the survival rate in this group did not differ from that of the larger group. Although removal of clinically suspicious LN's is reasonable, there appear be little justification for treating clinically uninvolved draining regional LN's in patients with sarcomas other than childhood rhabdomyosarcomas. (66 refs)

78-4097 **Electron Microscope Analysis of Myogenic Neoplasms.** (Rus) Galil-Ogly, G. A. (Res. Roentgenoradiological Inst., Moscow, USSR); Krylov, L. M.; Poroshin, K. K. *Vopr Virusol* 40(2): 19-25; 1978.

In an attempt to evaluate the differential diagnostic criteria of myogenic sarcomas, specimens of leiomyoma of the skin arteries, esophageal leiomyoma, leiomyosarcoma (metastatic node in the lung), and rhabdomyosarcoma of the soft tissues of the shin were analyzed electron microscopically. The leiomyoma cells contained myofibrils with characteristic focal indurations, basal membrane, and clusters of pinocytic vesicles. The blastomatous cells of the differentiated leiomyosarcoma had a reduced number of pinocytic vesicles and a fragmented basal membrane. Both the leiomyosarcoma and the esophageal leiomyoma contained intercellular desmosome-like formations. Some rhabdomyosarcoma cells contained striated myofibrils. (10 refs)

78-4098 **Origin of Familial Malignant Melanomas from Heritable Melanocytic Lesions: "The B-K Mole Syndrome".** (Eng) Clark, W. H. (Dept. Dermatology, Hosp. Univ. Pennsylvania, 36th and Spruce St., Philadelphia, PA, 19104); Reimer, R. R.; Greene, M.; Ainsworth, A. M.; Mastrangelo, M. J. *Arch Dermatol* 114(4): 732-738; 1978.

A distinctive syndrome occuring in six families affected by familial malignant melanoma is described. Among the family members examined, 15/17 patients with melanoma and 22/41 nonmelanoma relatives had unique melanocytic moles, whose clinical and histological features have been designated the B-K mole syndrome. The clinical features include the presence of <10 to >100 prominent moles on the upper trunk and extremities and variability of mole size (5-15 mm), outline, and color combination. Histologically, B-K moles show atypical melanocytic hyperplasia, lymphocytic infiltration, delicate fibroplasia, and new blood vessels that occur within a compound nevus or de novo. The transformation of two B-K moles into malignant melanomas was documented photographically. Six unsuspected new primary cutaneous malignant melanomas were diagnosed during the study. These observations coupled with the photographic evidence of transformation demonstrate that the large moles of the B-K mole syndrome may be formal precursors of malignant melanoma. (12 refs)

78-4099 **Hormonal Activation of Adenylate Cyclase in Mouse Melanoma Metastatic Variants.** (Eng) Niles, R. M. (Div. Surgery, Boston Univ. Sch. Medicine, Room 908, Instructional Building, 80 E. Concord St., Boston, MA, 02118); Makarski, J. S. *J Cell Physiol* 96(1): 355-360; 1978.

The ability of hormones that interact with cell-surface receptors, ie, melanocyte stimulating hormone (MSH), ACTH, and prostaglandin E_1 (PGE_1), to activate a plasma membrane-bound enzyme, adenylate cyclase, was examined in intact mouse melanoma cells (B16 F_1, F_5, and F_{10}) of varying metastatic potential (MP). F_1 cells (low MP) had significantly greater cyclic AMP (cAMP) levels in response to all three hormones than F_5 (intermediate MP) and F_{10} (high MP) cells. The ranking of the response was: MSH, $F_1 > F_5 > F_{10}$; ACTH, $F_1 > F_5 > F_{10}$; PGE_1, $F_1 > F_{10} > F_5$. In contrast, the degree of hormonal stimulation of adenylate cyclase in broken cell preparations was virtually identical in all three melanoma cell lines. Control enzyme activity was depressed in both F_5 and F_{10} relative to F_1. The discrepancy between the ability of hormones to increase cAMP levels in intact vs broken cells could not be accounted for by differences in cAMP phosphodiesterase activity. However, it is concluded that the results from intact cells provide the best approximation to physiological mechanisms and hormone action and that as melanoma cells increase in MP, there is a significant loss in the ability of their cAMP system to respond appropriately to hormonal stimuli. (11 refs)

78-4100 **New Contributions to the Study of Endometriosis. Local Tissue Characteristics Favoring Growth of Endometrial Tissue. Study of 1,097 Cases.** (Fre) Jonesco, M. V. (3, Blvd. Dimitrie Cantemir, Bucharest, Rumania); Popesco, C. *Gynecologie* 28(6): 549-556; 1977.

Various tissue characteristics that favor the growth of endometrial tissue are presented. From 1955 to 1967, histological studies were performed on 1,097 patients with endometrioses. The most common site for ectopic endometrial growth was the fallopian tubes (513), followed by the uterus (445) and the ovary (110). Factors favoring endometrial tissue growth include diminished local tissue immunity, vascularization, and the presence of mucopolysaccharides and glycogen. (10 refs)

78-4101 **Gastrointestinal Polyposis in Multiple Myeloma.** (Eng) Goeggel-Lamping, C. (Cancer Inst., Hahnemann Medical Coll. and Hosp., Philadelphia, PA, 19102); Kahn, S. B. *JAMA* 239(17): 1786-1787; 1978.

A 69-yr-old woman had myeloma beginning as solitary plasmacytoma with progression to extensive skeletal and extraskeletal (gluteal region, gastrointestinal polyposis) involvement. The major clinical problems were related to the gastrointestinal tract lesions. Postmortem exam revealed involvement of the marrow, small bowel, mesenteric nodes, ovaries, uterus, kidneys, pancreas, and stomach. At presentation, there was no evidence of immunoglobulin abnormalities or Bence-Jones protein excretion, making this case unusual. (3 refs)

78-4102 **Condyloma Acuminatum of the Nipple.** (Eng) Wood, C. (Dept. Pathology, Room B-1265, Univ. Maryland Hosp., 22 S. Greene St., Baltimore, MD, 21201). *J Cutan Pathol* 5(2): 88-89; 1978.

A 20-yr-old male mental patient had viral condyloma acuminatum (CA) of the nipple with underlying Darier's disease that, because of its often exudative nature, may have contributed to the unusual location of the lesion. There were numerous papules of Darier's disease in the skin adjacent to the base of the papilloma, but changes characteristic of this disease and CA were not both present at the same point. (5 refs)

78-4103 **DNA Repair Processes in Human Cells (Meeting Abstract).** (Eng) Cleaver, J. E. (Lab. Radiobiology, Univ. California, San Francisco, CA, 94143). *Mutat Res* 53(2): 169; 1978. (no refs)

78-4104 **Excessive Mutational Occurrences in Wild Peruvian House Mice (Meeting Abstract).** (Eng) Wallace, M. E. (Dept. Genetics, Univ. Cambridge, London, England); Berry, R. J. *Mutat Res* 53(2): 282-283; 1978. (3 refs)

78-4105 **Basal Cell Carcinoma in Identical Twins (Letter to Editor).** (Eng) Kaprio, J. (Dept. Public Health Science, Univ. Helsinki, Helsinki, Finland); Teppo, L. *Br Med J* 2(6134): 436; 1978.

Basal cell carcinoma occurred in four pairs of identical male twins. The crude proband concordance rate in identical twins is much higher than the risk in the general population, but the common familial environment is a confounding factor that should be considered. (1 ref)

78-4106 **Electrophoretic Shift Mutants in Chinese Hamster Ovary Cells: Evidence for Genetic Diploidy.** (Eng) Siciliano, M. J. (Biology Dept., M. D. Anderson Hosp. and Tumor Inst., Univ. Texas System Cancer Center, Houston, TX, 77030); Siciliano, J.; Humphrey, R. M. *Proc Natl Acad Sci USA* 75(4): 1919-1923; 1978.

Chinese hamster ovary (CHO) cells were screened for electrophoretic shift variants of enzymes coded by approx 40 genetic loci to test the hypothesis of functional hemizygosity as a general theory to explain the expression of recessive mutations in CHO cells. Clones isolated after exposure to UV radiation were examined by starch gel and Cellogel electrophoresis. Shift variants were recovered for enzymes representing 11 different loci. Variant clones were subcloned to demonstrate the heritability of the variations. Mutants at nine loci produced multiple-banded patterns consistent with the patterns expected of genes at loci represented twice (diploid loci). Only one locus appeared to be functionally haploid. Chromosome localization of these diploid loci in other mammalian species where they have been mapped (mouse and man) suggests that they represent a random sample of CHO genes. Chromosome analysis of mutant subclones indicated that the variation did not occur in tetraploid cells. The data indicate that the quasi-diploid CHO cells are only as functionally hemizygous as would be expected of a slightly hypodiploid cell line derived from an organism in which the haploid number is 11. (33 refs)

78-4107 **Genetic Analyses of Differences in Incidence o Mammary Tumors and Reticulum Cell Neoplasms with the Use of Recombinant Inbred Lines of Mice.** (Eng) Dux, A. (Netherlands Cancer Inst., Sarphatistraat 108, Amsterdam C, Netherlands); Muhlbock, O.; Bailey, D. W. *J Natl Cancer Inst* 61(4): 1125-1129; 1978.

The influence of genes, in addition to genes in the *H-2* complex, that affect the mammary tumor incidence differences between BALB/c and B6 mice was studied with the use of seven recombinant inbred (RI) CXB lines. These lines are well-suited for investigation of the genetics of a trait for which the genotype affects probability of phenotype expression and which therefore is measured as incidence. Females of lines CXBD, CXBE, CXBG, CXBH, CXBI, CXBJ, and CXBK and their progenitor strains C57BL/6By (B6) and BALB/c-By (BALB/c) were inoculated ip with MuMTV at 3 mo of age and were force bred. They were observed for mammary tumors. The B6 strain was least susceptible, and mammary tumors appeared late in life. The BALB/c strain was most susceptible, and the tumors appeared early in life. The course of tumor development in the RI lines fell between these extremes. The RI strain distribution pattern of mammary tumor incidence indicated that at least one and probably several loci in addition to those at *H-2* determined the difference between the BALB/c and B6 strains. Effects of the other gene(s) appeared to be even more important than those of *H-2* The locations of these loci were not elucidated by this study. The spontaneous incidence of reticulum cell neoplasms was also recorded. The most frequent neoplasm was a Hodgkin's-like lesion. The data suggest an influence of the *H-2* complex. (9 refs)

78-4108 **Expression of Transformation in Cell Hybrids II. Nonsuppression of the Transformed Phenotype in Hybrids Between a Chemically Transformed and Nontransformed Derivatives of BALB/3T3.** (Eng) Jha, K. K. (Dept. Biological Sciences, Hunter Coll., CUNY, 695 Park Ave., New York, NY, 10021); Cacciapuoti, J.; Ozer, H. L. *J Cell Physiol* 97(2): 147-152; 1978.

Hybrids between a nitrosocarbaryl-transformed BALB/3T3 cell line, clone H, and a nontransformed cell line THO2, were studied. Both cell lines were derived from BALB/3T3 clone A31. Line THO2 is deficient in hypoxanthine-guanine phosphoribosyltransferase (HGPRT) and is resistant to cytotoxic concentrations of ouabain. Clone H exhibits morphologic and growth characteristics associated with the transformed phenotype: it grows to high saturation density, it is anchorage-independent, and it is able to form clones on monolayers of normal 3T3 cells. In contrast, THO2 retains growth properties characteristic of untransformed cells similar to the 3T3 parental cells. The deficiency of HGPRT, a recessive marker, and resistance of ouabain, a codominant marker, in THO2 allow selection of cell hybrids in medium supplemented with hypoxanthine, aminopterin, thymidine, and ouabain without the need for any induced selective markers in the other parent. The results show that these hybrid clones resemble the transformed parent in the clone morphology, higher saturation density, colony formation in medium with reduced serum concentration, growth in agarose, and ability to form clones on BALB/3T3 monolayers. The results are discussed in the framework of genetic models that permit or require dominant mutations for expression of the transformed phenotype. (33 refs)

78-4109 **Dysplastic Gangliocytoma of the Cerebellum - An Ultrastructural Study.** (Eng) Pritchett, P. S. (Cooper Green Hosp., 1515 6th Ave. S., Birmingham, AL, 35233); King, T. I. *Acta Neuropathol (Berl)* 42(1): 1-5; 1978.

Ultrastructural findings in a case of dysplastic gangliocytoma of the cerebellum are described. The patient was a 24-yr-old man who underwent surgery to remove a firm solid tumor that was diffusely infiltrating the right cerebellar hemisphere. Light microscopy revealed an enlarged cerebellar cortex with a densely staining outer zone and a pale inner zone. The outer zone was homogeneous, acellular and spongiotic, but the inner zone (granular layer) was cellular. Electron microscopy revealed that the granular layer was populated almost exclusively by large neurons 40-60μm in size. Cell shape was irregular and cell size variable. Intranuclear inclusions 1-2μm in size were seen infrequently. On high magnification, these inclusions were fibrillogranular in nature, and no viral bodies were identified. There was no evidence of a viral etiology for the disease. Both the light and electron microscopy findings support the theory that the abnormal neurons are hypertrophied granule cells. (13 refs)

78-4110 **Development of Cerebellar Malignant Astrocytoma at Site of a Medulloblastoma Treated 11 Years Earlier. Case Report.** (Eng) Kleriga, E. (Dept. Neurological Surgery, State Univ. New York, Downstate Medical Center, Brooklyn, NY); Sher, J. H.; Nallainathan, S. K.; Stein, S. C.; Sacher, M. *J Neurosurg* 49(3): 445-449; 1978.

A boy treated with surgery and radiation for a desmoplastic medulloblastoma of the left cerebellar hemisphere at age 10 mo developed a malignant astrocytoma at the site 11 yr later. It is not clear whether the second tumor represented a recurrence with differentiation or another primary. The origin of the second tumor is discussed in relation to general concepts of the genesis of medulloblastoma. (25 refs)

78-4111 **Growth of Human Gliomas in Immune-deficient Mice: A Possible Model for Pre-clinical Therapy Studies.** (Eng) Bradley, N. J. (Royal Marsden Hosp., Fulham Road, London S.W.3, England); Bloom, H. J.; Davies, A. J.; Swift, S. M. *Br J Cancer* 38(2): 263-272; 1978.

An attempt was made to provide an experimental system for screening cytotoxic and chemical radiosensitizing agents of possible value in the treatment of human gliomas and for studying the metabolism of human tumors. Thirteen gliomas from 55 neurosurgical specimens (11/25 derived from adults, 2/30 derived from children) were grown successfully after implantation (1-4 pieces/animal) into the sc flank tissue of immune-deprived or nude mice. The tumors were astrocytomas of Grades III and IV malignancy (9 adults, 1 child), medulloblastomas (1 adult, 1 child), and an oligodendroglioma from an adult. Tumor growth usually occurred several months after implantation; their human origin was confirmed by karyotypic analysis. The growth rates and histopathological picture of the initial specimens were maintained in initial and subsequent transplants, although future take rates improved in most instances. Primary human glioma tissues did not grow preferentially in nude mice, compared with immune-deprived mice. Preliminary observations suggest that these tumors in immune-reduced mice respond to agents that have been successful in humans. (15 refs)

78-4112 **Adenocarcinoma of the Nose and the Paranasal Sinuses: Association with the Woodworking Industry?** (Ger) Lobe, L. P. (Hals-Nasen-Ohren-Klinik, Bereich Medizin der Friedrich-Schiller-Universitat, Lessingstrasse 2, DDR-69 Jena, E. Germany); Ehrhardt, H. P. *Dtsch Gesundheitsw* 33(22): 1037-1040; 1978.

A detailed occupational history was obtained for 179/420 patients treated since 1931 at an ear, nose, and throat clinic in Jena, E. Germany, for malignant tumors of the nose and paranasal sinuses. Thirty-seven of the patients were woodworkers, a value that is high considering the percentage of the total population engaged in woodworking. Thirteen of the total 18 cases of adenocarcinoma occurred in these individuals ($p < 0.001$), who had worked under conditions of continuous exposure to wood dust for many years. The involvement of an exogenous factor in this type of cancer is supported by the facts that it is prevalent in the mucous membrane of the inner nose and ethmoid and that the disease occurs only after the age of 40. (13 refs)

78-4113 **Malignant Transformation of Laryngeal Papillomas.** (Jpn) Ishii, H. (Dept. Otorhinolaryngology, Gunma Univ., Sch. Medicine, Gunma, Japan); Kawabata, I. *J Otolaryngol Jpn* 80(3): 40-46; 1977.

A series of 32 patients (22 males and 10 females) treated for laryngeal papillomas since 1964 is described. Most of the patients were aged 30-59 yr; 10 were in their 60's, and 1 was < 10 yr old. The lesions appeared hyperkeratotic on laryngoscopy, and the child's lesions were not typical of the so-called multiple juvenile papillomas. Patients with localized papillomas were completely cured by surgery, but diffuse papillomas were resistant to radiotherapy or chemotherapy. Malignant transformation occurred in five patients (all men), with the interval between the onset of the symptom (hoarseness) and the transformation ranging from 4 to 9 yr. Laryngectomy was performed on all five patients, and three of the tumors were examined by serial large sections. In each case, the tumor extended to the underlying tissue, and in one case, it invaded the laryngeal cartilage. All patients survived without evidence of recurrence or metastasis. (9 refs)

78-4114 **Association Between Pulmonary Tuberculosis and Bronchial Carcinoma (Letter to Editor).** (Eng) Howie, A. J. (Dept. Pathology, Medical Sch., Birmingham B15 2TJ, England). *Lancet* 1(8089): 881; 1978.

A previously reported negative association between pulmonary tuberculosis and cancer of the bronchus is contradicted. It is currently thought that they coexist commonly, and probably more often than expected by chance. Although the exact frequency at which active tuberculosis occurs with lung cancer is not known, one study indicated that men who had tuberculosis had a higher than expected death rate from bronchial carcinoma. It is suggested that the cancer may reactivate the tuberculosis. (5 refs)

78-4115 **Enhancing Effect of Thoracotomy on Tumor Growth in Rats.** (Eng) Hattori, T. (Dept. Surgery, Res. Inst. Nuclear Medicine and Biology, Hiroshima Univ., Kasumi 1-2-3, Hiroshima 734, Japan); Hamai, Y.; Ikeda, H.; Harada, T.; Ikeda, T. *Gann* 69(3): 401-406; 1978.

To determine the influence of operative stress on tumor growth, thoracotomy (TC) and/or laparotomy (LO) were performed 48 hr after ip or iv inoculation of Sato lung cancer cells into Donryu rats. The size of the inoculum ranged from 2.5×10^6 to 10^3 cells for the ip route and from 10^5 to 10^2 cells for the iv route. Survival period, number of metastatic nodules on the surface of the lungs, and the percentage-area of metastases in the frontal section through the pulmonary hilus were examined. TC and LO+TC reduced the survival period of the tumor-inoculated rats significantly compared with control (nonoperated) rates, but difference between the two test groups was not significant. Also, there was no significant difference between the LO group and controls. The number and percentage-area of metastatic nodules were increased significantly by TC and LO+TC, but not by LO. Correlation between the number and the percentage-area of metastatic nodules was highly significant. The meaning of the stress of TC in cancer treatment is discussed. The enhancement of tumor growth by TC may depend on general host resistance rather than local factors. (15 refs)

78-4116 **Morphometric and Histological Analysis of the Lungs of Syrian Golden Hamsters.** (Eng Kennedy, A. R. (Dept. Physiology, Harvard Sch. Public Health, Boston, MA, 02115); Desrosiers, A.; Terzaghi, M.; Little, J. B. *J Anat* 125(3): 527-553; 1978.

Adult male Syrian golden hamster lung morphometry and histology were studied in an attempt to determine differences between hamster and human lungs that may have relevance for lung carcinogenesis studies. Morphometric measurements were made on fresh lungs, lung casts, and histological sections. Since the epithelium is the site of origin of most human lung cancers, numbers and types of epithelial cells in the various regions of the lungs were determined using 1-μm plastic (glycol methacrylate) and electron microscopic sections. The principal morphometric difference between hamster and human lung models resides in the declining surface area of peripheral airways encountered in the hamster lung. The decreases in length, diameter, and number of airways seen in the hamster lung combine to decrease the surface area in regions distal to the fifth generation. In models for the human lung, which have been developed and used to estimate particle deposition and clearance, the number of airways and surface area always increase as a function of generation. In its histological features the hamster trachea most closely resembles the human bronchus, the site of most human lung cancer. The types of mucous secretions produced by the lungs are different for the hamster and man, and these variations may influence the efficacy of the mucous ciliary escalator and alter the incidence and locations of tumors. Species variations in epithelial cell types may also affect tumor incidence. (56 refs

78-4117 **Localization of Bronchogenic Carcinoma in Tuberculous Lobes.** (Eng) Farwell, D. J. (87 Crystal St., New Orleans, LA, 70124); Rutledge, L. J.; Bryant, L. R.; Schechter, F. G. *South Med J* 71(4): 377-379 1978.

Twenty-seven cases of coexisting pulmonary tuberculosis and bronchogenic carcinoma were examined. Tuberculosis was confined to one or two lobes in 24/27 patients with coexisting disease; 21 of these patients had the neoplasm in a tuberculous lobe. Tuberculosis was diagnosed concurrently with carcinoma in 13 patients and 2-20 yr earlier in 12. It was confirmed 1 yr later than the cancer in one patient and 2 yr later in another. Carcinoma was diagnosed within 1 to 18 mo from hospital admission. When this series of 27 patients was combined with 11 other similar series, an av of 52% had the neoplasm in a tuberculous lobe. A low index of suspicion can delay diagnosis of the carcinoma until hilar enlargement, tumor, or metastasis is apparent radiologically. By then, the tumor has often become inoperable. It is suggested that carcinoma should be suspected in a tuberculous man of cancer age who smokes and who has a lesion that is enlarging, is in an unusual location, or is drug resistant. Cytologic study of sputum, bronchoscopy, and early thoracotomy are advocat ed. It is concluded that bronchogenic carcinoma has a striking predilection for a lobe of the lung that is or has been the seat of tuberculous infection, the most likely explanation being that this is scar carcinoma. (16 refs)

78-4118 **Microcystic Adenomas of the Pancreas and Constitutional Abnormalities (Letter to Editor).** (Eng) Soloway, H. B. (Associated Pathologists, P.O. Box 14220, Las Vegas, NV, 89107). *Am J Clin Pathol* 70(3): 445-446; 1978.

A recent study provides additional evidence for a multiorgan tumor diathesis and endocrinopathy associated with pancreatic cystadenomas. In two series of pancreatic cystadenoma patients, the combined incidence of thyroid cancer was 6.25% and that of renal cell carcinoma was 3.1%. (1 ref)

78-4119 **Duodenal Carcinoid with Cytoplasmic Whorls of Microfilaments.** (Eng) Carstens, P. H. (Dept. Pathology, Univ. Louisville Sch. Medicine, P.O. Box 1055, Louisville, KY, 40201); Broghamer, W. L. *J Pathol* 124(4): 235-238; 1978.

An unusual ultrastructure was found in a duodenal carcinoid in a 51-yr-old black man: most of the cells contained juxtanuclear whorls of microfilaments. This unique feature is described and discussed in relation to the cellular origin of this type of carcinoid. The filaments had a thickness of 8-10 nanometers (nm), and they very frequently occupied a large portion of the cytoplasm. Another prominent cytoplasmic feature was ubiquitous electron-dense, round granules. These granules, which had a diameter of approx 200 nm and were incompletely surrounded by unit membranes, were considered secretory granules. Some of the microfilament whorls contained the electron-dense granules and, in addition, centrosomelike structures. None of the tumor cells appeared to have microvilli. Similar microfilaments have been found in the D_1 cell of the gastroenteropancreatic endocrine system, and apparently identical whorllike accumulations of filaments have been described previously in the bronchial cells of animals treated with nitrosamines and in human giant cell carcinoma of the lung. It is suggested that the D_1 cell is the cell of origin for the duodenal carcinoid as well as for the above-mentioned lung tumors. (16 refs)

78-4120 **Hyperplastic Foci in Precancerous Rat Liver: Light Microscopic and Electron Microscopic Study.** (Eng) Timme, A. H. (Dept. Pathology, University Cape Town Medical Sch., Cape Town, S. Africa). *J Natl Cancer Inst* 61(2): 407-409; 1978.

Five hyperplastic foci (≤ 1 mm in diameter) induced by dimethylaminoazobenzene (DAB) in the siderotic livers of male Wistar-derived albino rats were investigated by light and electron microscopy. Rats were fed a basic maize diet containing 4% ferric citrate (wt/wt) for 2 mo, at which time the compound was removed and DAB was added (0.5%, wt/wt). The hepatocytes in the foci were larger than the surrounding cells, and they had a granular, moderately basophilic cytoplasm. Most were iron-free, although some Kupffer's cells contained hemosiderin. There were heavy deposits of iron in surrounding cells, possibly due to a defective storage mechanism in the foci cells. Under the electron microscope, the hepatocytes were shown to be well-differentiated, as evidenced by the presence of a well-developed rough endoplasmic reticulum, numerous free ribosomes, peroxisomes, Golgi bodies, bile canaliculi, and cytoplasmic junctional complexes. Thus, they resembled normal hepatocytes. Occasional ferritin-laden lysosomes persisted in some cells. In contrast, the proliferating cells of hyperbasophilic foci are poorly differentiated and bear no resemblance to normal hepatocytes. (8 refs)

78-4121 **Wilms' Tumor and Adrenocortical Carcinoma with Hemihypertrophy and Hamartomas.** (Eng) Muller, S. (Dept. Pediatrics, Freie Universitat Berlin, Heubnerweg 6, D-1000 Berlin 19, W. Germany); Gadner, H.; Weber, B.; Vogel, M.; Riehm, H. *Eur J Pediatr* 127(3): 219-226; 1978.

The case report of a 14-yr-old girl with hemihypertrophy and hamartomas who successively developed Wilms' tumor and adrenocortical carcinoma is presented. She developed the Wilms' tumor at age 2 yr, 11 mo, and, after nephrectomy, was given 3,000 rads to the right renal fossa and 12 courses of actinomycin D over 2 yr, 10 mo (total dose, 16 mg). At age 7 yr, 6 mo, she presented with rapid development of pubic hair, marked hypertrophy of the clitoris, acne vulgaris, and a sudden growth spurt. Plasma testosterone was increased markedly. An adrenocortical carcinoma was detected by arteriography at the site of the previously irradiated area, and it was removed. The girl also had hemihypertrophy (marked in the tongue), and hamartomas of the right occipital region and of the left forearm had been removed surgically at the end of the first year of life. Her parents and members of her father's family also have hamartomas. The occurrence of the two tumors with hemihypertrophy, hamartomas and advanced physical development supports the hypothesis that certain cases of Wilms' tumor and of adrenocortical carcinoma have common etiological factors, which are both teratogenic and oncogenic. Moreover, this case may provide another link between the hemihypertrophy syndrome and the EMG (exomphalos, macroglossia, and gigantism) syndrome. An alternative explanation of the induction of the second tumor by the previous radio- and chemotherapy is also given. (37 refs)

78-4122 **The Metastatic Spread of Cancers of the Digestive System in Man.** (Eng) Viadana, E. (Dept. Health, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263); Bross, I. D.; Pickren, J. W. *Oncology* 35(3): 114-126; 1978.

The most common disseminating routes for the spread of primary cancers of the human digestive system were determined by statistical analysis of autopsy reports of 58 patients with pancreatic adenocarcinomas, 143 with stomach cancer, 165 with colon cancer, and 150 with rectal cancer, all collected between 1956 and 1966. The cascade spread of metastases (previous seeding of 1 or 2 key organs is required for the disease to spread from the primary tumor throughout the body) is more common than a direct spread. With the exception of the rectal adenocarcinomas, it appears that the liver cascades metastasize to the lungs and that this is the most common route for the dissemination of metastases to the lungs. The abdominal and pelvic nodes are not responsible for the seeding of the liver. The lungs alone or the liver and lungs together very often disseminate blood-borne metastases to the thyroid, pituitary, CNS, vertebrae and other bones, kidney, adrenal glands, and spleen. For other organs, such as the ovaries, uterus, ureters, stomach, small and large intestines, pancreas, and extrahepatic biliary tract, metastases seem to originate directly from the primary cancer in 50% of the cases. In the other 50%, metastases may originate in the lungs alone or in both the liver and lungs. It is concluded that the closer the metastatic organ to the primary cancer, the less important the role of the lungs and liver in disseminating metastases to those organs. (7 refs)

78-4123 **Experimental Colo-rectal Tumours in the Rat.** (Eng) Lindstrom, C. G. (Dept. Pathology, Univ. Lund, General Hosp., Malmo, Sweden). *Acta Pathol Microbiol Scand (A)* Suppl 268: 1-75; 1978.

The induction of colorectal tumors in rats by 1,2-dimethylhydrazine and N-methyl-N'-nitro-N-nitrosoguanidine is reviewed. Comparison of sequential findings in studies of serum-blocking activities in tumor-bearers with those of double-contrast radiography and histopathologic examinations showed that the immunologic test for tumor detection is specific and reliable. (85 refs)

78-4124 **Primary Malignant Melanoma of the Small Intestine and the APUD Cell Concept.** (Eng) Krausz, M. M. (Dept. Surgery, Hadassah Univ. Hosp., Kiryat Hadassah, Jerusalem, Israel); Ariel, I.; Behar, A. J. *J Surg Oncol* 10(4): 283-288; 1978.

The case report of a 70-yr-old woman with primary melanoma of the small intestine is presented. It is suggested that this tumor could have arisen from APUD (amine precursor uptake and decarboxylation) cells derived from the neural crest. (18 refs)

78-4125 **Precancerous Changes and Histogenesis of Cancer of the Stomach.** (Rus) Dikshtein, E. A. (Dept. Pathoanatomy, Medical Inst., Gorki, USSR); Vasilenko, I. V. *Vopr Onkol* 24(7): 22-29; 1978.

The relationship between the type of precancerous change i the stomach and the histogenesis of stomach cancer was studied in 142 stomach cancer patients, 20 patients with chroni gastric ulcer, and 800 patients with chronic gastritis. Amon the cancer patients, 47 had highly differentiated adenocarcinomas, 73 had undifferentiated carcinomas, and 22 ha mixed carcinomas. Analysis of the gastric mucosa showe that 39/47 adenocarcinoma patients had severe symptoms c atrophic gastritis, compared with 12/73 patients with undi ferentiated carcinomas. The undifferentiated carcinoma patients had an increased frequency of chronic gastric ulcer (50/73, compared with 12/47 adenocarcinoma patients Proliferation of the gastric pit epithelium was observed in th gastritis patients and proliferation of the glandular epithel um, particularly of the glandular ducts, was observed in th gastric ulcer patients. These findings confirmed the association of gastritis with adenocarcinoma and of ulcers with ur differentiated carcinomas. (32 refs)

78-4126 **Influence of Duodenogastric Reflux on the Induction of Cancer of the Gastric Stump.** (Ger Dahm, K. (Chirurgische Universitatsklinik, Martinistrass 52, 2000 Hamburg 20, W. Germany); Schreiber, H. W Werner, B.; Knipper, A.; Mitschke, H. *Inn Med* 4(7 297-302; 1977.

The effect of duodenogastric reflux on the incidence of carcinomas near a gastroenteric (GE) anastomosis was studie in 72 male Wistar rats. Group I consisted of 39 rats wh underwent a short-loop anastomosis (Billroth II), which resulted in reflux of the bile and duodenal secretions into th remaining stomach. A Y-shaped GE (Roux) techniqu which delivers the bile and pancreatic juices directly into th jejunum without contacting the stomach, was used for 33 rat (Group II). The control group (27 rats) had no surgical trea ment. N-Methyl-N'-nitro-N-nitrosoquanidine (MNNG: 12 mg/liter drinking water) was given to all groups for 33 wl One week after the end of MNNG exposure, 13/22 Grou I rats had malignant tumors: 11 had adenocarcinomas, 2 sarcomas. The most common histological type of adenocarcinoma was the intestinal type, according to the new nomenclature. Carcinomas were found in 7/21 Group II rats, 4/2 Group III rats. There was also one sarcoma in the latter. Th incidence of malignancy in Group I was significantly highe ($p < 0.05$) than that in Group II. Thus, duodenogastric reflu increases the incidence of cancer in the resected stomach an small intestine of rats. Patients who have had an anastomos that permits this type of reflux should be considered high-ris and should be followed with x-ray and endoscopic stud Surgery for stomach ulcers should employ techniques whic do not allow duodenogastric reflux. (10 refs)

78-4127 **Hypercalcemia Associated with Uroepithelia Neoplasms.** (Eng) Mandell, J. (Dept. Surgery Div. Urology, Univ. North Carolina Sch. Medicine, Chape Hill, NC); Magee, M. C.; Fried, F. A. *J Urol* 119(6): 844-845 1978.

Three recent cases of transitional cell carcinoma associated with significant hypercalcemia are described. Each of the patients (2 men 57 and 66 yr old, 1 woman 60 yr old) had histologically proved transitional cell carcinoma of the bladder and/or renal pelvis, markedly elevated serum calcium levels, a negative bone scan, and elevated parathormone levels. In the one patient in whom surgical extirpation was possible, there was an immediate and dramatic decrease in serum calcium to a normal level. Hypercalcemia without known bone invasion has previously been documented in 2 cases of transitional cell tumor, 2 cases of epidermoid carcinoma of the bladder, and 3 cases of bladder carcinoma, histologic cell type unknown. The synthesis of a parathormonelike polypeptide by the neoplastic tissues appears to be the most likely mechanism for the production of hypercalcemia in all of these cases. The hypercalcemia of neoplastic disease is more severe than that of primary hyperparathyroid disease. (10 refs)

78-4128 **Starch Granuloma of the Urinary Bladder.** (Eng) McLean, P. A. (Dept. Urology, Jervis St. Hosp., Dublin 1, Ireland); Doyle, G. D.; Komaranchat, A. *Ir Med J* 71(1): 13-14; 1978.

A starch granuloma of the urinary bladder occurred in a 9-yr-old girl as a result of tissue contamination by the corn starch used as a surgical glove lubricant during a bilateral ureteroneocystostomy. A prominent granulomatous reaction was found 3 wk later upon biopsy prompted by pyrexia, bladder pain, and urinary leakage through the wound. (18 refs)

78-4129 **Chromosome Constitution of Gestational Trophoblastic Disease (Letter to Editor).** (Eng) Jacobs, P. A. (Dept. Anatomy and Reproductive Biology, Univ. Hawaii Sch. Medicine, Honolulu, Hawaii, 96822); Hassold, T. J.; Matsuyama, A. M.; Newlands, I. M. *Lancet* 2(8079): 49; 1978.

The chromosome set of cells from four patients with trophoblastic disease (3 hydatidiform moles, 1 possible malignancy) was studied. The first three patients had a 46,XX chromosome set, and they responded well to therapy. The fourth patient had an extremely abnormal karyotype (none of the trophoblasts had 46 chromosomes), and the cells were clearly androgenetic in origin. Although the cytogenetic evidence might indicate clinically over malignancy, there was no evidence of frank malignancy. Sections contained a mixture of normal- and abnormal-appearing villi. (no refs)

78-4130 **Vestigial Cells in the Endoexocervical Transition Zone. Optical and Ultrastructural Study.** (Fre) Hoang-Ngoc Minh (Laboratoire d'Anatomie Pathologique, C.H.U. d'Amiens, Amiens, France); Lecomte, D.; Coupez, F.; De St Maur, P. P.; Smadja, A.; Orcel, L. *Gynecologie* 28(6): 523-527; 1977.

A light and electron microscope study was made of tissue samples from the cervix of normal women, free of cervical lesions, in their reproductive period. Biopsies were taken from the exocervical periorificial ring, and all zones of typical or atypical cicatrization were eliminated from study. The endoexocervical transition zone is not constant in size (range 1-10 mm) around the cervix. Using colposcopy with fluorescent dyes, the islands of vestigial cells could be seen as tiny iodonegative plaques. Light and electron microscopy identified these cells as malpighian, which relates them to the germinal cells of the basal layer of the exocervical malpighian epithelium. The vestigial cells are "sandwiched" between the malpighian epithelium and the cylindrical cell epithelium of mullerian origin. They are the vestigial elements of the cranial portion of the epithelial layer of sinus origin that contributed to the formation of the exocervical and vaginal malpighian epithelium. It is hypothesized that these cells are usually quiescent but under the influence of certain stimuli, they will proliferate, giving rise to cervical dysplasia or carcinoma in situ. (22 refs)

78-4131 **Adipose Tissue and Aetiology of Breast Cancer (Letter to Editor).** (Eng) Adami, H. O. (Dept. Surgery, Univ. Hosp., S-750 14 Uppsala, Sweden); Rimsten, A. *Lancet* 2(8091): 677-678; 1978.

The results of a case-control study of 179 unselected breast cancer patients and 179 age-matched controls do not support the hypothesis that the amount of adipose tissue, as reflected in breast size, is related to breast cancer risk. However, mean serum levels of estrone and androstenedione were higher in 122 postmenopausal breast cancer patients than in age-matched controls. There was no quantitative influence of adipose tissue on these levels. (1 ref)

78-4132 **Cancer Risk in Patients with Congenital Polycystic Kidney.** (Ita) Ribacchi, R. (Universitario di Medicina, Perugia, Italy); Berardi, C. *Lav Ist Anat Istol Patol (Peru* 3(1): 5-16; 1978.

From 1947 to 1977, 45 cases of congenital polycystic kidney (CPK) were found in an autopsy series of about 12,000 at Pisa University Hospital, Pisa, Italy. Tumors were found in 6/45 cases, without any sex bias. Median age at death was 71 yr among the 6 tumor-bearing patients and 57 yr among the 39 tumor-free patients. Analysis of these 45 cases and the 196 cases reported in the literature showed that the tumors usually develop in subjects > 50 yr of age and that they are preferably localized to the esophagus, stomach, rectum, bile ducts, kidney, and urinary bladder in men and to the ovaries, breast, and external genitals in women. The findings indicate a relationship between CPK and tumors. Compared with the general population, tumor risk is increased 23-fold in men with CPK and 12-fold in women with CPK (25-fold in men and 28-fold in women in the present series). Immunosuppression, possible exposure to endogenous carcinogens, and, especially, a genetic factor are hypothesized as the possible causes of the significantly increased tumor incidence in CPK patients. (22 refs)

78-4133 Condyloma Acuminata and Cancer of the Vulva, Vagina, and Penis. (Ger) Schmauz, R. (Abteilung fur Pathologie der Med. Hochschule, Ratzeburger 160, 2400 Lubeck, E. Germany); Elsasser, E.; Lalwak, A.; Cordes, B. *Geburtshilfe Frauenheilkd* 38(5): 342-351; 1978.

Among biopsy specimens examined in Lubeck, East Germany, between 1947 and 1976, condyloma acuminata (CA) were associated with 11/21 vaginal carcinomas, 50/71 vulvar carcinomas, and 10/49 penile carcinomas. In most cases, the CA preceded the carcinomas by decades; in the vulva, however, there was a second group of lesions that preceded by the carcinomas by only a few years. There were four cases of mixed forms showing various stages of malignant degeneration of CA. Additional lesions seen were Bowen's disease, giant condyloma, verrucous carcinoma, and overt squamous cell carcinoma. Case reports recording malignant transition after 20-30 yr and geographic correlations observed in Uganda support the concept that CA occurring in the young are precancerous lesions with a low risk of malignant transformation. Accordingly, CA occurring in older patients could be precancerous lesions with a relatively high risk of transformation. Nonetheless, the risk may be independent of age factors in a small percentage of CA patients. It is suggested that variants between acuminata and giant condyloma should be termed condyloma only. Additionally, the term dysplastic should be used as an indication of the high risk associated with these variants. The possibility that the condyloma virus is involved in the etiology of genital cancer is discussed. (28 refs)

78-4134 Rapid Development of Malignant Metastases in the Abdominal Wall after Laparoscopy. (Eng) Dobronte, Z. (First Dept. Medicine, Univ. Medical Sch., H-6701 Szeged, Hungary); Wittmann, T.; Karacsony, G. *Endoscopy* 10(2): 127-130; 1978.

A bilateral cystadenocarcinoma of the ovary in a 52-yr-old woman metastasized rapidly to the wounds of the abdominal wall made by laparoscopy. The metastases were generated by penetration of ascites containing tumor cells into the wound of the parietal abdomen. (10 refs)

78-4135 Second Primary Neoplasms Following Ovarian Cancer. (Eng) Reimer, R. R. (Dept. Hematology and Medical Oncology, Cleveland Clinic Foundation, 9500 Euclid Ave., Cleveland, OH, 44106); Hoover, R.; Fraumeni, J. F.; Young, R. C. *J Natl Cancer Inst* 61(5): 1195-1197; 1978.

Data on second primary neoplasms in women treated for ovarian cancer were obtained from the end results progra (ERP) of the National Cancer Institute, which include 13,309 ovarian cancer patients treated between 1935 an 1972, and from a survey of 5,455 patients treated with a kylating agents between 1970 and 1975 at 70 institutions. Th expected numbers of second cancers in the ERP group ca culated by applying age-, sex-, and time-specific incidenc rates from the general population to the corresponding pe son-years of follow-up in the ovarian cancer patients (rate used were from the Connecticut tumor registry). The expec ed numbers of second cancers in the other group were ca culated by applying age- and sex-specific incidence rates o tained in the Third National Cancer Survey to the numbe of person-years at risk. In both groups, there was an increase risk of cancer of the uterine corpus, colon, bladder, breas and hemopoietic system. The excess risk of uterine corpu cancer was independent of therapy. The risk of colon cance was increased in all treatment groups but was particular high among patients who had received radiation or chemoth erapy. The predisposition to other neoplasms was limited certain treatment groups: bladder cancer to radiation, le kemia to chemotherapy, and lymphoma to radiation chemotherapy. Thus, the pattern of second neoplasms follo ing ovarian cancer appears as common etiologic factors. (1 refs)

See also:
*(Rev.): 78-3607, 78-3617, 78-3623, 78-3651, 78-365
78-3663, 78-3666, 78-3667, 78-3669.
*(Chem.): 78-3688, 78-3691, 78-3694, 78-370
78-3703, 78-3704, 78-3708, 78-371
78-3713, 78-3744, 78-3774, 78-377
78-3779, 78-3786, 78-3792, 78-379
78-3794, 78-3795, 78-3811, 78-381
78-3814, 78-3821, 78-3829, 78-383
78-3834, 78-3835, 78-3836, 78-384
78-3866, 78-3894, 78-3902, 78-390
*(Phys.): 78-3908, 78-3918, 78-3922, 78-392
78-3925, 78-3929, 78-3932, 78-393
78-3936, 78-3940.
*(Viral.): 78-3954, 78-3978, 78-4043, 78-404
78-4049.
*(Immun.): 78-4062, 78-4067, 78-4071, 78-407
78-4081.
*(Epid.-Biom.): 78-4142, 78-4147, 78-4152, 78-415
78-4163, 78-4168, 78-4181.

78-4136 **Childhood Cancer in Sweden, 1958-1974. I. Incidence and Mortality.** (Eng) Ericsson, J. L. (Central Lab. Clinical Pathology, Univ. Hosp., Univ. Uppsala, Uppsala, Sweden); Karnstrom, L.; Mattsson, B. *Acta Paediatr Scand* 67(4): 425-432; 1978.

Analyses were made of all cases of tumors and tumor-like conditions in children 0-14 yr old reported to the Swedish cancer registry during 1958-1974. The material consisted of 3,797 individuals. The most common cancer diagnoses were leukemia and CNS tumors, both of which constituted approx 58% of all cases. Almost half of the cancers affected children < 5 yr old. The lowest incidence occurred at age 7-8 yr, the highest during the first year of life. In addition to neuroblastomas, Wilms' tumors, and retinoblastoma, certain uncommon tumors occurred at a high frequency during the first year of life. These included primary liver tumors, vulvovaginal tumors, and reticuloses. During 1958-1974, the incidence of childhood cancer increased significantly but the mortality rates decreased slightly. Most of the increase was due to the increased incidence of nervous system tumors, especially in boys (sex ratio, 3:1). The decrease in mortality rates was most obvious with respect to Wilms' tumor, and leukemia in children 0-4 yr old. (13 refs)

78-4137 **Epidemiological Aspects of Gastric Cancer in Costa Rica.** (Spa) Miranda, M. (No affiliation given); Macaya, J.; Moya de Madrigal, L. *Acta Med Costarric* 20(3): 207-214; 1977.

An epidemiological study was made of 1,426 patients with gastric cancer diagnosed in Costa Rica between 1969 and 1973. Most of the patients were men (1,002), and most were from a low socioeconomic class (64.3%); 34.57% had a middle-class background, 1.05% an upper-class background. An unusual finding was that 80 of the patients were < 29 yr old; however, approx 75% of the men and 72% of the women were 40-69 yr old. Positive diagnoses were arrived at by combinations of radiography, endoscopy, laparotomy, and biopsy. The incidence of gastric cancer was highest in patients from impoverished, rural, mountainous areas; the incidence was much lower in urban areas and coastal settlements. It was noted that two towns in which the principal occupation was dairy farming had relatively low incidences. A few towns had markedly lower or higher incidences than those of neighboring towns; no apparent cause could be found for these variations. Analysis of the diet of Costa Ricans in relation to the geographical variations revealed that in areas with the highest levels of gastric cancer, the diet is high in carbohydrates and low in animal and vegetable proteins. (8 refs)

78-4138 **Sheep Tumours in North East Yorkshire. I. Prevalence on Seven Moorland Farms.** (Eng) McCrea, C. T. (Veterinary Investigation Centre, West House, Thirsk, Yorkshire, England); Head, K. W. *Br Vet J* 134(5): 454-461; 1978.

The results of a mortality survey covering sheep on seven farms situated in northeast Yorkshire are reported. Between 1964 and 1977, 86 tumors were found in 62 sheep, an unusual geographical cluster of high prevalence. However, these figures may be underestimates, as a large number of sheep > 2 yr old died each yr, but only one-fourth of these were available for postmortem examination. In addition, several animals were sold and not traced further. There were 45 fibrosarcomas (41 in the jaw); 14 papillomas in the rumen; 7 squamous cell carcinomas (6 in the upper alimentary tract), 14 adenocarcinomas of the intestine, 3 primary carcinomas of the liver, 2 localized lymphosarcomas, and 1 epithelial thymoma. The percentage of tumors found at routine disease investigations in the rest of north Yorkshire during 1960-1968 was 0.9% in 2,398 carcasses. (23 refs)

78-4139 **Cancer Incidence in 1975 in the French Region of Bas-Rhin.** (Fre) Schaffer, P. (Institut d'Hygiene et de Medicine Preventive, 4,rue Kirschleger, F67085 Strasbourg Cedex, France); Cayemittes, P. A.; Arfeux, F.; Kuntzmann, Y. *Bull Cancer (Paris)* 65(1): 9-18; 1978.

Data from a cancer registry established in 1975 in the Bas-Rhin region of France (population 883,000 persons) are reported. A total of 3,073 cancer cases were registered, with an incidence of 424/100,000 men and 309/100,000 women. Lung cancer was the most frequent cancer (17.1%) among men, followed by cancer of the rectum and colon (7.9%), cancer of the prostate (7.6%), and cancer of the stomach (6.0%). In the female population, breast cancer was the most frequent type (27.5%), followed by cancer of the uterus (9.3%) and cancer of the cervix (6.8%). The av age at diagnosis was 63.5 yr in men and 62.7 yr in women; 95% of the cancers were diagnosed between the ages of 35-84 yr in men and 26-84 yr in women. The incidence of the most frequent cancers in the men and women of Bas-Rhin are compared with that reported in registries in Geneva, Sarre, Birmingham, Norway, and Connecticut. (5 refs.)

78-4140 **Cancer Mortality During the Last Twenty-five Years in Italy on Sardinia, with Special Regard to the City of Cagliari.** (Ita) Meloni, F. (Insituto Igiene, Cagliari, Italy); Dodero, G.; Contu, A. *Ig Mod* 70(1): 33-49; 1977.

Cancer mortality statistics are presented for Italy, Sardinia, and Cagliari, the capital of Sardinia. Overall cancer mortality (per 100,000 inhabitants) has increased from 52.77 in 1901 to 73.68 in 1931, 150.61 in 1960, and 192 in 1972. Mortality is highest in the densely populated and industrialized northern and central provinces and lowest in the underdeveloped southern provinces, being lowest in Calabria (98.7 in 1972) and highest in Emilia Romagna (289.5 in 1972). In 1951, mortality was 88 in Cagliari and 66.8 in Sardinia, vs 99.6 and 127.4, respectively, in 1971, a definite increase. In 1970, mortality in Sardinia and Cagliari was above the national for malignant tumors of the liver, bile ducts, trachea, and bronchi; mortality due to tumors of other organs was below the national av. Mortality was significantly lower among men than among women. The findings indicate an increase in cancer mortality in Sardinia and Cagliari that is parallel to the national trend. It may be related to the industrialization of the island. (no refs.)

78-4141 **Case-Control Study of Antihypertensive and Diuretic Use by Women with Malignant and Benign Breast Lesions Detected in a Mammography Screening Program.** (Eng) Williams, R. R. (Dept. Internal Medicine, Univ. Utah Medical Center, Salt Lake City, UT, 84132); Feinleib, M.; Connor, R. J.; Stegens, N. L. *J Natl Cancer Inst* 61(2): 327-335; 1978.

A case-control study of the association between breast cancer and the use of antihypertensive and diuretic drugs was conducted on subjects selected from among 80,000 women screened in a national mammography screening project. The study subjects consisted of 481 newly identified breast cancer patients group-matched with 1,268 controls and an additional case group of 421 women with benign breast lesions. Data were obtained by questionnaires sent to subjects and attending physicians. A significant but low-level association was revealed between breast cancer and the use of rauwolfia derivatives (reserpine) for ≥5 yr. This association was confined to women > 50 yr old who were also heavier than av. No confounding effects could be held responsible for this association after adjustment for variables such as presence of hypertension, wt, age at first pregnancy, and other breast cancer risk factors. Other antihypertensive and diuretic drugs as well as multiple drug use also showed some association with breast cancer (chlorthalidone, hydralazine, furosemide). There was a significant association between benign breast lesions and use of thiazides (for edema or hypertension) for ≥ 5 yr. Reserpine seemed to have a protective effect against breast cancer in short-term users, a finding consistent with observations in rats. These and previous data suggest serious consideration of long-term reserpine use as a cause of breast cancer. (16 refs.)

78-4142 **An Epidemiological Study of the Relationship Between Benign Breast Diseases and Breast Cancer (Meeting Abstract).** (Eng) Coombs, L. J. (John Hopkins Univ., Baltimore, MD, 21218) *Diss Abstr Int* 39(4): 1713-1714; 1978. (no refs.)

78-4143 **Epidemiological Aspects of Breast Carcinoma** (Rus) Chapliuk, M. I. (P. A. Hertsen Res. Oncological Inst., Moscow, USSR). *Khirurgiia (Mosk)* 4: 12-15 1978.

In an attempt to explain the heterogeneity of breast carcinoma incidence, 189 patients with mammary gland cancer and 137 healthy control women were asked to answer a specially devised epidemiologic questionnaire. The questionnaire contained > 350 questions on nationality, age, sexual history pregnancies, delivery, duration of lactation, etc. The only significant difference between the two groups concerned the lactation period: 28.94% of the women with breast carcinoma had lactation periods > 1 yr, compared with only 17.55% of the controls. (10 refs)

78-4144 **Estrogen Therapy and Endometrial Carcinoma** (Ger) Volker, W. (Frauenklinik der Medizinischen Hochschule Hannover, Pasteurallee, D-3000 Hannover 51, W. Germany); Kannengiesser, U.; Majewski, A.; Vasterling, H. W. *Geburtshilfe Frauenheilkd* 38(9): 735-743; 1978

The possible carcinogenic effect of estrogen compounds was studied in 130 women with endometrial carcinoma (EMC) (118 FIGO Stage I, 9 Stage II, 3 Stage III cases) and in matched controls. Matching traits were age at diagnosis and age at menopause, number of children and abortions, weight presence of diabetes, participation in cancer screening programs, educational level, use of heart drugs, antihypertonics or sedatives, blood pressure, and histological findings in addition to EMC (eg, glandular hyperplasia). All controls had been treated by curettage or hysterectomy for bleeding disturbances. All patients were questioned about estrogen usage and the relative risk (RR) was calculated by the method of Mantel and Haenszel. The RR found for the use of any estrogen was 0.44 ($p < 0.01$), and similar risks were found if the estrogens were separated into types (eg, conjugated, estriol, estradiol). The RR for estrogen-androgen mixtures was 1.28 ($p > 0.05$), that for inhibitors of ovulation, 0.1 ($p < 0.01$). There was no increased risk associated with duration of estrogen usage. Childless women who used estrogens had an RR of 0.34 ($p < 0.05$), users with high blood pressure had an RR of 0.23 ($p < 0.01$), and users who were obese had an RR of 0.30 ($p < 0.001$), indicating that estrogen usage may be protective for patients with these other known risk factors. The careful matching of the control group in this study may explain the finding of no increased risk of EMC due to estrogen usage. Several previous studies have shown an increased risk, but recent publications have indicated that as control matching improves, the risk decreases. (41 refs)

78-4145 **Urinary Tract Lesions and Analgesic Abuse.** (Ger) Brunner, F. P. (Departement fur Innere Medizin, Kantonsspital, CH-4031 Basel, Switzerland); Richtmann, L.; Thiel, G.; Dubach, U. C. *Schweiz Med Wochenschr* 108(27): 1013-1019; 1978.

A review of the records of the University Hospital in Basel, Switzerland, indicated that 20 patients with analgesic nephropathy and tumors of the urinary tract (excluding hypernephroma) were admitted between 1966 and 1976. There were 17 uroepithelial tumors, 2 sarcomas, and 1 chorioepithelioma. Phenacetin, a common ingredient of analgesic mixtures, is regarded as being carcinogenic on the basis of the structural similarity of some of its metabolites to industrial carcinogens and the results of animal experiments. Malignant tumors have been induced in animals by the 2-hydroxylated metabolites and arene oxides of phenacetin and paracetamol. There is even more convincing evidence of the carcinogenicity of the N-oxidized metabolites, such as N-hydroxyphenetidine and N-hydroxyphenacetin. Comparison of analgesic mixtures used in Australia and Sweden (countries with high rates of analgesic abuse) suggests that dimethylnitrosamine formed from aminophenazone does not play a role in the development of uroepithelial tumors. (46 refs)

78-4146 **Sperm Basic Proteins in Cervical Carcinogenesis (Letter to Editor).** (Eng) Issidorides, M. R. (Marine Biological Lab., Woods Hole, MA, 02543). *Lancet* 2(8091): 688; 1978.

Contradictory results of two studies relating sperm basic proteins to socioeconomic status and, in one, to cervical cancer may be due to the fact that subjects in one study were not screened for the use of psychotropic drugs, which reversibly increase, at least in peripheral blood cells, certain histone basic protein fractions. (2 refs)

78-4147 **Cancer of the Cervix--A Sexually Transmitted Disease. Cytologic Screening in a Prostitute Population.** (Eng) Sebastian, J. A. (Duluth Clinic, Ltd., 400 E. Third St., Duluth, MN, 55805); Leeb, B. O.; See, R. *Am J Obstet Gynecol* 131(6): 620-623; 1978.

Cervical cytology was performed at the time of required gonorrhea screening in 750 registered prostitutes in Taipei, Taiwan to determine whether cervical cancer can be transmitted sexually. This population was unique because the variables frequent intercourse with multiple partners and frequent infections could be separated from early intercourse (before the age of 18), which is socially and morally unacceptable in Taiwan. A clinical data sheet concerning age, length of employment, frequency of intercourse, contraception, and pregnancy history also was completed. The cytologic results were: normal in 253, mild inflammation in 426, inflammatory atypic in 33, dysplasia in 7, and suspicious in 1. Suspicious or positive cytology was found in only 8/750 prostitutes, for a rate of 10.7/1,000. This low yield of abnormal cytology suggests that early coital activity is the most important epidemiologic risk factor in the development of cervical cancer and that frequent intercourse with multiple partners and multiple infections are not important epidemiologically if they occur after the period of active metaplasia found during adolescence or during the first pregnancy. (21 refs)

78-4148 **Sperm Basic Proteins in Cervical Carcinogenesis: Correlation with Socioeconomic Class.** (Eng) Reid, B. L. (Dept. Obstetrics and Gynaecology, Univ. Sydney, New South Wales 2006, Australia); French, P. W.; Singer, A.; Hagan, B. E.; Coppleson, M. *Lancet* 2(8080): 60-62; 1978.

An attempt was made to obtain circumstantial evidence for sperm basic proteins as carcinogens in squamous cervical cancer by relating them to social class and to epidemiological variables thought to be correlated with the onset of the disease. These variables are the incidence of veneral disease and the precedence of carcinoma in situ, both of which are highly correlated with social class. Single ejaculates were obtained from 202 Australian and English men, mostly outpatients at infertility clinics, but some healthy students. Each individual was assigned to one of five social classes. Two types of basic protein, a histone and a protamine, were separated from the ejaculates, and a ratio between their contents in a given number of sperm was established. The ratio varied widely among the men and it correlated directly with social class: the lower the class, the greater the proportion of protamine. Statistically similar correlations link social class with the incidence of venereal disease and of carcinoma in situ. Thus, the basic proteins of the sperm head, especially the protamines, may also have a role in the etiology of squamous cervical cancer. (27 refs)

78-4149 **Risk of Cervical Cancer among an Electrocoagulated Population.** (Eng) Kauraniemi, T. (Finnish Cancer Registry, Inst. Statistical and Epidemiological Cancer Res., Liisankatu 21B, SF-00170 Helsinki, Finland); Rasanen-Virtanen, U.; Hakama, M. *Am J Obstet Gynecol* 131(5): 533-538; 1978.

Data from a nationwide mass screening registry plus data on malignant cervical lesions reported to the Finnish Cancer Registry, both covering the years 1963-1972, were used to assess the risk of cervical lesions among women who had been subjected to electrocoagulation of the uterine cervix (EC group) and among the rest of the Finnish female population (N-EC group). During this period, 429,832 women were screened, 67,321 of whom had undergone EC. The procedure was performed mainly in women 25-34 yr old (56,909 subjects) and in married women (60,884). Compared with the EC group, N-EC group had a sixfold risk of frankly invasive carcinoma, a fourfold risk of carcinoma in situ or dysplasia gravis, and a twofold risk of dysplasia levis. The diagnostic accuracy of cytologic tests, compared with the histologic malignancy of the lesion, was better in the EC group. For unmarried women, the total risk of premalignant and malignant lesions was the same in both groups. However, the total risk of any cancerous lesion was approx four times greater among those N-EC than among those EC in the other marital groups (married, widowed, divorced). The frequency of *Trichomonas vaginalis* infection (TVI) was higher among all N-EC women, including healthy subjects and cancer patients. The prevalence rates (per 100,000) of premalignant and malignant lesions by EC treatment and by occurrence of TVI were: 1,193 (N-EC + TVI), 535 (N-EC - TVI), 394 (EC + TVI), and 138 (EC - TVI). The potential effect of EC in reducing cervical cancer may exceed the effect of organized mass screening programs. (16 refs)

78-4150 **Cause of High Risk of Cervical Cancer in Socially Unclassified Women.** (Eng) Sibary, K. (Dept. Social Res., Univ. Hosp. S. Manchester, Manchester, England); Burslem, R. W.; Wakefield, J. *Br J Cancer* 38(1): 166-168; 1978.

The social class distribution of women with abnormal cervical smears who had not previously been allocated to a specific social class because of incomplete data was determined. Previous comparisons of the percentage of abnormal smears in the various classes had indicated that the unclassified women had a higher rate of suspicious and positive smears than the classifiable women. Questionnaires were sent to a random sample of 300/71,000 unclassified women; 235 acceptable replies were received. Distribution of these 235 women by social class showed only small and insignificant differences from the distribution of all classifiable women with abnormal smears screened between 1971 and 1976. However, significant differences in distribution by marital status and age were apparent. The 235 women included a significantly greater proportion of widows and divorcees (15%) than the remainder of the screened population (3.1%), and significantly more women were > 60 yr old (6.9% vs 2%) or < 25 yr old (40% vs 18.4%). Thus, the excess of abnormal smears in the unclassified women was determined mainly by the large proportion of widowed/divorced and elderly women (high-risk groups). (6 refs)

78-4151 **Incidence of Cervical Cancer by Marital Status.** (Eng) Leck, I. (Regional Cancer Epidemiology Unit, Univ. Hosp. S. Manchester, Manchester M20 9BX, England); Sibary, K.; Wakefield, J. *J Epidemiol and Community Health* 32(2): 108-110; 1978.

The incidence of invasive cervical cancer by age and marital status was examined using census statistics and 1968-1971 cancer registry data for women who lived in Manchester, (England) Regional Hospital Board area. The disease was
relatively rare in the unmarried. Formerly married wome
were at a considerably higher risk than married women. Th
incidence was highest at the ages of 40-49 for widows an
40-59 for divorced women. The standardized incidence rati
was 0.42 for single, 1.04 for married, 1.12 for widowed, an
1.66 for divorced women. It is suggested that the higher inci
dence rate in divorced women and young widows is relate
to the association of cervical cancer with number of sexua
partners. (15 refs)

78-4152 **Paroxysmal Nocturnal Haemoglobinuria i
Thailand with Special Reference to an Associa
tion with Aplastic Anaemia.** (Eng) Kruatrachue, M. (Di
Haematology, Dept. Medicine, Siriraj Hosp., Mahidol Univ
Bangkok 7, Thailand, Wasi, P.); Na-Nakorn, S. *Br J Ha
ematol* 39(2): 267-276; 1978.

The characteristics of 85 cases of paroxysmal nocturna
hemoglobinuria (PNH) in Thailand were compared wit
those of 80 European (Eur) cases, and the association of thi
disease with aplastic anemia (AA) was explored. Thai PN
patients tended to be younger (56 were < 30 yr old) than th
Eur patients (only 25 were < 30 yr old), and 23 of the Tha
patients were women, compared with 47 in the Eur series
The clinical presentations of Thai PNH were generally indis
tinguishable from those of the Eur cases, except that com
plications attributable to arterial and venous thrombosis wer
rare in the former (2 patients); they are reportedly commo
in the latter. Analysis of 232 Thai cases of AA revealed tha
the age of onset was similar to that of PNH, but the propor
tion of women was higher (103/232). An aplastic marro
could be found in cases diagnosed initially as PNH or subse
quently, during the course of illness diagnosed initially a
AA. Both groups had the same clinical symptoms, and bot
had features of AA. Cases diagnosed primarily as AA\"com
plicated" by PNH could be divided into two categories; (I
after various intervals, patients enter a remission of severa
years' duration that is followed by typical PNH; (II) the clas
sical picture of AA dominates the entire course of the disease
and there is no remission period. AA dose not precede PN
in all cases. Of the 232 AA patients, 47 were found to b
positive for PNH RBC in an initial test, but not in repeate
tests. According to the somatic mutation theory, the associa
tion of PNH and AA might be based on the occurrence o
abnormal RBC clone(s) in the bone marrow in one of severa
ways. The factor involved in promoting or eliminating thi
clone seems to be sex-related, and it might be ethnic in origin
(18 refs)

78-4153 **Epidemiologic Assessment of the Viral Hypoth-
esis of Human Lymphomas and Leukemias**
(Eng) Khokhlova, M. P. (Lab. Leukemia Epidemiology an
Histopathology, Central Inst. Haematology and Bloo
Transfusion, Moscow 125167, USSR); Osechinskiy, I. V.
Martirosov, A. R.; Yashanova, N. D. *In: Advances in Com*

parative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977. Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 269-270; 1978.

Leukemia and lymphoma incidence in the rural populations of the Lithuanian SSR and the Minsk region of the USSR was studied to determine if there is a viral etiology for human hemoblastoses in these areas. Knox analysis of the data revealed a rise in probability of the appearance of a second case of the disease within 4.5 km of a given case within 1.5 yr. However, there was no increase in human hemoblastoses as a result of residence near dairy farms or resulting from contact with cattle. (no refs)

78-4154 **Familial Leukemia.** (Fre) Schmitt, T. A. (Institut de Recherchde sur les Maladies du Sang, Hopital Saint-Louis, 2,place du Dr. Fournier, F75010 Paris, France); Degos, L. *Bull Cancer (Paris)* 65(1): 83-88; 1978.

Literature on the genetic factors in acute leukemia is reviewed, and a study of familial incidence among 2,966 patients is reported. In the series of 2,966 patients, there were 26 patients from families with more than one affected member. The relative risk for a sibling of a leukemic patient is four times greater than that for the population at large. Parents of leukemic patients may also run a greater risk of developing the disease. The leukemia was usually of the same type in patients from the same family, and the onset of the disease occurred approx at the same age. There were 2 pairs of identical twins with the disease among the patients in this series and 28 pairs of twins in the literature, 13 pairs that were definitely identical and 5 that were probably so. The relative risk of a twin developing leukemia decreases as the age at onset in the affected twin increases. (20 refs)

78-4155 **Subsequent Cancer in Patients with Chronic Lymphocytic Leukemia--A Possible Immunologic Mechanism.** (Eng) Greene, M. H. (Environmental Epidemiology Branch, NCI, NIH, Bethesda, MD, 20014); Hoover, R. N.; Fraumeni, J. F. *J Natl Cancer Inst* 61(2): 337-340; 1978.

The occurrence of a second primary cancer was examined among 4,869 white patients (3,094 men, 1,775 women) with chronic lymphocytic leukemia (CLL) diagnosed between 1935 and 1971. A second primary cancer developed in 234 CLL patients compared to 204.9 expected (Exp). The difference was due primarily to excesses of malignant melanoma [9 observed (Obs) vs 1.34 Exp], soft-tissue sarcomas (7 Obs vs 1.33 Exp), lung cancer (33 Obs vs 40.6 Exp). For several other sites (lip, salivary glands, tongue, Hodgkin's disease, and non-Hodgkin's lymphoma) the risk exceeded expectation, but the numbers were too small for further analysis. For the four main excess cancer sites, the excess risk affected both men and women, untreated and treated patients, and patients at all stages of follow-up. The finding of an excess risk throughout the follow-up period suggests an intrinsic association with CLL rather than an effect of methodology, diagnosis, or therapy. There was no excess risk of leukemia following the diagnosis of these second primary cancers, indicating a one-way association. This argues against a common etiology and suggests that a susceptibility state associated with CLL may underlie the development of a second primary cancer. These and similar previous data suggest that certain nonlymphoid tumors may result from impaired immunologic surveillance. (22 refs)

78-4156 **Cancer Incidence Amongst Cosmetologists (Meeting Abstract).** (Eng) Walrath, J. (Yale Univ., New Haven, CT, 06520). *Diss Abstr Int B* 39(4): 1714; 1978. (no refs)

78-4157 **A Case-Control Study of Hodgkin's Disease in Israel.** (Eng) Abramson, J. H. (Dept. Social Medicine, Hebrew Univ.-Hadassah Medical Sch., P.O. Box 1172, Jerusalem, Israel); Pridan, H.; Sacks, M. I.; Avitzour, M.; Peritz, E. *J Natl Cancer Inst* 61(2): 307-314; 1978.

A case-control study was made of 473 Jewish residents of Israel diagnosed as having Hodgkin's disease (HD) in 1960-1972 and matched controls drawn from the general population. There was a significant association between parental consanguinity and HD (14.1% of patients vs 8.4% of controls). Among men, the case-control difference was highly significant and the estimated relative risk of HD was 4.2, whereas among women the association was weaker and statistically nonsignificant. Associations between HD and a high educational level and the presence of a flush toilet and piped water in the childhood home were significant, giving limited support to the hypothesis that a high standard of living in childhood increases the risk of subsequent HD. Women with HD tended to have a lower parity than did their controls. Tonsillectomy and a history of work with wood or trees were significantly associated with mixed cellularity but not with other histologic subtypes. A weaker relationship was found between appendectomy and nodular sclerosis. Differences between HD patients and controls with respect to cigarette smoking, exposure to animals, marital status, previous blood transfusions or jaundice, contact with asbestos, and residental or occupational mobility were not significant. (37 refs)

78-4158 **Familial and Sporadic Hodgkin's Disease Associated with Occupational Wood Exposure (Letter to Editor).** (Eng) Greene, M. H. (Environmental Epidemiology Branch, NCI, Bethesda, MD, 20014); Brinton, L. A.; Fraumeni, J. F.; D'Amico, R. *Lancet* 2(8090): 626-627; 1978.

Occupational exposure to wood may have contributed to a familial concentration (3 sibs and a first cousin) of Hodgkin's disease. Two of the brothers had been employed (for 12 and 15 yr) by a cedar fence installation company, where they prepared and applied the wood preservative pentachlorophenol (PCP). One brother had high levels of PCP in serum (1,299 ppb) and urine (90 ppb). The other two afflicted family members had no such exposure. One unrelated employee of the same company also developed Hodgkin's disease. The fact that only 2/4 family members were occupationally exposed suggests that there is an interplay between genetic and environmental determinants. Occupational statements on death certificates were examined in North Carolina counties where furniture manufacturing and lumbering are common. Each of the 167 cases of Hodgkin's disease found between 1956 and 1974 were matched with two controls. An excess risk (1.4) was found only among occupational groups with wood and paper exposure. The major difference occurred for carpentry and lumbering, with a relative risk of 4.2. Use of PCP is common in many phases of wood processing. It is therefore suggested that the association between occupational wood exposure and Hodgkin's disease may be related to the use of particular wood preservatives. (12 refs)

78-4159 **Infectious Mononucleosis and Hodgkin's Disease.** (Eng) Munoz, N. (International Agency Res. Cancer, Lyon, France); Davidson, R. J.; Witthoff, B.; Ericsson, J. E.; de-The, G. *Int J Cancer* 22(1): 10-13; 1978.

A large follow-up study was made of 9,454 patients with serologically confirmed infectious mononucleosis (IM) to evaluate the association between IM and Hodgkin's disease (HD). The names of 1,759 patients identified in Scotland from 1959 to 1971 and 7,695 identified in Sweden from 1952 to 1970 were matched against the names of patients with lymphoma or nasopharyngeal or colon carcinoma registered through 1972 in Sweden and through 1973 in Scotland. There were 7 cases of HD among the IM patients (2 in men, 5 in women), compared with an expected number of 1.8, a fourfold increase in risk. The increased risk for HD was greater within 3 yr of IM diagnosis. These results are similar to those of four other cohort studies, and it is concluded that there is a positive association between IM and HD. Several explanations of the association are considered: some persons who are in the process of developing HD may be temporarily misdiagnosed as having IM; IM may increase the susceptibility of some individuals to a factor that causes HD; IM may promote the rapid (2-3 yr) development of HD in a subject in whom the disease is latent or progressing slowly; and IM and HD may have a common cause. (10 refs)

78-4160 **Epidemiological Features of Leukemia and Lymphoma and Other Malignant Hemopathies.** (Por) Jamra, M. (Disciplina de Hematologia do Departamento de Clinica Medica, Universidade de Sao Paulo, Sao Paul
Brazil). *Rev Braz Pesqui Med Biol* 11(1): 67-75; 1978.

Epidemiological data are presented on lymphomas and le
kemias in Brazil. Hodgkin's disease shows a unimodal distr
bution typical of underdeveloped countries. The risk
Hodgkin's disease is significantly increased among tonsille
tomized persons compared with their nontonsillectomize
relatives. In Brazil, Hodgkin's disease of lymphocy
predominance type accounts for 21.4% of cases, nodul
sclerosis for 7.2%, mixed cellularity for 64%, and lym
phocyte depletion type for 7.2%. In Sao Paulo, lymphosa
coma accounts for 10.53% of lymphoreticular tumor
reticulosarcoma for 17.2%, other lymphomas for 4.39%, le
kemias for 37.28%, Hodgkin's disease for 26.5%, Burkitt
lymphoma for 0.74%, and multiple myeloma for 3.36%
Epidemiological data are also given on the incidence of lym
phomas and leukemias in other parts of the world. (13 ref

78-4161 **Solvent Exposure and Cancer (Letter to Editor**
(Eng) Capurro, P. U. (Dept. Lab., Union Hos
Cecil County, Elkton, MD, 21921); Eldridge, J. E. *Lanc*
1(8070): 942; 1978.

Among 117 persons in a community near a chemical rec
cling plant who were heavily exposed to solvent vapors, th
number of deaths due to all cancers (1968-1974) was 5.4 time
higher than that expected. The death rate due to reticulu
cell sarcoma was 73 times greater than that expected. Th
incidence of lymphatic cancer in the entire local populatio
(4 km from plant) was 11 times greater than that expecte
Experiments with mouse skin (unpublished) indicate that th
presence of organic solvents within the body can potentia
the carcinogenic effect of cutaneously applied carcinogen
(7 refs)

78-4162 **Increased Incidence of Malignant Melanom**
after Peaks of Sunspot Activity. (Eng) Houghton, A. (Oncology Div., Dept. Medicine, Univ. Connecticu
Sch. Medicine, Farmington, CT, 06032); Munster, E. W.;
Viola, M. V. *Lancet* 1(8067): 759-760; 1978.

A review of 2,983 cases of histologically proved maligna
melanoma registered in the Connecticut Tumor Registry i
1935-1975 indicated that the age-adjusted incidence rate fo
malignant melanoma in that state has risen from 1.1/100,00
population in 1935 to 6.2/100,000 in 1975. Superimposed o
a steady rise in incidence are 3- to 5-yr periods in which th
rate of increase in incidence rises. These periods have a cycl
of 8-11 yr and follow times of max sunspot activity. In Ne
York state, like Connecticut, there were significant partia
correlations between the annual sunspot number and th
melanoma incidence rates in the following 1-2 yr. In Finlan
the increased incidence rates were significantly correlate
with the years of sunspot activity and with the first subse

uent year. The longer lag period between sunspot cycles and e cyclic increases in melanoma incidence in Connecticut d New York, compared to Finland, may be latitudinal and uld be important in constructing a mechanism of sun- ot-induced carcinogenesis. (26 refs)

8-4163 **Secondary Malignant Melanoma in Lymph Nodes: Incidence, Time of Occurrence, and ortality.** (Eng) Little, J. H. (Pathology Dept., Princess lexandra Hosp., Ipswich Road, Woolloongabba, Brisbane 102, Australia); Davis, N. C. *Aust NZ J Surg* 48(1): 9-13; 978.

atients registered in the Queensland Melanoma Project ustralia) with histologically proved secondary deposits of elanoma in the lymph nodes were studied. Of the 1,514 atients registered since 1963, 234 had metastases in their mph nodes by 1975: 146/685 men and 88/829 women. The v age for each sex was 50 yr. Among the fatal cases were 8 men and 54 women. Thirty-two patients had positive odes with no known primary lesion. Metastases developed men with lesions on the foot (50%), on the thigh (29%), nd on the back (22%) and in women with lesions on the wer leg (9%) and thigh (20%). Approx one-half of the odes were removed at the time of treatment of the primary rowth or within 2 mo; three-fourths were removed in the rst yr. However, it was found that a tumor could remain ormant for > 8 yr. On regrowth, the dormant tumors ehaved in a similar aggressive fashion as the nondormant econdary tumors. Nodal metastases were present in 5% of he patients at the time of their first presentation with prinary melanoma. Elective node dissections were performed n 6% of the men and 11% of the women. (10 refs)

8-4164 **Increased Risk of Brain Tumors in Children Exposed to Barbiturates.** (Eng) Gold, E. (Dept. pidemiology, Sch. Hygiene and Public Health, Johns Hopins Univ., Baltimore, MD, 21205); Gordis, L.; Tonascia, J.; zklo, M. *J Natl Cancer Inst* 61(4): 1031-1034; 1978.

patient-control study was conducted with children with rain tumors (BT) to investigate the possibility that the use f barbiturates, either prenatally by the mother or by the hild prior to his illness, might be associated with the development of BT in children. Interviews were conducted with 4 mothers of children with BT, and the results were comared with those obtained from 76 mothers of normal chilren and 12 mothers of children with other cancers. Mothers f children with BT more frequently reported using barbituates during their pregnancy with the index child than did nothers of normal children or mothers of children with other ancers. In addition, more children with BT were reported o have used barbiturates than normal children or children vith other malignant diseases, with most of the uses appearng to be unrelated to the symptoms resulting from the BT itself. The results suggest that barbiturates may play an etiologic role, and it is estimated that as many as 8% of BT in children may be attributable to the use of barbiturates either by the child or prenatally by the mother. (19 refs)

78-4165 **Cancer Mortality and Morbidity among Rubber Workers.** (Eng) Monson, R. R. (Dept. Epidemiology, Harvard Sch. Public Health, 677 Huntingdon Ave., Boston, MA, 02115); Fine, L. J. *J Natl Cancer Inst* 61(4): 1047-1053; 1978.

Mortality and morbidity from cancer were examined among a group of 13,570 white men who worked for at least 5 yr at an Akron, Ohio, rubber plant. The potential period of follow-up was from January 1, 1940, to June 30, 1976. Departmental work histories were based primarily on records maintained by the local union. The occurrence of cancer was measured by death certificates and by a survey of area hospital tumor registries from 1964 to 1974. Two analyses were made: (1) an external comparison of mortality rates among rubber workers vs rates among US white men, and (2) an internal comparison of morbidity rates among persons who were employed in various work areas of the plant. Excess cases of specific cancers (observed/expected numbers) among workers in specific work areas included: stomach and intestine: rubber making (30/14.4); lung: tire curing (31/14.1), fuel cells and/or deicers (46/29.1); bladder: chemical plant (6/2.4), tire building: (16/10.7); skin cancer: tire assembly (12/1.9); brain cancer: tire assembly (8/2.0); lymphatic cancer: tire building (8/3.2); and leukemia: calendering (8/2.2), tire curing (8/2.6), tire building (12/7.5), elevators (4/1.4), tubes (4/1.6), and rubber fabrics (4/1.1). Agents that may be responsible for these excesses include carbon blacks, softeners, extenders, plasticizers, antioxidants, and accelerators. Whether specific chemicals can be identified as having been directly responsible is problematic because of the complex nature of the work environment. (37 refs)

78-4166 **Further Analysis of Cancer Mortality Patterns among Workers Exposed to Cutting Oil Mists.** (Eng) Decoufle, P. (Environmental Epidemiology Branch, NCI, NIH, Bethesda, MD, 20014). *J Natl Cancer Inst* 61(4): 1025-1030; 1978.

Mortality patterns among two groups of oil mist-exposed workers at a heavy-industry machining plant were studied retrospectively, over a 30-yr period, to determine whether specific forms or sites of cancer occurred at greater than expected rates. The two groups comprised (1) 2,485 white men employed ≥5 yr in oil mist-exposed jobs between 1938 and 1967 and (2) a subgroup of 1,137 who spent at least 5 yr in jobs with heavy exposure to oil mist. The cause-specific mortality rates were compared to similar rates for the total US white male population. The total number of cancer deaths was very close to that expected. Analysis of mortality for each of 15 cancer sites showed no significant differences between observed and expected deaths for almost all comparisons. However, there was a twofold risk of cancer of the stomach and large intestine (combined) after 20 yr of follow-up in a subgroup of men with ≥5 yr exposure to cutting oil mists prior to 1938. Deaths from nonmalignant respiratory disease were significantly less than expected. It is concluded that exposure to mists of insoluble and soluble mineral oils in machining operations does not alter mortality for most cancer sites, including the respiratory system, but may affect the long-term occurrence of cancer of the stomach and large intestine. (32 refs)

78-4167 **Intestinal Metaplasia of the Stomach in Hawaii and Japan. A Study of Its Relation to Serum Pepsinogen I, Gastrin, and Parietal Cell Antibodies.** (Eng) Stemmermann, G. N. (Japan-Hawaii Cancer Study, 347 N. Kuakini St., Honolulu, HI, 96817); Ishidate, T.; Samloff, I. M.; Masuda, H.; Walsh, J. H.; Nomura, A.; Yamakawa, H.; Glober, G. *Am J Dig Dis* 23(9): 815-820; 1978.

The relation of serum pepsinogen I (PG I), fasting and postprandial gastrin, and antibodies against parietal cells to intestinal metaplasia (IMP) of the stomach was assessed in Hawaiian Caucasians (HC), Hawaiian Japanese (HJ), and indigenous Japanese (J), populations at low, intermediate, and high risk for gastric carcinoma, respectively. In addition, endoscopic mucosal biopsies were taken from parts of the stomach at minimal, max, and intermediate risk for IMP. PG I levels were low in 16.1% of the J, 9.3% of the HJ, but only 2.3% of the HC men, a finding consistent with stomach cancer risk. A low PG I level and IMP appeared to follow similar trends: each was more common among the J men, each appeared to be positively correlated with age, and each was more common in men than in women. A low PG I level was found to be highly specific for the presence of extensive IMP (162/169 patients with negative for <3 positive biopsies had normal levels) but only moderately sensitive for this condition (only 16/44 subjects with IMP at three sites had low PG I level). The absence of a low PG I level in the latter may be due to the fact that a significant portion of their oxyntic gland mucosa was not involved in the metaplasia. Neither abnormal serum gastrin levels nor the presence of parietal cell antibodies could be correlated with IMP. (19 refs)

78-4168 **Malignant Tumors in Relatives of Stomach Cancer Patients.** (Rus) Arion, A. I. (Dept. Epidemiology Malignant Tumors, Cancer Res. Center, Moscow, USSR). *Vopr Onkol* 24(7): 78-82; 1978.

The potential genetic predisposition to cancer of the stomach was studied in 223 stomach cancer patients and 3,831 relatives. Of the relatives, only 166 had any form of cancer (compared with expected incidence of cancer in the Russian population of 197.3). However, among first-degree relatives alone, the observed frequency of stomach cancer was significantly greater than the expected frequency in general population. In mothers, sisters, and daughters of female probands, stomach cancer frequency was 5.2 times the expected value. This increased frequency was not seen in male relatives of the female probands, but male and female relatives of male probands with stomach cancer were affected by the disease twice as frequently as control subjects. (14 refs)

78-4169 **Malignancies in German Thorotrast Patients and Estimated Tissue Dose.** (Eng) van Kaick, G. (Institut fur Nuklearmedizin, Deutschen Krebsforschungszentrums, Heidelberg, W. Germany); Lorenz, D.; Muth, H.; Kaul, A. *Health Phys* 35(1): 127-136; 1978.

Preliminary data on a follow-up study of 6,000 patients who had been subjected to Thorotrast (TT) examination and 6,000 controls are reported. Subjects from both groups (1,162 patients, 883 controls) who died prior to the beginning of the study (1968) were analyzed via their records. The av age at injection was 40 yr and the av time from injection to death was 17 yr. The av volume of TT injection was 30 ml. The following ratios compare the incidence of neoplastic diseases in the TT group to that in controls: liver cancer, 88/6; myeloproliferative diseases, 16/1; skeletal cancer, 2/0; lung cancer, 25/24; and spleen cancer 0/0. The absence of spleen tumors is surprising, because the radiation dose to the spleen was high (2,400 rads at 30 yr for 30 ml TT). To date, 802 of the living TT patients and 622 of the controls have been examined clinically and biophysically. The av age at injection was 28 yr and the av time from injection to first examination was 26 yr. The volume of TT (calculated from whole-body counting) was 22 ml. Since these examinations, 171 TT patients have died, 51 with liver tumors and 5 with leukemia. The av TT dose was about 17 ml in four of the leukemia patients, 33 ml in those who died with liver tumors. There were also three cases of reticulosarcoma. Including liver cirrhosis and leukemias, it is estimated that $>40\%$ of the patients with a known cause of death died from TT-related diseases. (12 refs)

78-4170 **Prevalence of Hepatitis B Virus Infection and Hepatocellular Carcinoma in Chinese-Americans.** (Eng) Szmuness, W. (Lab. Epidemiology, New York Blood Center, 310 E. 67th St., New York, NY, 10021); Stevens, C. E.; Ikram, H.; Much, M. I.; Harley, E. J.; Hollinger, B. *J Infect Dis* 137(6): 822-829; 1978.

The prevalence of hepatitis B virus (HBV) markers and excess mortality due to hepatocellular carcinoma (HCC) were studied in 666 Chinese permanently living in New York city. Approx 90% of the subjects were born outside the US, but 51% of the foreign-born subjects had lived in this country for at least 10 yr. Hepatitis B surface antigen (HBSAg) was detected in 9.3% of the subjects tested, antibody to this antigen (anti-HBs) in 57%, and antibody to the core antigen (anti-HBc) in 8.8%. These rates are 10-40 times higher than those found in other ethnic groups. Nearly all persons positive for HBsAg and anti-HBs were reactive to the subtypes prevalent in Asia; the e antigen was detected in only 3% of the HBsAg carriers. The incidence of anti-HBc alone or anti-HBs alone increased with age, but the incidence of HBsAg and of coexisting anti-HBs decreased with age. The age-standardized death rate from HCC among Chinese men, but not women, was 4 times higher than that among black men and 10 times higher than that among white men. An excess mortality was seen in Chinese of all ages. The findings that Chinese-Americans are at very high risk for both HBV infections and HCC support the hypothesis that these two conditions are causally associated. (24 refs)

78-4171 **Geographic Patterns of Bladder Cancer in the United States.** (Eng) Blot, W. J. (Environmental Epidemiology Branch, NCI, NIH, Bethesda, MD, 20014); Fraumeni, J. F. *J Natl Cancer Inst* 61(4): 1017-1023; 1978.

The geographic distribution of the 1950-1969 bladder cancer mortality rates (BCMR) in the US was correlated with demographic, socioeconomic, ethnic, and industrial data for the 3,056 US counties. Rates among whites and nonwhites of both sexes rose sharply with urbanization. For whites, male BCMR were not only high in urban areas, especially New Jersey, but also in rural New England, along the Illinois-Wisconsin border, in southern Michigan, and in southern Louisiana. A regional excess was most evident for white women in New England and northern New York. Increases in BCMR correlated with increasing population density, increasing socioeconomic standard, and a high percentage of British- and German-American residents. The largest industrial increases were seen in counties with chemical manufacturing operations, eg, 20/21 New Jersey counties. A positive correlation was also seen with the printing industry. Even after controlling for demographic variables, the Northeastern excess in BCMR among whites was sizable. For nonwhites, regional differences were small, but men showed a slight Southern excess and a positive urban gradient was obvious for both sexes. Male BCMR were considerably higher for whites than for nonwhites in the North but only marginally higher in the South. Female BCMR were greater for nonwhites than for whites across the country. During 1950-69, BCMR remained constant for white men and nonwhite women, increased slightly for nonwhite men, and decreased slightly for white women. (34 refs)

78-4172 **Lung Cancer Mortality and Urban Air Pollution.** (Eng) Weiss, W. (Dept. Medicine, Hahnemann Medical Coll., 6401 New College Building, 230 N. Broad St., Philadelphia, PA, 19102). *Am J Public Health* 68(8): 773-775; 1978.

The geographic variation of lung cancer mortality rates in Philadelphia was compared with the earliest obtainable information on the geographic variation in air pollution in this city. Study of lung cancer mortality in persons > 30 yr of age revealed that most cases occurred in white men; therefore, evaluation was confined to this segment. The areas of the city with high and intermediate rates of lung cancer mortality formed a broad band from the southern section into the near northeastern section along the eastern border of the city. These sections comprised a central business area with heavy automotive traffic and a high building density, low-income residential and commercial areas, and heavily industrialized areas. The sections with the lowest mortality rates were largely residential areas in the west, northwest, and far northeast sections of the city. Study of the concentrations of settled dust and the sulfation rate, based on pollution data obtained in 1960, revealed that the most heavily polluted areas had the highest lung cancer mortality rates a decade later. The highest rates for white women occurred in the most heavily industrialized sections. No data on smoking habits or occupational exposure were obtained, so interpretation of these results as a cause and effect relationship is unjustified. (7 refs.)

78-4173 **Lung Cancer after Employment in Shipyards During World War II.** (Eng) Blot, W. J. (Environmental Epidemiology Branch, Landow Bldg, Room 3C-07, NCI, Bethesda, MD, 20014); Harrington, J. M.; Toledo, A.; Hoover, R.; Heath, C. W.; Fraumeni, J. F. *N Engl J Med* 299(12): 620-624; 1978.

A case-control study was conducted to identify reasons for the exceptionally high rate of lung cancer diagnosed among male residents of coastal Georgia. The study group consisted of 458 lung cancer patients and 553 controls, most of whom were long-time residents of the area. Personal interview of the patients and controls, or next of kin in the event of death, covered place, type, and length of employment for all jobs held for 6 mo or more; duties performed and materials handled; and cigarette smoking habits. Estimates of relative risk of employment on lung cancer were calculated for 16 industrial categories. A significantly increased risk (1.6) was associated with employment in area shipyards during World War II. The excess risk increased sharply with smoking habits, indicating that there is strong synergism between these two risk factors. Assuming that 14% (the proportion among controls) of the general population had worked in shipyards and were at 1.6 times the usual risk of lung cancer, the coastal lung cancer rate would be expected to exceed the usual rate by 8%. Estimates of lung cancer mortality rates for 1970-1975 show that the rate in this area exceeds the national rate by 30%. This suggests that the coastal excess may be only partly attributable to wartime shipyard exposure. The failure to find a higher-than-av number of mesotheliomas, which occur even after relatively light exposures to asbestos, raises the possibility that hazards other than asbestos may also exist in the shipyards. (24 refs)

78-4174 **Computerized Retrieval of Case Histories.** (Ger) Crivelli, M. (Pneumologische Abteilung, Medizinische Universitatsklinik, Inselspital, CH-3010 Bern, Switzerland); Zeller, C.; Scherrer, M. *Schweiz Med Wochenschr* 108(27): 1026-1031; 1978.

The efficiency of the STAIRS-IBM computer program for the retrieval of case histories stored in text form was tested with the use of chronic bronchitis in lung cancer patients as an example. Computer selection was compared with manual selection, which involved reading all 473 case histories stored from 1974 to 1977. According to manual selection, which took 3 mo, 219/473 lung cancer patients had a history of chronic bronchitis of > 5 yr duration, and only 180 had no history of preexisting chronic bronchitis. The computer correctly selected 87% of the former group and 74% of the latter within 4 hr. With further modifications in the program, the accuracy of selection reached 90%. (10 refs)

78-4175 **Population Distribution of Histological Types of Lung Cancer. Epidemiologic Aspects in Israel and Review of the Literature.** (Eng) Modan, B. (Dept. Clinical Epidemiology, Chaim Sheba Medical Center, Tel-Hashomer, Israel). *Isr J Med Sci* 14(7): 771-784; 1978.

A nationwide study of lung cancer in Israel during 1968-1970 demonstrated a low to moderate incidence in men and a high incidence in women, compared with other countries. The sex ratio was relatively lower than expected. This discrepancy results from a proportionately lower rate of squamous cell carcinoma in men compared with the rates found in most other reports and a higher rate of adenocarcinoma in women, and it is consistent with similar trends in North American Jews from New York, Pittsburgh, and Montreal. Literature related to the relative frequencies of various histological subcategories of lung cancer is reviewed, and the etiologic and prognostic importance of a separate assessment of each distinct cell type entity is emphasized. (52 refs)

78-4176 **Primary Lung Cancer and Asthma.** (Eng) Ford, R. M. (Div. Allergy, Royal Adelaide Hosp., Adelaide, S. Australia). *Ann Allergy* 40(4): 240-242; 1978.

A total of 6,021 persons ≥ 40 yr of age who were chronic asthma suffers (> 5 yr duration) were followed over a 20-yr period to determine the incidence of lung cancer. Only two (0.032%) developed lung cancer, compared with 0.314% in the general population. The two were a 40-yr-old woman who had smoked an av of 20-25 cigarettes a day for 25 yr and a 55-yr-old man who had averaged 30-35 cigarettes a day f
20 yr. Analysis of the smoking habits of the asthmatics i
dicated that only 16.9% had smoked over any significa
period during the previous 5 yr, compared with 40% of t
general population. Further investigations indicated that
those who did smoke, they did so to a lesser degree th
persons in the general population. About 15% of the asthr
patients had received long-term immunosuppressive thera
with corticosteroids and/or hyposensitization therapy
some time during the 20-yr period. Enhanced immunologi
activity in the asthmatics could be a factor in reducing th
cancer incidence. (23 refs)

78-4177 **Probabilities of Eventually Developing and
Dying of Cancer (Risk Among Persons Pre
ously Undiagnosed with the Cancer).** (Eng) Seidman,
(Dept. Epidemiology and Statistics, American Cancer Soc
ty, New York, NY); Silverberg, E.; Bodden, A. *Ca* 28(
33-46; 1978.

A new method for computing the probabilities (P) of develo
ing and dying of cancer among persons not previously dia
nosed with that cancer is described, and the resulting da
are tabulated. The P are based on the age-, race-, a
sex-specific incidence rates/100,000 found in National Ca
cer Institute studies conducted in 1947 and in 1969-1971,
conjunction with 1949-1951 US life tables and mortality da
Nonmelanoma skin cancer is not included. The P of eventu
ly developing cancer, including carcinoma in situ (CIS),
increasing in all race-sex groups, the chances in 1970 bei
> 1/4 in whites and close to 1/4 in nonwhites. This is a
true for invasive cancer (IC) alone except in white wome
in whom the decline in IC incidence has been great enou
to balance the added number of cases resulting from increa
in life expectancy. Whether or not CIS is included, wh
women still showed the highest 1970 P for all forms of canc
27% for IC and 30% for IC + CIS. In men, the P of develo
ing lung and prostate cancer have increased substantial
The sharpest increases for all cancers occurred in nonwh
men, from 15% in 1950 to 23% in 1970. The chances at bi
of eventually dying of cancer are increasing, and in 1970 th
were > 1/7. The eventual P for men exceeded those for wo
en, reversing the situation observed in 1950. Lung canc
accounted for a large part of this increase. For white wome
the increase was not due to any increase in cancer death ra
but to a decrease in other causes of death. In 1970, the P
dying of cancer were 17% for white men, 16% for wh
women, 15% for nonwhite men, and 13% for nonwhite wo
en. (18 refs)

78-4178 **Frequency of Tonsillectomy in Bronchial Ca
cer Patients.** (Ger) Jentgens, H. (HNO-Klin
Koln, W. Germany); Matzker, J.; Steinhaus, C. *Laryn
Rhinol Otol (Stuttg)* 57(3): 190-193; 1978.

Relationships between bronchial cancer and tonsillectomy were studied in 1,415 bronchial cancer patients an 1,415 matched normal controls. The cancer patients included 1,289 men and 126 women aged 30-79 yr. Only 7.5% were nonsmokers; 80% smoked 10-50 cigarettes/day, and 12.5% smoked a pipe or cigars. Squamous epithelial carcinoma was found in 777 patients. The incidence of tonsillectomy was 84/1,415 among the cancer patients and 265/1,415 among the controls. The findings, which agree with previous findings concerning the incidences of tonsillectomy in children and adults with leukemia and ear-nose-throat tumors, suggest a causal relationship between the presence of the tonsils and malignant tumors. (4 refs)

78-4179 **Relationship Between Respiratory Cancer and Wetlands Residency in Louisiana.** (Eng) Voors, A. W. (Dept. Preventive Medicine, Louisiana State Univ. Medical Center, 1542 Tulane Ave., New Orleans, LA, 70112); Johnson, W. D.; Steele, S. H.; Rothschild, H. *Arch Environ Health* 33(3): 124-129; 1978.

A possible association between respiratory cancer and residence in the wetlands of southern Louisiana was assessed with the use of two other environmental factors known to influence respiratory cancer mortality, smoking and residence in urban communities. There was an association between wetlands residency and mortality due to tracheal, bronchial, and lung cancers among white and black men, but not among women. A smaller but significant association was also found for wetlands residency and other smoking-related cancers among men and black women. The rates for cancer of the trachea, bronchus, and lung among wetlands men were higher than the national av. Since the observed association between respiratory cancer mortality and residence in the wetlands was confined to men, and since the men smoked more than women, the existence of a wetlands-associated substance promoting respiratory cancer in smokers is suggested. (31 refs)

78-4180 **Patterns of Laryngeal Cancer in the United States (Letter to Editor).** (Eng) Blot, W. J. (Environmental Epidemiology Branch, NCI, Bethesda, MD, 20014); Fraumeni, J. F.; Morris, L. E. *Lancet* 2(8091): 674-675; 1978.

Examination of factors that may be responsible for the geographic distribution of laryngeal cancer in the US, where rates during 1950-1969 increased sharply with urbanization, suggests that not only alcohol consumption but also tobacco smoking and occupational exposures are involved. (10 refs)

78-4181 **Occupational and Nonoccupational Factors Involved in Laryngeal Cancer.** (Ger) Wolf, O. (Hals-Nasen-Ohren-Klinik, Bezirkskrankenhaus, Auenweg 38, DDR-4502 Dessau-Alten, E. Germany). *Z Gesamte Hyg* 24(3): 174-177; 1978.

Comparison of the incidence of laryngeal cancer among residents of three neighboring districts and among individuals employed in the wood and chemical industries revealed a significant increase in the risk of this disease among the latter. Occupational and nonoccupational factors involved in the development of laryngeal cancer are discussed. Occupational exposure to harmful substances via inhalation, tobacco smoke inhalation, and chronic disease of the upper respiratory tract are important contributing factors. (20 refs)

78-4182 **Malignancies in Danish Thorotrast Patients.** (Eng) Faber, M. (Finsen Lab., Strandboulevard 49, Copenhagen, Denmark). *Health Phys* 35(1): 153-158; 1978.

Results of a Thorotrast (TT) follow-up study conducted in Denmark since 1949 are reported. As of 1973, 1,012 exposed subjects had been identified (injected between 1935 and 1946, 391 of whom were still living. Of the 95 cancers and leukemias detected in this population, 43 were considered to have been induced by TT: 11 hemangioendotheliomas, 9 cholangiocarcinomas, 12 hepatomas, and 11 leukemias. The three types of liver tumors had a similar incidence, latency period (minimum of 17 yr), rise in cumulative incidence with time, and radiation dose to the liver. The minimum latency period for the leukemias was 8 yr; no cases of leukemia appeared >23 yr after the injection. No bone tumors were observed. Four of the 11 cases of respiratory cancer (all men) had latency periods <15 yr, which does not support a radiation induction. However, the high ratio of anaplastic lung tumors/epidermoid lung tumors (5/2) agrees with findings in uranium miners. It is uncertain whether the two cases of pleural mesothelioma in TT patients may have been induced by TT, because there is no evidence for significant irradiation of the pleura. It is concluded that liver cancers and leukemias can be induced by TT, but a larger population or a longer follow-up time is needed to determine whether bone sarcomas and lung tumors are induced by iv-injected TT. (12 refs)

78-4183 **Current Status of the Study of ^{226}Ra and ^{228}Ra in Humans at the Center for Human Radiobiology.** (Eng) Rowland, R. E. (Center Human Radiobiology, Argonne Natl. Lab., Argonne, IL, 60439); Stehney, A. F.; Brues, A. M.; Littman, M. S.; Keane, A. T.; Patten, B. C.; Shanahan, M. M. *Health Phys* 35(1): 159-166; 1978.

Populations being examined in a large national study of radium effects in humans and malignancies recorded to date are reported. Of 3,803 known individuals with documented exposure to Ra, 2,828 were exposed through the dial-painting industry, in which luminous Ra paints are used; 492 through laboratory work; 383 through medical usage; and 100 through miscellaneous routes. As of April 1974, complete studies had been made of 1,572 individuals. An additional 1,163 individuals (589 living, 574 dead) have been identified but not yet studied, and 1,068 have not been located. As the hazards of Ra became known, the dial-painting industry adopted control measures that were so effective, no dial painters have been found with a cumulative skeletal dose > 1,000 rads who started painting after 1926. However, the individual habits of each employee determined their actual intake of Ra, because the length of employment did not correlate with Ra intake. Two general types of malignancies in this population were considered radiation-induced: malignant bone tumors (54 cases) and carcinomas of the mastoids and paranasal sinuses (26 cases). The lowest radiation dose to a person who subsequently developed a bone tumor was 888 rads; the lowest does at which a carcinoma of the mastoids or paranasal sinuses was diagnosed was 605 rads. (13 refs)

78-4184 **Chemical Studies on Tobacco Smoke. LIX. Analysis of Volatile Nitrosamines in Tobacco Smoke and Polluted Indoor Environments.** (Eng) Brunnenmann, K. D. (Div. Environmental Carcinogenesis, Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY); Hoffmann, D. *IARC Sci Publ* (19): 343-356; 1978.

The possible contribution of N-nitrosamines (NA's) from tobacco mainstream and sidestream smoke to the contamination of indoor atmospheres was investigated. Chemical analysis indicated that the mainstream smoke from cigarettes and cigars contains trace amounts of the volatile NA's N-nitrosodimethylamine [5.7-6.5 nanograms/cigarette (ng/cig)], N-nitrosoethylmethylamine (0.4-7.7 ng/cig), N-nitrosodiethylamine (1.1-3.8 ng/cig), and N-nitrosopyrrolidine (5.1-34 ng/cig). The volatile NA's in sidestream smoke exceeded those in mainstream smoke by 10-50 times. Air was sampled in bar cars of trains, a local bar, a jai-a-lai arena, and other indoor areas polluted by tobacco smoke. The results showed that during 1 hr in a smoke-polluted indoor environment, a nonsmoker may inhale volatile NA's in quantities equal to those in the mainstream of 0.5-30 cigarettes. It has yet to be determined whether volatile NA's play a role in cancer induction in tobacco-related cancer sites. They do not appear to be contact carcinogens, even in animals. It also has yet to be determined whether they play a role at those cancer sites where they are actively metabolized; eg, the esophagus, kidney, bladder, and pancreas. (9 refs)

78-4185 **Analytical Determination of N-Nitroso Compounds in Pesticides by the United States Environmental Protection Agency--A Preliminary Study.** (Eng) Cohen, S. Z. (Environmental Protection Agency, Washington, DC); Zweig, G.; Law, M.; Wright, D.; Bontoyan, W. R. *In: Environmental Aspects of N-Nitroso Compounds. Proceedings of a Working Conference Held at the New England Center for Continuing Education, University of New Hampshire Durham, 22-24 August 1977.* International Agency for Research on Cancer. (Lyon, France): Vol. 19, pp. 333-342; 1978

Preliminary results from an analytical survey of 70-80 pesticides for N-nitroso compounds are presented. Representativ pesticides were selected on the basis of three routes of potential N-nitroso contamination: (1) industrial process formatio and carryover; (2) in situ (container) nitrosation, and (3) us of contaminated amines in the manufacturing process. Duplicate samples were analyzed by two different laboratories using gas chromatography and high-pressure liquid chromatography interfaced with Hall, UV, and chemiluminescen (thermal energy analyzer) detectors. The analysis has bee directed toward the detection of six compounds: N-nitrosodimethylamine (NDMA), N-nitrosodiethanolamine N-nitrosodipropylamine, N-nitrosobutylethylamine, N-nitrosobutralin, and N-nitrosoatrazine (NAT). Eight of 25 pesticides analyzed contained nitrosamines, 4 of which wer dinitroaniline-type compounds. Fourteen samples wer amine salts; two of these contained nitrosamines at or abov the 1-ppm limit of sensitivity of the tests. Both samples o dimethylamine showed > 25 ppm NDMA. No NAT was detected in the atrazine products. Based partly on these results the Environmental Protection Agency has decided to conduct risk-benefit analyses of pesticides suspected of containing N-nitroso contaminants, with special consideration to b given to oncogenic and mutagenic potential. (16 refs)

78-4186 **Recent Studies in Canada on the Analysis an Occurrence of Volatile and Non-volatile N-Nitroso Compounds in Foods.** (Eng) Sen, N. P. (Food Res. Div. Health Protection Branch, Ottawa, Canada); Donaldson, B A.; Seaman, S.; Iyengar, J. R.; Miles, W. F. *IARC Sci Pub* (19): 373-393; 1978.

Research devoted to the monitoring of N-nitroso compound in foods and to the development of new analytical technique for this purpose is reviewed. Analysis of 62 cheese samples using methods similar to those used for meat, indicated tha traces of nitrosamines (2-20 μg/kg), specifically N-nitrosodimethylamine, N-nitrosodiethylamine, and N-nitrosopiperidine, can occur in cheese. Nitrosamines were detected in samples prepared without the addition of nitrate making it difficult to determine whether there is any relatio between the addition of nitrate and the occurrence of nitrosamines in cheeses. Several new analytical methods were developed for the determination of nonvolatile nitroso compound such as nitrosohydroxypyrrolidine, nitrosodiethanolamine nitrosoproline (NPRO), nitrosohydroxyproline (NHPRO) and nitrosoglyphosate (a nitrosated herbicide). All of thes compounds can be converted to volatile derivatives and the analyzed by gas-liquid chromatography-mass spectrometr (GLC-MS) or by conventional GLC using a specific detector Very low levels (22-44 μg/kg) of NPRO were detected in si samples of raw bacon analyzed by GLC-MS. None of these samples contained any detectable level of NHPRO. (35 refs)

78-4187 **The Influence of Membrane Lipids on the Proliferation of Transformed and Untransformed Cell Lines.** (Eng) Hatten, M. E. (Dept. Neuropathology, Harvard Medical Sch., Boston, MA, 02115); Horwitz, A. F.; Burger, M. M. *Exp Cell Res* 107(1): 31-34; 1977.

A cell cycle analysis of untransformed 3T3 cells and transformed SV101-3T3 cells grown in a medium depleted of lipid and biotin revealed that 3T3 cells accumulate in G1, whereas SV101-3T3 cells do not. The addition of insulin to the arrested 3T3 cells triggered their escape from G1 only when biotin or fatty acids were present. The proliferation of 3T3 and SV101-3T3 cells was markedly inhibited when the cells were grown in Dulbecco's modified Eagle's medium (DMEM) supplemented with a lipid-depleted calf serum. This inhibition was reversed for both cell types by addition of the lipid extracted from the serum, the exogenous lipid (as free fatty acid or esterified to the phospholipids), or biotin. The reversal depended quantitatively on the lipid supplement and the cell line. For example, both the growth rate and density saturation of 3T3 cells grown in DMEM with biotin or oleate were markedly less than those of 3T3 cells grown in DMEM supplemented with regular calf serum. Of the fatty acids tested, only a mixture of those initially present in the serum (palmitate, stearate, oleate, and linoleate) restored growth to densities approaching those of cells grown in DMEM supplemented with regular calf serum. The proliferation of SV101-3T3 cells in lipid-depleted medium (LDM) supplemented with biotin or fatty acid differed from that of 3T3 cells in three ways: (1) the proliferation of SV101-3T3 cells grown in LDM supplemented with biotin or oleate was nearly identical with that of cells grown in media supplemented with regular calf serum or reconstituted serum; (2) SV101-3T3 cells grown in LDM supplemented with reconstituted serum had their growth restored to densities similar to those of the same cells grown in DMEM with regular calf serum; (3) SV101-3T3 cells appeared to be more sensitive to the toxic effects of saturated fatty acids. Relatively low densities and a lack of proliferative response with SV101-3T3 cells enriched with elaidate leads to the speculation that the physical properties of the cell membrane were altered so that proliferative signals were not transmitted and/or received. (14 refs)

See also:

*(Rev.): 78-3604, 78-3610, 78-3611, 78-3612, 78-3614, 78-3615, 78-3616, 78-3626, 78-3627, 78-3636, 78-3652, 78-3655, 78-3656, 78-3657, 78-3664, 78-3665, 78-3668, 78-3669, 78-3670, 78-3671.
*(Chem.): 78-3716, 78-3721, 78-3726, 78-3752, 78-3762, 78-3773, 78-3777, 78-3778, 78-3780, 78-3796, 78-3799, 78-3801, 78-3809, 78-3844, 78-3861, 78-3874, 78-3885.
*(Phys.): 78-3930.
*(Viral): 78-4000, 78-4003, 78-4014.
*(Immun.): 78-4073, 78-4077.
*(Path.): 78-4096, 78-4113, 78-4132, 78-4133.

MISCELLANEOUS

78-4188 **Experimental Studies on Factors Affecting Tumor Growth in the Liver. I. A Scrutiny of Number of Cells and Volume of Fluid Inoculated.** (Eng) Kawaguchi, T. (Second Dept. Pathology, Fukushima Medical Coll., Fukushima, Japan); Ikeda, K. *Fukushima J Med Sci* 23(1/2): 11-16; 1976.

The effects of the intrahepatic injection of various numbers of AH 130 rat ascites hepatoma cells in various volumes of tumor cell suspension were studied in female Donryu rats. Injection of 2 x 10^7, 10^6, 10^5, 10^4, 10^3, and 10^2 cells resulted in tumor growth within 90 days in 100%, 100%, 87%, 57%, 37%, and 20%, respectively, of the treated rats. Tumor growth was not influenced by the volume of the tumor cell suspension except when 2 x 10^2 cells were injected; in this case, the tumor incidence increased with increasing volume. The larger the number of tumor cells injected, the more steeply the survival curves fell. In 95% of the animals, tumor growth occurred at the site of injection in the liver. Direct invasion into the peritoneum and metastasis to the lungs, kidneys, etc, were caused by the tumor node established at the injection site. The intrahepatic injection of tumor cells provides a satisfactory experimental method for studying factors related to the growth of tumors in the liver. (7 refs)

78-4189 **Neoplasms Produced from C3H/10T 1/2 Cells Attached to Plastic Plates; Saturation Density, Anchorage Dependence and Serum Requirement of In Vitro Lines Correlated with Growth Aggressiveness In Vivo.** (Eng) Paranjpe, M. S. (NCI, NIH, Bethesda, MD, 20014); Eaton, S. A.; Boone, C. W. *J Cell Physiol* 96(1): 63-72; 1978.

A number of in vitro lines established from C3H/10T 1/2 tumors wereexamined to determine if they showed one or more of the phenotypic alterations usually associated with transformation in vitro. Polycarbonate plates (1 x 5 x 10 mm) planted with 3 x 10^4 C3H/10T 1/2 cells were implanted sc into male C3H mice. Ten of 27 mice developed tumors at the site of implantation within 11-14 wk. (This cell line is nontumorigenic when inoculated sc in saline suspension.) Spontaneous neoplastic transformation occurred in vivo as a single-stage process of positive selection for cellular alterations directly associated with neoplastic behavior. Early passages of cell lines from four different C3H/10T 1/2 tumors explanted back in culture were quantitatively examined for tumorigenicity and for alteration in the properties of density inhibition, anchorage dependence, serum requirement, and plasminogen activator production. A consistent quantitative relationship was found between the degree of growth aggressiveness in vivo and the degree of expression of these phenotypic markers of the transformed state in vitro during early passages of the cell lines after tumor explantation. (21 refs)

78-4190 **Establishment of Cultured Cell Lines Derive from a Human Gastric Carcinoma.** (En Sekiguchi, M. (Dept. Clinical Oncology, Inst. Medic Science, Univ. Tokyo, Shirokanedai, Minato-ku, Tokyo 10 Japan); Sakakibara, K.; Fujii, G. *Jpn J Exp Med* 48(1): 61-6 1978.

A human gastric carcinoma cell line, KATO-II, and a s bline, KATO-III, were established in vitro from a pleur effusion of a 55-yr-old male patient. KATO-II cells were e tablished with the use of human cord serum, and they ha been successfully subcultured >129 times for >3 y KATO-III cells have been cultured in medium containi 20% fetal bovine serum, and they have been serially passag >176 times. Both cell lines are morphologically simil They are round and free floating and they have the cytolog cal features of signet ring cells. Population doubling times f KATO-II and KATO-III cells were 74 and 36 hr, respectiv ly. The modal chromosome numbers of these lines fell in t tetraploid range. KATO-III cells exhibited little alkali phosphatase activity. Pepsinogen contents of both cell lin were almost negligible. Both cell lines formed tumors in an thymocyte serum-treated hamsters at an inoculum size of 1 cells. The microscopic appearance of the resultant tumo was consistent with poorly differentiated adenocarcinom comparable to the original tumor. (22 refs)

78-4191 **Growth of Embryonal Carcinoma Cells Serum-free Medium.** (Eng) Rizzino, A. (De Biology, Univ. California at San Diego, La Jolla, CA, 9209 Sato, G. *Proc Natl Acad Sci USA* 75(4): 1844-1848; 1978

Two mouse embryonal carcinoma cell lines, PCC.4 aza-1 a F_9, were grown for extended periods of time in serum-f F-12 medium supplemented with Pedersen fetuin, insul transferrin, and 2-mercaptoethanol (EM-1 medium). Wh the cells were transferred from medium containing serum this serum-free medium, growth continued without any c tectable lag. However, the morphology of both PCC.4 az and F_9 cells differed somewhat from cells grown in mediu containing serum. The cells were smaller and they did r grow in close association with one another. PCC.4 aza-1 ce grown in this serum-free medium for >20 generations tained their capacity to differentiate in vivo. When nude m were inoculated with PCC.4 aza-1 cells grown in serum-f or serum-containing medium, tumors were formed withi wk in all mice, and the tumors were histologically simil It is concluded that EM-1 medium can be used for t long-term growth of embryonal carcinoma cells. (22 refs

78-4192 **Rate of Cell Division in Atypias and Invasi Carcinoma of the Uterine Cervix of the Mous** (Eng) Rubio, C. A. (Inst. Pathology, Karolinska Sjukhus

Karolinska Inst., 104 01 Stockholm 60, Sweden). *J Natl Cancer Inst* 61(1): 259-263; 1978.

The rate of cell division during a 6-hr period was determined in cervical epithelia from 64 normal C57BL mice, 26 mice with cervical atypias, and 8 mice with invasive carcinoma. Cervical atypias and carcinomas were induced by intravaginal application of 1% benzo(a)pyrene (BP) in acetone 2x/wk for 5 mo. Mitosis was arrested with colchicine. The ratio of dividing basal cells to total dividing cells and the basal cell division rates for the 6-hr period were both significantly greater in normal epithelia than in atypias and greater in atypias than in invasive carcinomas. The differences were not estrous cycle-dependent. Normal epithelia, epithelia with atypia, and invasive carcinomas all showed areas rich in mitotic figures alternating with areas showing no mitoses. These findings support the view that the proliferation of squamous cells in cervical tumors is lower than that in the tissue of origin, and they militate against the hypothesis that the invasion of the host is conveyed by an increased number of mitotic figures in the atypical epithelia. The occurrence of areas with mitotic activity suggests the existence of focally distributed and probably alternating, synchronous cell production in normal cervical epithelium, a process that apparently was not altered by topical BP application or by carcinogenesis. (20 refs)

78-4193 **The Malignant Transformation: The Nature of Its Effects on Cell Replication Characteristics.** (Eng) Gilbert, D. A. (Natl. Inst. Virology, Private Bag X4, Sandringham 2131, S. Africa). *S Afr Med J* 74(2): 48-49; 1978.

An earlier concept of the mechanism of malignant transformation has been extended to cover graded responses in cell behavior. It is now suggested that malignant transformation alters the probability of replication as a result of intracellular changes and that this occurs through an increase in the sensitivity of the cell cycle switch. When the intracellular regulators of replication overcome the sensitivity of the cell cycle switch, replication occurs. If the latter is lowered by malignant transformation, the cell can replicate at a higher frequency than normal cells; yet, under suitable conditions, the rates of continuous replication may be similar. Evidence has suggested that replication in vivo is regulated by the combined action of several interacting regulators, although one or the other may dominate in a particular case. A change in sensitivity toward the dominant agent as a result of transformation may thus make the cell more responsive to the other regulators. Therefore, tumor growth could be manipulated by levels of physiological agents other than that responsible for preventing proliferation in the tumor tissue. The concept provides an explanation for the ability of cancer cells to replicate more frequently than normal cells in vivo without requiring that they exhibit marked distinctions in behavior in vitro and for the ability of a tissue to yield tumors with a spectrum of growth rates (ie, the Morris hepatomas). (21 refs)

78-4194 **Motility of Leukemia Cells.** (Eng) Haemmerli, G. (Div. Cancer Res., Inst. Pathology, Univ. Zurich, Zurich, Switzerland); Strauli, P.; Felix, H. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 481-482; 1978.

The motility of human and rat leukemia cells was studied by time-lapse cinematography and scanning electron microscopy. With rat leukemias, the unclassifiable leukemia L5222 was highly locomotive and the myeloid leukemia BN ML was essentially nonlocomotive. Motility of human leukemias was independent of their myeloid, lymphoid, or unclassified origin, and a given cell type could display a wide range of motility. Motility is a key factor in modeling the shape and surface morphology of cells. (6 refs)

78-4195 **Studies on the Nature of Protease-induced Growth Stimulation in Normal and Transformed BHK Cells.** (Eng) Kiehn, D. (Dept. Microbiology and Immunology, Univ. Washington, Seattle, WA, 98195); Brown, M. *J Cell Physiol* 97(2): 169-176; 1978.

Studies of the nature of the protease-induced growth response(s) in normal and transformed baby hamster kidney (BHK) cells and of the molecular characteristics of trypsin that are involved in mitogenic activity are reported. In the presence of growth-limiting serum concentrations, trypsin displayed mitogenic activity on actively growing but not quiescent BHK cells. These results suggest that BHK cells arrested in G_1 (G_0) are not sensitive to protease-induced growth stimulation. Studies of denatured trypsin fragments indicated that at least some of trypsin did not require the conformation and size of the native enzyme. Cellular multiplication induced by the addition of fresh serum to quiescent BHK cultures was not inhibited by high concentrations of soybean trypsin inhibitor. Similar to previous findings it was observed that plasmin was not sufficient to initiate the growth of BHK cells in soft agar. Trypsin also failed to enhance the growth of a thermosensitive polyoma-transformed BHK line in soft agar at the restrictive temperature. Finally, the growth of transformed BHK cells in soft agar did not display a requirement for plasminogen and was not inhibited by soybean trypsin inhibitor. These studies argue against the involvement of plasmin or other exogenous trypsinlike enzymes in the growth and transformation of BHK cells. (23 refs)

78-4196 **Trichomonal Granuloma of the Pelvic Cavity in a Rhesus Monkey.** (Eng) Migaki, G. (Registry Comparative Pathology, Armed Forces Inst. Pathology, Washington, DC, 20306); Benirschke, K.; McKee, A. E.; Casey, H. W. *Vet Pathol* 15(5): 679-681; 1978.

In a study of the effects of diethylstilbestrol (DES) on gestational length, a normal Rhesus monkey underwent laparotomy before her normal parturition date and DES was injected im into the fetus. After a normal parturition, she became moribund and was killed 72 days postpartum. Autopsy revealed a trichomonal granuloma behind the uterus and attached to the sigmoid colon. It is suggested that the infection took place during laparotomy; the trichomonad species was not identified. (8 refs.)

78-4197 **Nutrition and Hormone Levels in Relation to Breast Cancer and Coronary Heart Disease.** (Eng) Hill, P. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Dana Road, Valhalla, NY, 10595); Wynder, E. L.; Helman, P. *Clin Oncol* 4(1): 34-45; 1978.

The hormone profiles and serum lipid levels of healthy pre- and postmenopausal Bantu and Japanese women (B and J; low risk of breast cancer) were investigated and compared with values found in Caucasian women (C; high risk of breast cancer). The cholesterol levels were lower in premenopausal B vs J or C women, but the estrogen levels were significantly higher in B and J women vs C women. Cholesterol levels were higher in postmenopausal C women than in the other two groups, but the estrogen levels were comparable. In C women with premature castration or late pregnancy, marked differences in estrogen levels among the groups was evident; the cholesterol content was only increased in the late pregnancy group. The results indicate that the hormone profiles in B and J women are different from those in C women and that the serum lipid levels in Caucasians are higher after puberty. No correlation was evident between the cholesterol and estrogen levels in C women at different risk for breast cancer. The difference between the races may be partly associated with dietary habits. (58 refs)

78-4198 **Correlation Between Characteristics of Transformation and Malignancy of Intra and Interspecific Somatic Hybrid Cells.** (Eng) Foa, C. (Unite 119 I.N.-S.E.R.M., 27, Boulevard Lei Roure, 13009 Marseille, France); Simonetti, J.; Berebbi, M.; Fischer, P.; Vetterlein, M. *Oncology* 35(2): 58-62; 1978.

The transformed properties of five hybrid cell lines that had either one or another parent in common were studied and compared with the tumorigenicity of the lines. Hybrids were formed from human, Chinese hamster, and mouse cell lines. Three hybrid cell lines derived from the Chinese hamster DC-3F/ADX/Aza line were resistant to actinomycin D. This property appeared to be correlated with the presence of a marker chromosome from the common parent. The tumorigenicity in cortisone-treated Syrian hamsters was intermediate between those of the parental lines. Agglutinability by concanavalin A, however, was variable. Three hybrid cell lines derived from strain A9 or clone 1D, both derived from mouse fibroblasts, showed similar transformation characteristics, but two were tumorigenic and one was not. It i
concluded that the properties of the common parent are ofte
expressed in hybrids, but that properties not found in eithe
parent are sometimes found in the hybrid line as a result o
complementation. It is also concluded that somatic cell hybridization is a useful tool in the analysis of cell transformation, but it is limited by the wide variation in the karyologica
evolution of hybrid cell lines. (27 refs)

78-4199 **Differential Response of Cultured Mouse Mammary Cells of Varying Tumorigenicity to Cytochalasin B'.** (Eng) Steiner, M. R. (Dept. Virology, Baylo
Coll. Medicine, Houston, TX, 77030); Altenburg, B.; Richards, C. S.; Dudley, J. P.; Medina, D.; Butel, J. S. *Cancer Re*
38(9): 2719-2721; 1978.

Cultured BALB/c mouse mammary gland epithelial cells o
varying oncogenic potential in vivo were examined for thei
ability to multinucleate in the presence of cytochalasin I
(CB). Highly tumorigenic cell lines derived from mammar
tumors with hormonal, viral, or chemical carcinogen etiologies were extensively multinucleated when cultured i
CB-supplemented medium. Normal mammary gland cell
from either pregnant or lactating animals were predominantly mono- or binucleate under comparable conditions. At lov
passage levels after cloning, cell lines derived from a chemica
carcinogen-induced mammary tumor were weakly oncogeni
and remained largely mono- or binucleated when cultured i
CB-supplemented medium. At higher cell passage level
both the ability to produce tumors in vivo and the degree o
multinucleation in CB-supplemented medium increase
dramatically with the clonal cell lines. Thus, the response o
cultured mouse mammary gland epithelial cells to CB in vitr
may be useful as a marker of the oncogenic potential of thes
cells. (18 refs)

78-4200 **Mutagenesis During In Vitro DNA Synthesis**
(Eng) Weymouth, L. A. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA, 02139); Loeb
L. A. *Proc Natl Acad Sci USA* 75(4): 1924-1928; 1978.

The error frequency of in vitro DNA synthesis using a natural DNA template was measured with a biological assay fo
nucleotide substitutions. φX174 bacteriophage DNA containing an amber mutation (*am3* locus) was copied in vitro
by *Escherichia coli* DNA polymerase I, and the reversion
frequency of the progeny DNA was determined by transfection of *E. coli* spheroplasts. *E. coli* polymerase I made les
than one mistake at the *am3* locus for every 7,700 nucleotide
incorporated under standard reaction conditions. Substitution of Mn^{2+} for Mg^{2+} and unequal concentrations of deoxynucleoside triphosphate substrates raised the mutation to >
1/1,000. This method makes it possible to measure the mutation rate of natural DNA synthesis in a biochemically define
in vitro system. (29 refs)

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CARCINOGENESIS ABSTRACTS

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VOLUME 16, ISSUE 8

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GENERAL INFORMATION

CARCINOGENESIS ABSTRACTS makes available abstracts, annotations or citations of significant carcinogenesis articles collected from the current major biomedical sources of world literature. This service is provided by the National Cancer Institute through a contract with the Franklin Research Center for preparation of the publication, under Contract No. NOI-CP-75885 with the National Cancer Institute, U.S. Department of Health, Education and Welfare. Published and distributed by the Franklin Institute Press^{SM}.

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ABBREVIATIONS

JOURNAL names are abbreviated according to the *List of Journals Indexed in Index Medicus, Abbreviation Listing*. If the journal is not listed in this, abbreviations are derived from the *International List of Periodical Title Word Abbreviations.*

LANGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

ABBREVIATIONS used in abstracts:

A	angstrom(s)
ACTH	adrenocorticotropic hormone
ADP	adenosine diphosphate
AMP	adenosine monophosphate
ATP	adenosine triphosphate
approx	approximately
av	average
BCG	bacillus Calmette-Guerin
bid	twice daily
C	degree(s) centigrade
cal	calorie(s)
kcal	kilocalorie(s)
cc	cubic centimeter(s)
Ci	curie(s)
mCi	millicurie(s)
μCi	microcurie(s)
cm	centimeter(s)
CNS	central nervous system
cpm	counts per minute
DNA	deoxyribonucleic acid
ED$_{50}$	median effective dose
EDTA	ethylenediamine tetraacetic acid
g	gram(s)
kg	kilogram(s)
mg	milligram(s)
μg	microgram(s)
Hb	hemoglobin
hr	hour(s)
ia	intra-arterial
id	intradermal
IgA	Immunoglobulin A
IgB	Immunoglobulin B
IgG	Immunoglobulin G
IgM	Immunoglobulin M
ILS	increased life span
im	intramuscular
ip	intraperitoneal
IU	International Unit(s)
iv	intravenous
Km	Michaelis constant
LD	lethal dose
LD$_{50}$	median lethal dose
M	molar
μM	micromolar
mOsm	milliosmolar
max	maximum
mEq	milliequivalent(s)
min	minute(s)
ml	milliliter(s)
μl	microliter(s)
mm	millimeter(s)
mo	month(s)
mol wt	molecular weight
N	normal concentration
NAD	nicotinamide adenine dinucleotide
NADH	reduced nicotinamide adenine dinucleotide
NADP	nicotinamide adenine dinucleotidephosphate
NADPH	reduced nicotinamide adenine dinucleotidephosphate
NCI	National Cancer Institute
NIH	National Institutes of Health
PAS	periodic acid-Schiff
po	orally
ppb	parts per billion
ppm	parts per million
qid	four times daily
qod	every other day
QO$_2$	oxygen quotient
R	roentgen
RBC	red blood cells (erythrocytes)
RNA	ribonucleic acid
rpm	revolutions per minute
sc	subcutaneous
sec	second(s)
SGOT	serum glutamic-oxaloacetic transaminase
SGPT	serum glutamic-pyruvic transaminase
soln	solution
TCD	tissue culture dose
TCD$_{50}$	median tissue culture dose
tid	three times daily
UV	ultraviolet
WBC	white blood cells (leukocytes)
wk	week(s)
wt	weight
X	times
yr	year(s)

8-4201 **Translational Products of Type-C RNA Tumor Viruses.** (Eng) Stephenson, J. R. (Lab. RNA umor Viruses, NIH, Bethesda, MD, 20014); DeVare, S. G.; eynolds, F. H. *Adv Cancer Res* 27: 1-53; 1978.

ecent research on the translational products of C-type RNA umor viruses is reviewed. Topics covered include: C-type iral genome structure and complexity, proteins of C-type NA tumor viruses, genetic mapping of the C-type RNA umor virus genome, relatedness of structural proteins coded or by leukemia and sarcoma C-type viral genomes, and B-nd D-type oncornavirus structural proteins. (357 refs.)

8-4202 **Characterization of Human Papilloma Viruses and Their Possible Role in Human Tumors.** Eng) zur Hausen, H. (Institut fur Virologie, Zentrum fur Hygiene, Universitat Freiburg, Hermann-Herder-Strasse 11, 8 Freiburg, W. Germany); Gissman, L.; Pfister, H. *In: Cha-acterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 20-21; 1978.

Recent studies on the heterogeneity of human papilloma viruses (HPV) are reviewed. There may be two distinct types of HPV in common warts. Studies of the roles of HPV in condylomata acuminata are suggestive of a role of human genital wart viruses in genital cancer. (8 refs.)

78-4203 **The Repair of DNA Modified by Cytotoxic, Mutagenic, and Carcinogenic Chemicals.** (Eng) Roberts, J. J. (Inst. Cancer Res., Royal Cancer Hosp., London SW3, England). *Adv Radiat Biol* 17: 211-436; 1978.

In the first part of this extensive review, the interaction of various compounds with cellular DNA and the biochemical and biological consequences in a variety of cell systems are surveyed. DNA is the principal target for several chemotherapeutic cytotoxic agents as well as known carcinogens. Two mechanisms that could produce required mutation(s) to malignancy are proposed. In the first, chemically-induced mutations replicate, resulting in further damage to the DNA. The mechanism assumes a direct relationship between this damage in newly replicated DNA and cell death, chromosome aberrations, and mutations. Modifications to DNA and/or the associated chromosome aberrations are likely to be amplified during subsequent cell divisions and lead to continuing chromosome instability. Eventually, some of these changes will lead to the expression of properties of malignancy. With respect to the second mechanism, direct-acting cytotoxic agents and the active forms of some carcinogens produce a characteristic cytotoxicity by electrophilic reaction with the cellular DNA, resulting in imperfect templates for DNA synthesis. The environment of normal, unreacted cells will be modified by the presence of enlarged and dying cells, possibly over a long time. The expected restorative hyperplasia will probably differ from that following damage by other agents; ie, it may lack the normal regulatory feedback controls involved in homeostasis. Under these abnormal growth conditions, cell division is more likely to give rise to nondisjunction and chromosome aberrations in otherwise normal, surviving cells. Further changes will result in an uncontrolled or mutated malignant cell. The postulated abnormal restorative hyperplasia occurring in response to this specific toxicity could, alternatively, permit epigenetic changes akin to differentiation. The second part of the review covers excision and postreplication repair of chemically modified DNA and genetic factors and DNA repair. (867 refs)

78-4204 **Progression of Tumours and Non-neoplastic Disorders as Manifestations of a Common Biological Phenomenon.** (Eng) Burch, P. R. (Dept. Medical Physics, General Infirmary, Leeds LS1 3EX, England). *Med Hypotheses* 4(2): 101-110; 1978.

A mechanism for the progression of both neoplastic and non-neoplastic disorders is proposed. It is suggested that all well-defined diseases and conditions that exhibit a reproducible age distribution satisfying certain statistical criteria should be regarded as autoaggressive disorders. These disorders are initiated in genetically predisposed persons when one or more stem cells of the central system of growth control have each acquired a specific complement of somatic gene mutations. A specifically mutant stem cell propagates a forbidden clone of descendant cells. These cells, or their secreted humoral products, attach target cells at one or more anatomical locations. Progression of the disease is the result of the random formation of more forbidden clones that attack more target cells. It is proposed that the stepwise progression of neoplastic disease resembles the similar progression in non-neoplastic disorders. The random event that initiates the fatal crisis in neoplastic diseases may arise in a cell derived from the proliferating clone or in some cell either belonging to or deriving from the central system of growth control. This proposed mechanism is related to events in the transition of multiple benign polyps of the large intestine to carcinoma of the large intestine, the progression of tumors in the uterine cervix, Parkinsonism, and male baldness. (23 refs)

78-4205 **Iatrogenic Leukemia.** (Dut) Nieweg, H. O. (No affiliation given). *Ned Tijdschr Geneeskd* 122(12): 398-401; 1978.

In view of the leukemogenic effects of alkylating drugs, their use should be limited to malignant diseases with poor prognosis, such as multiple myeloma, Hodgkin's disease with hematogenous spread, and metastatic ovarian carcinoma. (11 refs)

78-4206 **Iatrogenic Leukemia.** (Dut) van Swaay, H. (Zwolle, Netherlands). *Ned Tijdschr Geneeskd* 122(22): 804; 1978.

Iatrogenic leukemia may result from treatment with ionizing radiation or chemical agents, such as cytostatic alkylating compounds, and it is more common in persons with the following hereditary diseases: Bloom's syndrome, Fanconi's anemia, ataxia telangiectasia, and Down's syndrome. Individuals with an abnormal number of chromosome aberrations have a higher risk of leukemia. It is concluded that iatrogenic factors are related to the etiology of leukemia and that several types of these factors are suspect. (7 refs)

78-4207 **Pathogenesis of Chronic Myeloid Leukemia.** (Por) Jamra, M. (Dep. Clin. Medica, Fac. Medicina de Ribeirao Preto, Ribeirao Preto, Brazil). *Rev Hosp Clin Fac Med Sao Paul* 33(1): 52-56; 1978.

Studies on the pathogenesis of chronic myeloid leukemia are reviewed with special regard to the role of chromosome aberrations and of the Philadelphia chromosome. (60 refs.)

78-4208 **Gestational Trophoblastic Disease: Origin of Choriocarcinoma, Invasive Mole and Choriocarcinoma Associated with Hydatidiform Mole, and Some Immunologic Aspects.** (Eng) Brewer, J. I. (Dept. Obstetrics and Gynecology, Northwestern Univ. Medical Sch., Chicago, IL); Torok, E. E.; Kahan, B. D.; Stanhope, C. R.; Halpern, B. *Adv Cancer Res* 27: 89-147; 1978.

A review of cases in which choriocarcinoma occurred during a seemingly normal pregnancy in the presence of a fetus led to the concept that the origin of the neoplasm is in the trophoblast of an otherwise normal placenta. Also reviewed are data on the occurrence of invasive moles and choriocarcinomas in association with hydatidiform moles and the immunobiology of trophoblastic diseases. (94 refs.)

78-4209 **Asbestos and Respiratory Lesions.** (Fre) Roujeau, J. (Laboratoire Louis Fruhling, Service Central d'Anatomie et Cytologie pathologiques, Groupe Hospitalier Lariboisiere, 2, rue Ambroise-Pare, 75475 Paris Cedex 10, France); Lougnon, J. *Arch Anat Cytol Pathol (Paris)* 25(6): 431-437; 1977.

In a review of the histological changes in the lung observed in persons exposed to asbestos, the relationship between asbestos and cancer is discussed. Literature reports indicate that bronchogenic cancer is observed in 5.6%-15.2% and mesotheliomas in 0.18%-5.5% of persons with asbestosis who are autopsied. The incidence of gastrointestinal tumors is also elevated in asbestos workers (about 4 times that of the unexposed population). Carcinogenesis might be related to the chronic irritation caused by asbestos fibers or to the ease of fixation of carcinogens such as benzo(a)pyrene by the fibers after they have lost their magnesium. (25 refs)

78-4210 **Experimental Induction, Histology, an Ultrastructure of Hyperplasia and Neoplasia of the Urinary Bladder Epithelium.** (Eng) Hicks R. M. (Sch. Pathology, Middlesex Hosp. Medical Sch. London, England); Chowaniec, J. *Int Rev Exp Patho* 18: 199-280; 1978.

Experimental investigations of the early developmental stages of chemical-induced bladder cancer in laboratory animals are reviewed, along with the histology of the bladder. The histological appearance of normal transitional epithelium, reversible hyperplasia, irreversible hyperplasia and early neoplastic growth, and overt neoplasia are emphasized in an attempt to delineate some of the morphological and behavioral characteristics that distinguish benign hyperplasia from early neoplastic growth. (198 refs)

78-4211 **Does Bile Promote Extra-colonic Cancer** (Eng) Lowenfels, A. B. (Dept. Surgery Westchester County Medical Center, Valhalla, NY, 10595) *Lancet* 2(8083): 239-241; 1978.

Bile acids or their bacterial degradation products are believe to be involved as promoters or cocarcinogens in the pathogenesis of large bowel cancer. If this hypothesis is correct prolonged contact with infected bile might produce cancer in other portions of the alimentary tract. Of the biliary trac cancers, the gallbladder is the most common site, and gallstones are the major risk factor. Approx two-thirds of patients with biliary calculi have bacteriologically positive bil cultures. The incidence of bile duct cancer is high in patient with choledochal cysts, formations that impede bile flow an produce stasis. Bile duct cancer is associated with ulcerativ colitis and clonorchiasis. Portal bacteremia has been foun in ulcerative colitis, and bile duct cancer develops 10 yr earlier in patients with colitis than in patients without this disease. Clonorchiasis causes bile duct obstruction and sepsis Small bowel cancers are rare but found at sites (duodenum proximal jejunum) where there is exposure to high concentrations of bile. In addition, an association is suggested between regional enteritis and small bowel cancer. Gastric cancer is associated with bile gastritis, in which there is often bacterial overgrowth by anaerobic organisms. The risk of gastric cancer is increased after about a decade in Billroth I- and II-treated peptic ulcer patients, in whom bile freely enters the stomach. It is hypothesized that prolonged exposure to bile plus hypochlorhydria, which favors bacterial growth, increases the risk of gastric cancer. A common factor in a variety of alimentary cancers in young adults is stasis of bile or intestinal content. Finally, it is also likely that reflux of bile into the pancreas can promote pancreatic cancer. (33 refs.)

78-4212 **Development of Neoplasia in Children and the Elderly. Clinical, Biochemical, and Pathogenetic Considerations Related to Microcirculation.** (Ita) Malossi, M. (Primario Pediatra, Istito Ospedalieri di Trento, Centro Infantile "Angeli Custodi", Trento, Italy). *Recent Prog Med (Roma)* 64(5): 560-568; 1978.

Degenerative changes in the capillary system during old age may alter cell respiration, creating conditions that result in neoplastic transformation and abnormal cell growth. In vitro cell proliferation studies show that reduced O_2 supply increases polyamine synthesis and protease activity in cells and diminishes the production of antigenic proteins. In children, immune surveillance is effective because intercellular nutrition and transport function well; solid tumors are rare in childhood. (20 refs.)

78-4213 **Host Factors in the Development of Childhood Cancers.** (Eng) Li, F. P. (Sidney Farber Cancer Inst., 35 Binney St., Boston, MA, 02115). *Semin Oncol* 5(1): 17-23; 1978.

A review of the diverse host factors known to influence the development of childhood cancers reveals that they fall into three categories: single-gene defects, malformation syndromes, and familial cancers. The genetics of childhood cancer, birth defects and cancer, the familial aggregation of cancer, and host-environment interactions are surveyed. (89 refs)

78-4214 **Familial Cancer and Cancer Families.** (Eng) Anderson, D. E. (Dept. Biology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., 6723 Bertner Ave., Houston, TX, 77030). *Semin Oncol* 5(1): 11-16; 1978.

The clustering of intestinal, gastric, pancreatic, esophageal, and breast cancer in families is reviewed. The specific types of hereditary cancers can be identified by focusing on the histological types, sites of involvement, associated conditions, age of the patient at the time of diagnosis, tumor frequency, and the mode of inheritance. (44 refs)

78-4215 **Workshop on Persons at High Risk for Prostatic Cancer.** (Jpn) Watanabe, H. (Dept. Urology, Kyoto Prefectural Univ. Medicine, Kyoto, Japan); Mishina, T.; Hirayama, T.; Kato, H.; Kumamoto, Y.; Yoshida, O. *Acta Urol Jpn* 24(2): 129-144; 1978.

A review of the literature on prostate cancer epidemiology revealed racial differences in incidence and a high prevalence only in older patients. The frequency is highest among US blacks, but it is relatively high among US and European whites, compared with Orientals. In Japan, however, the incidence has increased markedly over the last two decades. Migration studies indicate that Japanese-Americans have a frequency intermediate between that of US whites and native Japanese. Although the cancer latency period is similar in the two races, the cancers of US whites or Japanese-Americans are more active histologically than those of the Japanese in Japan. Possible etiologic factors in prostatic cancer are sexual activity, heredity, cadmium pollution, and diet. (52 refs)

78-4216 **Susceptibility of Human Population Groups to Colon Cancer.** (Eng) Lipkin, M. (Memorial Sloan-Kettering Cancer Center, New York, NY). *Adv Cancer Res* 27: 281-304; 1978.

Literature on the susceptibility of various human population groups to colon cancer is reviewed. Topics discussed include the effects of the environment, inherited diseases, and proliferative abnormalities on susceptibility to colon cancer, recent immunologic studies, nuclear protein and enzyme alterations, studies of cutaneous cells, and examination of fecal contents. (90 refs.)

78-4217 **Epidemiology of Colorectal Cancer.** (Eng) Miller, A. B. (Epidemiology Unit, Natl. Cancer Inst. Canada, McMurrich Bldg, Univ. Toronto, Toronto, Ontario M5S 1A8, Canada). *Can J Surg* 21(3): 209-210; 1978.

International and Canadian data on colorectal cancer incidence are reviewed. The incidence is highest in Canada, with the United States not far behind. Japan and Nigeria have the lowest incidence rate. Evidence suggests that 80% of colorectal cancers would be preventable if the environmental factors involved in their etiology were identified and eliminated. There are differing sex ratios for cancer at different sites of the colon and rectum. Nutrition has an important bearing on the incidence of colorectal cancer, as determined by studies of migrant populations. The disease occurs more frequently in urban than in rural populations and in persons of higher socioeconomic status. Colorectal cancer also seems to be characteristic of populations with a short intestinal transit time and of persons who have a high excretion of fecal bile acids and steroids. (12 refs)

78-4218 **Dietary Carcinogens.** (Ger) Sailer, D. (Abteilung fur Stoffwechsel und Ernahrung, Medizinische Universitats-Klinik Erlangen, Krankenhausstrasse 12, D-8520 Erlangen, W. Germany). *Fortschr Med* 96(9): 446-449; 1978.

Studies of dietary carcinogens (polycyclic aromatic hydrocarbons, nitrosamines, and mycotoxins) are reviewed. Benzo(a)pyrene, a product of incomplete combustion, is found throughout the environment, especially in urban and industrial areas and near highways. It also occurs in air, plants, drinking water, fish, and in smoked, pickled, and roasted meat. Nitrosamines occur in certain foods, and they are also synthesized in the stomach. The synthesis is catalyzed by thiocyanate, which is present in high concentrations in the saliva, especially in that of smokers. Epidemiological studies revealed a correlation between nitrate and nitrite levels in food and the incidence of human tumors. A relationship was found between the incidence of liver tumors in humans and dietary aflatoxin intake. The use of alcohol, especially when combined with smoking, causes a considerable increase in the risk of cancer of the mouth, larynx, and esophagus, which is possibly due to the characteristic vitamin B deficiency in alcoholics. Relationships were also found between vitamin A deficiency and cancer of the cervix and between obesity and certain tumors. The increased incidence of gastric cancer among Japanese may be due to the consumption of rice polished with talc, a potential carcinogen. Talc crystals were found in tumor tissues from 6/7 gastric cancer patients in Japan. (35 refs)

78-4219 **Industrial Viewpoints on Cancer Caused by Metals as an Occupational Disease.** (Eng) Norseth, T. (Inst. Occupational Health, Oslo 3, Norway). *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 159-167; 1977.

Identifying a metal as carcinogenic is not sufficient, as the oxidation state of the metal, its interactions with other metals and chemicals, and factors, such as smoking habits may influence its carcinogenicity. Occupational exposure to metal carcinogens, ie, nickel, chromium, and arsenic, is difficult to record and characterize chemically. Using Ni as an example, a procedure for assessing carcinogenic risk due to metals in the work environment is presented. (16 refs.)

78-4220 **The Importance of Asbestos as a Harmful Sub
stance in the Environment.** (Ger) Konetzke, G
W. (Sitzungsberichte der Akademie der Wissenschaften de
DDR, Berlin, E. Germany). *Sitzungsber Akad Wiss DD*
(2): 26-32; 1978.

Various aspects of exposure to asbestos, which can result i
the development of fibrosis and cancer of the respiratory trac
and lungs, are reviewed. At present, the health risks due t
asbestos in the atmosphere are low. (42 refs)

78-4221 **Atmospheric Pollution and Lung Cancer.** (Eng
Doll, R. (Univ. Oxford, 13 Norham Gardens
Oxford OX2 6PS, England). *Environ Health Perspect* 22
23-31; 1978.

Factors that might be responsible for the increase
incidence of lung cancer in urban compared with rur
areas are discussed. The excess cannot be accounte
for by specific occupational hazards, but some o
it might be due to the presence of carcinogens in urba
air. The excess cannot be wholly due to such agent
because it is small and variable in nonsmokers. Occu
pational studies confirm that pollutants present in tow
air are capable of causing lung cancer in man and suggest tha
the pollutants and cigarette smoke act synergistically. Lun
cancer mortality trends in young and middle-aged men i
England and Wales support the hypothesis that atmospheri
pollution has contributed to the disease. In the absence o
cigarette smoking, the combined effect of all atmospheric carcinogens is not responsible for >5 cases of lung cancer/
100,000 persons/yr in European populations. (31 refs)

78-4222 **Late Effects of Air Pollution with Special Reference to Lung Cancer.** (Eng) Friberg, L. (Dept.
Environmental Hygiene, Karolinska Inst., 104 01 Stoc
holm 60, Sweden); Cederlof, R. *Environ Health Perspe*
22: 45-66; 1978.

Epidemiological and experimental studies of the late effects
of air pollution are reviewed with emphasis on lung cancer.
Epidemiological studies have shown an increased incidence
of lung cancer in urban compared with rural areas; this is als
true for several other forms of cancer (pancreas, bladder, and
cervix uteri). General air pollution, while proportionately a
smaller factor in lung cancer than tobacco smoking, pro
bably contributes to 10%-20% of the lung canc
incidence in heavily polluted urban areas. Studies o
laboratory animals have shown that air pollutants ma
be carcinogenic. Application of condensates of th
particulate fraction of air pollutants to the skin o
animals has given rise to tumors. A number of pote
carcinogenic polycyclic organic substances have bee
isolated from the particulate fraction. Although inhal
tion experiments have been carried out with polycyclic hy-

drocarbons in pure form, no association between exposure and an increase in cancer frequency has been demonstrated. Such an association has been shown, however, under special experimental conditions. Despite problems in extrapolation of data on late effects from experimental systems to man, all substances that are carcinogenic in animals should be regarded as potential human carcinogens. (139 refs)

78-4223 **Air Pollution and Cancer: Risk Assessment Methodology and Epidemiological Evidence. Report of a Task Group.** (Eng) Cederlof, R. (No affiliation given). *Environ Health Perspect* 22: 1-12; 1978.

Current scientific knowledge about carcinogenic air pollutants is considered in relation to epidemiological data on lung cancer, and available methods for assessing cancer risks from experimental data are reviewed. Carcinogenic or cocarcinogenic chemical agents derived from the use of fossil fuels are present at higher levels in urban than in rural atmospheres. In risk assessment, positive results with a substance in a combination of appropriate short-term bioassays are indicative of possible carcinogenicity. In the absence of relevant dose-response data, the most appropriate way to estimate the lung cancer risk is to assume that it is directly proportional to the increase in dose. For small added doses, a simple linear dose-response curve, as used in radiation carcinogenesis, is appropriate. For larger added doses or special population groups already at high risk, other forms of relationship may need to be considered. Epidemiological evidence indicates that fossil fuel combustion products in ambient air, probably acting together with cigarette smoke, contribute to lung cancer in large urban areas (5-10 cases/100,000 males/yr). Urban-rural differences in the incidence of other cancers are well documented, but the role of air pollution in such differences is not known. (37 refs)

78-4224 **Applicability of Bacterial Models of DNA Repair and Recovery to UV-Irradiated Mammalian Cells.** (Eng) Hewitt, R. R. (Univ. Texas System Cancer Center M.D. Anderson Hosp. and Tumor Inst., Houston, TX); Meyn, R. E. *Adv Radiat Biol* 7: 153-179; 1978.

The repair of damage produced in DNA by UV irradiation of bacteria and mammalian cells was reviewed, and the applicability of a bacterial model to mammalian cells was evaluated. The bacterial model was not found to be strictly applicable, based on observations that (1) cells from different species exhibit widely differing capacities for dimer excision that are not reflected in their survival sensitivities; (2) variable relationships exist between dimer excision and excision repair, as indicated by repair replication or unscheduled DNA synthesis; and (3) equivalent sensitivities of the rate of DNA synthesis are seen in mammalian cells, including xeroderma pigmentosum cells, regardless of their survival sensitivities or capacities for excision repair. Since photoreactivation of survival has not been observed in mammalian cells, the role of pyrimidine dimers in lethality cannot be demonstrated rigorously in photoreactivation experiments similar to those performed in bacteria. The development of two separate models, one for human cells and one for all other mammalian species, implies species-specific differences in photochemistry, chromosome structure, enzymology, or cellular physiology that at present are not sufficiently obvious to justify this approach. It is likely that no one simple DNA repair and recovery model may be applied to all physical or chemical environmental hazards, if the model is to be specific about classifying different types of reparable DNA damage and their modes of repair. (117 refs)

78-4225 **Risk of Thyroid Cancer due to Diagnostic Doses of Radiation.** (Ger) Schmitz-Feuerhake, I. (Fachsektion Physik, Universitat Bremen, Bibliothekstrasse, 2800 Bremen, W. Germany); Batjer, K.; Prevot, H. *Fortschr Geb Roentgenstr Nuklearmed* 128(5): 622-627; 1978.

Diagnostic radioiodine and x-ray examinations of the thyroid have led to thyroid carcinoma as a late consequence in a surprisingly large number of subjects. An analysis of literature data on the radiation doses delivered in these instances indicates, under the assumption of a linear nonthreshold dose-effect relationship, that the risk of thyroid cancer can be estimated at 20-60 cases/10^6 diagnostic examinations per rad during the first 20 yr after exposure. Thus, the radioiodine test should be restricted to carefully selected individual cases. (39 refs)

78-4226 **Panel Discussion: Sources of Carcinogens.** (Eng) Albert, R. E. (Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY); Upton, A. C.; Carnow, B. W.; Wynder, E. L. *Bull NY Acad Med* 54(4): 435-441; 1978.

A panel discussion of the effects of mammography on breast cancer risk and of cigarettes and other pollutants on lung cancer risk is presented. As a woman ages, her risk of breast cancer increases but the risk of a radiation-induced carcinoma decreases. With lung cancer, one cannot speak of a threshold value for cigarette smoking; only the total body burden of carcinogenic material is important. Since carcinogens abound in the air, their regulation will be difficult. (3 refs)

78-4227 **Energy, Environment, and Health: What Can We Learn from the Nuclear Experience? Fourteenth Failla Memorial Lecture.** (Eng) Richmond, C. R. (Assoc. Director for Biomedical and Environmental Sciences, Oak Ridge Natl. Lab., P.O. Box X, Oak Ridge, TN, 37830). *Radiat Res* 73(3): 395-419; 1978.

The effects of energy-related pollutants, both nuclear and nonnuclear, on environment and health are reviewed. A survey is made of the various types of radiations and the inorganic and organic pollutants that are carcinogens, mutagens, or teratogens. Problems in establishing dose-response curves for chronic low-level exposures to ionizing radiation are presented, along with the detection of, and establishment of standards for environmental pollutants in general. (43 refs)

78-4228 **Bedside Etiology of Childhood Cancer.** (Eng) Miller, R. W. (Clinical Epidemiology Branch, NCI, Bethesda, MD). *CA* 27(5): 273-280; 1978.

Important new observations about the origins of childhood cancer are often made by the practicing physician, and several are reviewed briefly. Many observations concern environmental factors, such as prenatal exposure to diethylstilbestrol and childhood exposure to ionizing radiation, therapeutic radiation, radioisotopes, androgenic anabolic steroids for aplastic anemia, asbestos, metals (lead, copper, zinc), and viruses (Epstein-Barr virus). Other observations concern host peculiarities that carry a high risk of certain neoplasms. They include chromosomal abnormality (leukemia, retinoblastoma); cell-mediated immunosuppression (lymphoma); congenital hemihypertrophy, Beckwith-Wiedemann syndrome, congenital aniridia, renal dysplasia in the subcapsular area of the kidney (Wilms' tumor); and gonadal dysgenesis and a Y-chromosome cell line (gonadoblastoma). Certain other neoplasms seem to aggregate excessively in families: these cancers may be of a single histologic type or of dissimilar cell types. Types of familial cancer found in the literature are reported. (52 refs)

78-4229 **Factors Influencing Mutagenic Potency In Vitro.** (Eng) Ashby, J. (Central Toxicology Lab., Imperial Chemical Industries, Ltd., Alderley Park, Nr. Macclesfield, Cheshire, England); Styles, J. A. *Nature* 274(5666): 20-22; 1978.

Attempts to compare carcinogenic potency with mutagenic potency in the *Salmonella typhimurium* assay should acknowledge the established variability of in vitro mutagenic effects. The mutagenic potency of a chemical in *Salmonella* is influenced by at least 14 major factors [eg, tester strain, test protocol (plate incorporation, liquid culture, etc), S-9 mix source (organ, species)]. As a result, over the last 3 yr, the responses for several compounds have changed from reproducibly negative to reproducibly positive after the test procedure was modified. In addition, some chemicals produce mutation in vitro through the formation of metabolites not associated with those that cause cancer in vivo [benzo(a)pyrene, 7,12-dimethylbenz(a)anthracene]. Furthermore, it is probable that most mutagens will give an in vitro response with a thousandfold range and that the limits of this range will be populated by a few very potent or very weak mutagens. It is necessary to decide which species of rodent and which test protocol most closely resemble the manner of xenobiotic transformation of chemicals in humans. Correlations between mutagenic and carcinogenic potency may be found in some specific cases, and these may prove useful as a research tool, but they could easily (and wrongly assume the mantle of an invariable correlation. (41 refs)

78-4230 **Carcinogenic Activity and Biological Effects in Short-term Tests: Quantitative Aspects.** (Eng) Bartsch, H. (Lyon, France). *Staub-Reinhalt Luft* 38(6): 240-244; 1978.

The scientific basis and the efficacy of short-term tests used to predict carcinogenic effects of chemicals in animals are briefly summarized. Because compounds that produce false-negative or false-positive results in one system do not necessarily give the same false results in a different short-term assay, it is advantageous to use multiple short-term tests having different end-points to overcome the pitfalls of the individual assay. Methodological problems affecting the qualitative correlations between carcinogenicity and the biological end-points in short-term tests are illustrated by the Salmonella/microsome mutagenicity assay. These problems include the pharmacokinetics of the active mutagen, mutagen specificity, metabolic activation, and activation of carcinogenesis through deconjugation by beta-glucosidase. False-positive results may be obtained when the activation of premutagens occurs through bacterial enzymes that are not present or operating with different substrate specificities in mammalian cells. In addition, base analogs are mutagenic but may not produce tumors in animals and the rapid detoxication of chemicals in an intact organism may not occur in vitro conditions. A brief summary of experimental data that do or do not support a correlation between the carcinogenic potency of a chemical and its activity as a mutagen in the Salmonella/microsome assay, or other short-term tests, is presented. Factors that affect numerical results in in vitro and in vivo tests include the assay conditions, the type of metabolic activation system, and the time span. Short-term tests can at best serve as qualitative indicators of the potential carcinogenicity of chemicals. (34 refs.)

78-4231 **Extrapolation of Carcinogenic Risk from Animal Experiments to Man.** (Eng) Ehrenberg, L. (Dept. Radiation Biology, Wallenberg Lab., Univ. Stockholm, Stockholm, Sweden); Holmberg, B. *Environ Health Perspect* 22: 33-35; 1978.

The hypothesis that one or more mutational events are involved in tumor initiation is supported not only by the strong, jualitative correlation between mutagenic and carcinogenic ictivities of chemicals, but also by certain reaction-kinetic considerations. Experiments with microorganisms, plants, ind animals suggest that a limiting step in tumor origin s one mutational event in one cell and that the dose-esponse curve is linear from dose zero onward. An nverse relationship between dose and latency time ias been observed frequently and has been taken as in indication that it should be possible to extrapolate o such a low dose that the mean latency time would exceed the expected lifetime of the species studied. Iowever, a small dose may give individual latency times vithin the expected lifetime, even though the mean latency ime for a group exceeds the life span. The most important contribution of dose-response studies is in the application of nathematical models to epidemiologic data. It should be emembered, however, that a deviation from the linear lose-response relationship, with a higher effectiveness at lower doses, may occur for radiation, urethane, and vinyl chloride. (23 refs)

8-4232 **Ways of Detecting Carcinogen Potential in Human Populations. How Good Is the Correlation?** Eng) Newill, V. A. (Medical Dept., Res. and Environmental Iealth Div., Exxon Corporation, Linden, NJ). *Cancer Bull* 9(6): 175-178; 1977.

The reliability of animal models for predicting the carcinogenicity of a given chemical in man is reviewed. A survey of the IARC (International Agency for Research on Cancer) nonographs indicates that for 124/196 chemicals studied, no uman case studies or epidemiological data exist. Of the 108 hemicals that were positive carcinogens in animals, only 14 vere known human carcinogens. In addition, in only 18 cases vere the chemicals considered negative by both animal creening and human data. However, when known carcinogens such as diethylstilbestrol were tested in animals after heir carcinogenicity in humans had been established, positive results were obtained. It appears reasonable to assume hat the lifetime cancer incidence resulting from chronic exposure in humans can be approximated for the lifetime incidence in animals exposed similarly to same total dose per ody wt. When the animal data are extrapolated to humans, hough, the limitations of the animal system must be understood. It is suggested that the data obtained from animal studies be used in regulatory decisions for human exposure evels. (7 refs)

8-4233 **Mechanisms of Metabolic Activation of Carcinogens.** (Eng) Kriek, E. (Chemical Carcinogenesis Div., Antoni van Leeuwenhoek-Huis, Netherlands Cancer Inst., Amsterdam, Netherlands). *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 51-57; 1978.

Studies of the metabolic activation of carcinogens are reviewed under the following topics: chemicals as initiators of carcinogenesis: formation of reactive intermediates: metabolism of nitrosamines, aromatic amines, and amides: and the nature of reaction products with DNA and their repair. Schemes of the metabolic activation of diethylnitrosamine, 1-acetoxydiethylnitrosamine, and N-hydroxy-2-acetylaminofluorene and of alkylation sites on the DNA helix are given. (25 refs.)

78-4234 **Genetic Hazards of Anesthetic Gases: An Epidemiologic Study. Prevention and Extrapolation from Experimental Test Systems (Meeting Abstract).** (Eng) Infante, P. F. (Natl. Inst. Occupational Safety Health, Room 515, Post Office Building, Cincinnati, OH). *Clin Genet* 13(1): 123; 1978. (no refs)

78-4235 **Limits and Possibilities of Animal Experiments with Carcinogenic Inhalants.** (Ger) Horn, K. H. (No affiliation given). *Akademie der Wissenschaften* (2): 52-62; 1978.

Animal inhalation studies with chemical carcinogens or cocarcinogens are reviewed and their relevance to cancer induction in humans is discussed. Current inhalation techniques are not completely reliable due to the short life span of the test animals (eg, rats and Syrian golden hamsters) and the long observation periods needed to approximate human exposures. With the development of more sophisticated inhalation equipment, more positive results may be obtained with definite carcinogens. Another difficulty with the inhalation method is the evaluation of a positive result; ie, whether a substance such as ferric trioxide is carcinogenic, cocarcinogenic, or causes cytopathological conditions that promote development. Intratracheal intubation and implantation techniques are more accurate in the production of respiratory tract tumors, and they are useful in screening possible carcinogenic inhalants. The system application method is adequate only when testing a known carcinogen whose activity (po or sc) is organ-specific in the test species. However, this technique is useful in that the exact dose can be applied and the synergistic and/or cocarcinogenic properties of the test substance can be ascertained. To relate respiratory tract carcinogenesis in man to animal experiments will require the perfection of application techniques and the identification of more suitable animal models. (61 refs.)

78-4236 **The Problem of Threshold Values for Carcinogens in the Atmosphere.** (Ger) Schramm, T. (No affiliation given); Teichmann, B. *Akademie der Wissenschaften* (2): 46-51; 1978.

The problem of no-effect levels and acceptable threshold values for atmospheric carcinogens is discussed. Although there seem to be no-effect levels and doses for certain carcinogens, it is impossible to predict individual threshold values, because of great individual variations, or to determine safe levels for the general population. The cancer risk can be reduced but not eliminated by setting technical concentration standards, at first in areas where max reduction of the atmospheric concentrations of carcinogens can be achieved at acceptable cost. (21 refs.)

78-4237 **Use of Cultured Human Cells in Short-Term Tests for Chemical Carcinogens.** (Eng) Stich, H. F. (Cancer Res. Centre, Univ. British Columbia, Vancouver, Canada); San, R. H.; Lam, P.; Koropatnick, J. *Environ Health Perspect* 22: 131-132; 1978.

The advantages of using cultured human cells as indicators in short-term assays for chemical carcinogens and DNA fragmentation/DNA repair as endpoints are considered. The chief advantage of this system is its adaptability to situations that prevail in humans. The interaction of chemical and physical agents can be studied, as can the effect of human viruses on the response of cells to chemical carcinogens. Two criticisms can be directed at this system: (1) studies have been performed exclusively on fibroblasts instead of the epidermal cells from which most human tumors arise; and (2) the assay uses cultured cells forced to grow on glass or plastic surfaces rather than human organ pieces. For the large-scale screening of carcinogens, combining (1) bioassays that are the most rapid, economic, and reproducible with (2) bioassays that closely simulate conditions prevailing in humans should increase their predictive value. (13 refs)

78-4238 **Estrogens and Endometrial Cancer (2 Letters to Editor).** (Eng) Novak, E. R. (3106 St. Paul St., Baltimore, MD, 21218); Kistner, R. W. *Obstet Gynecol* 51(2): 254-255; 1978.

Criticism is made of a previous article in which a causal role for estrogen in endometrial cancer was denied. Several cancerous or precancerous situations in which estrogen is extremely suspect are summarized. Furthermore, progesterone therapy following estrogen treatment will not prevent the development of adenocarcinoma. In rebuttal, it is stated that estrogen will be a factor only in 10%-12% of the predisposed women receiving estrogen therapy. (4 refs)

78-4239 **N-Nitrosofolic Acid.** (Eng) IARC Working Group (Lyon, France). *In: Some N-Nitroso Compounds.* Magee, P. N., Chairman (International Agency for Research on Cancer: Switzerland): IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 17, 365 pp.; 217-220; 1978.

Data on the carcinogenicity of N-nitrosofolic acid (NFA) are reviewed. Ip injection of the compound into newborn mice resulted in an insignificant increase in tumors. NFA was mutagenic in several strains of *Salmonella typhimurium* in the presence of a rat liver microsomal fraction. There were data on human exposure. The carcinogenicity of NFA could not be evaluated on the basis of available data. (4 refs.)

78-4240 **The Carcinogenicity of Chloro Ethers and Related Compounds: A Brief Note.** (Eng) Nelson, N. (Inst. Environmental Medicine, New York Univ Medical Center, New York, NY, 10016). *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 115-118; 1977.

Experimental data on the mutagenicity and carcinogenicity of bis(chloromethyl)ether, chloromethyl methyl ether, dimethylcarbamoyl chloride, and diethylcarbamoyl chloride are reviewed briefly. (11 refs.)

78-4241 **Mutagenic, Carcinogenic, and Teratogenic Hazards Arising from Human Exposure to Plastics Additives.** (Eng) Withey, J. R. (Health Protection Branch, Bureau Chemical Safety, Toxicology Div., Ottawa K1A OL2, Canada). *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 219-241; 1977.

The mutagenic, carcinogenic, and teratogenic hazards arising from human exposure to plastics additives are reviewed. There are 10 major classes of additives: antioxidants, antistatic agents, blowing agents, colorants, flame retardants, free radical initiators, lubricants, plasticizers, solvents, and stabilizers. The nature of these chemical classes and species and the roles they play in a finished plastic product are discussed briefly. The antioxidants butylated hydroxyanisole and butylated hydroxytoluene appear to be noncarcinogenic and nonteratogenic, but both possess mutagenic potential. Benzoquinone and its derivatives have shown considerable antitumor activity against lymphoid leukemia L1210. Antistatic agents appear to pose little or no carcinogenic threat, but a teratogenic effect has been observed with acetyltrimethylammonium bromide in mice. Various forms of leukemia are associated with chronic exposure to blowing agents, some of which also produce liver and kidney lesions Carcinogenic colorants include 4-dimethylaminoazobenzene, 4-o-tolylazo-o-toluidine, 1-o-tolylazo-2-naphthol, 2,2′-azonaphthalene, Ponceau 3R, R-acid, and trypan blue. Some of these dyes may also be teratogenic. Among the flame retardants, halogenated aromatic hydrocarbons have little or no carcinogenic or teratogenic potential, but aliphatic halogenated hydrocarbons and halogenated monomers are well-documented carcinogens or suspected carcinogens. Organophosphate derivatives have weak mutagenic properties. No conclusive evidence has been established for the carcinogenic potential of free radical initiators, but many of these chemicals are highly suspect. There is little data on the toxicity or carcinogenic potential of lubricants; however, carcinogenesis has been linked to industrial exposure to waxes. Phthalates, used as plasticizers, are teratogenic and mutagenic, but no carcinogenic effects have been reported. Many industrial solvents are carcinogenic and teratogenic. Little is known about stabilizers and UV absorbers, except that they are highly toxic in cell culture tests. There is little literature on the testing of miscellaneous additives used in plastics with the exception of well-established carcinogenic hazards such as asbestos. (132 refs.)

78-4242 **Salbutamol: Lack of Evidence of Tumour Induction in Man (Letter to Editor).** (Eng) Finkel, M. J. (Dept. Health Education and Welfare, Public Health Service, Food and Drug Admin., Rockville, MD). *Br Med J* 1(6113): 649; 1978.

Salbutamol, a β-stimulant bronchodilator, causes mesovarian leiomyomas in rats, but no association of this or related compounds with leiomyomas in humans has been detected. Although it would be wrong to extrapolate these findings to all related compounds, it would also be wrong to dismiss these findings as species-specific. It is suggested that although some toxic manifestations are species-specific, humans should be considered equivalent to the least-favored species. (2 refs)

78-4243 **The Carcinogenicity of Organochlorine Pesticides.** (Eng) Epstein, S. S. (Sch. Public Health, Univ. Illinois Medical Center, Chicago, IL, 60612). *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 243-265; 1977.

The carcinogenicity of organochlorine pesticides (OCP) is reviewed.The 18 OCP have been proven to be carcinogenic include dieldrin, chlorobenzilate, endrin, kepone, methoxychlor, benzene hexachloride, dichlorodiphenyltrichloroe-reviewed. The 18 OCP which have been proved to be carcinogenic include dieldrin, chlorobenzilate, endrin, kepone, methoxychlor, benzene hexachloride, dichlorodiphenyltrichloroethane (DDT), lindane, pentachloronitrobenzene, perthane, dichlorodiphenyldichloroethane, aldrin, chlordane, ethylene dichloride, heptachlor, mirex, strobane, and toxaphene. Epidemiological studies were carried out to examine the relationships between chronic pesticide exposure and morbidity and mortality. Examination of age-adjusted mortality rates revealed a rising death rate, compared with a stable age-adjusted rate for the entire population. Men aged 35-44 yr with a long history of pesticide exposure had an excess death rate when compared to men in the state of Florida. Data from six studies in mice and seven studies in rats on the carcinogenicity of dieldrin and aldrin are summarized. Results of carcinogenicity studies on chlordane and heptachlor are also presented. Constraints in the generation and interpretation of carcinogenicity data are illustrated. Possible remedies for these constraints include the introduction of a disinterested advisory group or agency as an intermediary between manufacturers and commercial and other testing laboratories, and the development of independent nonprofit research centers concerned with the problems of consumer, occupational, and environmental safety. (33 refs.)

78-4244 **Antineoplastic Agents as Potential Carcinogens.** (Eng) Adamson, R. H. (Lab. Chemical Pharmacology, NCI, NIH, Bethesda, MD, 20014); Sieber, S. M. *In: Incidence of Cancer in Humans. Book A. Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 429-443; 1977.

Literature data on the animal and human carcinogenicity of various antineoplastic agents are reviewed. Although no anticancer or immunosuppressive agent can actually be implicated as being carcinogenic in humans, the agents most frequently associated with neoplasia are melphalan, Imuran, and combinations such as the MOPP regimen (nitrogen mustard, vincristine, prednisone, procarbazine). Evaluation of the role of chemotherapy in the development of acute leukemia in cancer patients is complicated by the possibility that a patient with cancer is at high risk for developing multiple primary malignancies and the possibility that acute leukemia is part of the natural history of myeloma and Hodgkin's disease. The latter is only now becoming evident because of the increased survival due to chemotherapy. Further studies in animals, particularly in nonhuman primates, and careful monitoring of all patients treated with antineoplastic agents will provide a more accurate analysis of the carcinogenic potential of these drugs. (55 refs.)

78-4245 **Carcinogenesis in an Industrial Age (Meeting Abstract).** (Eng) Gronow, M. (Cancer Res. Unit, Univ. York, York, England). *Br J Radiol* 51(602): 149; 1978. (no refs)

78-4246 **The Special Relevance of Cancer.** (Eng) Tarr, J. (Toxcon Engineering Co., P.O. Box 57310, Webster, TX, 77598); Damme, C. *Chem Eng* 85(10): 86-89; 1978.

Problems in the identification and regulation of chemical carcinogens and their implications for the chemical process industries are summarized. Human chemical carcinogens and industrially important organic chemicals recognized or suspected as human carcinogens are listed. The Toxic Substances Control Act is the newest US environmental law likely to significantly affect the use and distribution of these chemicals. (no refs)

78-4247 **Are Physiological Oxygen Concentrations Mutagenic?** (Eng) Bruyninckx, W. J. (Laboratorium voor Fysiologische Scheikunde, Faculteit Diergeneeskunde, R.U.G., Casinoplein 24, B-9000, Ghent, Belgium); Mason, H. S.; Morse, S. A. *Nature* 274(5671): 606-607; 1978.

Oxygen-sensitive mutants of the *Salmonella typhimurium* histidine auxotroph strain TA100 were used to show that exposure of oxygen-sensitive cells to oxygen concentrations physiological for mammals can lead to mutation. Agar plates were incubated anaerobically at 37 C for 3-5 days or under 5% oxygen at 37 C for 2 days. Exposure to oxygen increased the frequency of histidine-dependent revertants significantly in 3/5 mutant strains. Although oxygen mutagenicity is improbable, but not impossible, in normal aerobic mammalian cells, higher rates of formation of reactive forms of oxygen or lower rates of their destruction could lead to significant rates of mutagenesis along with the molecular pathologies arising from mutation. (7 refs.)

78-4248 **A Method for Separation and Analysis of Body Fluids in Rodents and Humans for Mutagenic Components Using *Salmonella typhimurium* as the Indicator of Mutagenicity (Meeting Abstract).** (Eng) Connor, T. (Univ. Texas Medical Branch, Galveston, TX, 77550); Legator, M. S. *Mutat Res* 53(1): 71-72; 1978. (no refs)

78-4249 **Vinyl Chloride Carcinogenicity: An Experimental Model for Carcinogenesis Studies.** (Eng) Maltoni, C. (Inst. Oncology, Bologna, Italy 40138). *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 119-146; 1977.

The carcinogenicity of vinyl chloride (VC) was evaluated in Sprague-Dawley rats, Wistar rats, Swiss mice, and golden hamsters. In inhalation studies, 50-30,000 ppm VC produced Zymbal gland carcinomas, nephroblastomas, hepatic angiosarcomas (HAS) and angiosarcomas of other sites, sc angiomas, skin carcinomas, hepatomas, brain neuroblastomas, and mammary carcinomas in adult Sprague-Dawley rats after 52 wk of exposure. Rats exposed to only 25 ppm VC for 87 wk developed HAS. Reducing the length of treatment sharply reduced the onset of several tumor types, particularly HAS: when rats were treated for 5 wk at 10,000 and 6,000 ppm, no HAS were observed. VC also had a transplacental effect: exposure of pregnant rats to 10,000 or 6,000 ppm VC on days 12-18 of pregnancy produced VC-dependent tumors in the offspring. Newborn rats were also more susceptible to VC carcinogenesis than older ones. Exposure of Wistar rats to varying VC concentrations for 52 wk resulted in basically the same tumors as those produced in Sprague-Dawley rats. Exposure of Swiss mice to 50-10,000 ppm VC for 30 wk resulted in lung tumors, mammary carcinomas, HAS, vascular tumors of other types and/or sites, and epithelial tumors of the skin. In golden hamsters, the same VC concentrations resulted in HAS, skin trichoepitheliomas, melanomas, and forestomach epithelial tumors. In addition, the latency time of lymphomas was decreased from 82 wk (in controls) to 48 wk. Ingestion of 50, 16.65 or 3.33 mg/kg/day VC by Sprague-Dawley rats, 4-5 days/wk for 52 wk produced essentially the same spectrum of tumors as the inhalation studies. One nephroblastoma and one sc angiosarcoma were found among 240 Sprague-Dawley rats that had received one to four ip injections of 4.25 mg VC, and one nephroblastoma was found among 75 animals that had received the same dose sc. It is concluded that the neoplastic response to VC depends largely on the species and strain of animal and that age is also important with respect to tumor incidence and distribution. (5 refs.)

78-4250 **Resolution of Dose-Response Toxicity Data for Chemicals Requiring Metabolic Activation: Example--Vinyl Chloride.** (Eng) Gehring, P. J. (Health and Environmental Res., Dow Chemical USA, Midland, MI, 48640); Watanabe, P. G.; Park, C. N. *Toxicol Appl Pharmacol* 44(3): 581-591; 1978.

The concept that the toxicity of many chemicals may not be a function of exposure to the chemical per se but rather to a biotransformation product is illustrated with vinyl chloride (VC). In such a case, it is necessary to determine the amount of the chemical undergoing biotransformation as a function of dose or exposure before a meaningful dose-response relationship can be established. Male Sprague-Dawley rats were exposed via inhalation to 1.4-4,600 ppm VC for 6 hr, and the total amount of VC metabolized was determined. Activation to the toxic form followed apparent Michaelis-Menten kinetics. A logarithmic probability plot of the incidence of hepatic angiosarcoma vs the amount of VC transformed, rather than the exposure concentration of VC, was linear. Assuming no threshold dose, extrapolation of the data below the range of doses causing experimentally observable responses predicted a 0.01% hepatic angiosarcoma incidence in rats exposed to 4.6 ppm VC. Theoretical extension of the extrapolation to humans exposed daily for 8 hr to 1 ppm led to a predicted incidence of 1.5/100,000,000. This incidence, although likely an overestimate in the light of evidence for at least a practical threshold in both rats and humans, is less than that expected to occur spontaneously. Although this method of estimation is not without flaws, the rationale involved represents a new approach that utilizes more logic than current extrapolation methods. The concepts that evolved from this analysis reveal why pharmacokinetics must be considered in designing toxicology experiments as well as in interpreting the resulting data. (22 refs.)

78-4251 **Some Aspects on Dose-Response in Vinyl-Chloride-induced Liver Injury and Tumors in Mice (Meeting Abstract).** (Eng) Holmberg, B. (Section Occupational Toxicology, Natl. Board Occupational Safety and Health); Kronevi, T.; Winell, M. *Scand J Clin Lab Invest* 37(Suppl 147): 74; 1977. (2 refs)

78-4252 **Presence of Mutagenic Substances in the Urines of Anesthesiologists (Meeting Abstract).** (Eng) McCoy, E. C. (Dept. Microbiology, New York Medical Coll., Valhalla, NY, 10595); Hankel, R.; Robbins, K.; Rosenkranz, H. S.; Giuffrida, J. G.; Bizzari, D. V. *Mutat Res* 53(1): 71; 1978. (no refs)

78-4253 **Mutagenicity of Epichlorohydrin in the Bacterial Assay System: Evaluation by Direct In Vitro Activity and In Vivo Activity of Urine from Exposed Humans and Mice (Meeting Abstract).** (Eng) Kilian, D. J. (Dow Chemical Co., Freeport, TX, 77541); Pullin, T. G.; Connor, T. H.; Legator, M. S.; Edwards, H. N. *Mutat Res* 53(1): 72; 1978. (no refs)

78-4254 **Modification of Ethionine Liver Carcinogenesis by Choline Deficiency (Meeting Abstract).** (Eng) Shinozuka, H. (Univ. Pittsburgh, Sch. Medicine, Pittsburgh, PA, 15261); Lombardi, B.; Iammarino, R. M.; Sell, S. *Proc Am Assoc Cancer Res* 19: 97; 1978. (no refs)

78-4255 **The Effect of Dioxane on Crystalline Isocitric Dehydrogenase (ICDH).** (Eng) Baykut, F. (Faculty Chemistry, Istanbul Univ., Istanbul, Turkey); Sengun, A.; Enginun, M. *Chim Acta Turcica* 4(2): 173-180; 1976.

The effect of 1,4-dioxane on crystalline isocitric dehydrogenase (ICDH) activity was investigated spectrophotometrically at concentrations ranging from 0.1 to 100 μl/ml. The test mixture contained 0.01 M $MgCl_2$, 0.01 M DL-isocitrate trisodium salt, 7 mg/ml NAD, and 0.1 M Tris buffer at a pH of 7.6, 0.15 ml enzyme, and varying concentrations of dioxane. The control mixture contained all chemicals with the exception of the enzyme. ICDH activity increased at low concentrations of dioxane and reached a max value at 0.5 μg/ml. The enzyme activity then decreased gradually with increasing dioxane concentration up to 10 μl/ml and then leveled off, at values slightly below those of control, at dioxane concentrations up to 100 μl/ml. Although dioxane is claimed to be a hepatocarcinogen, these results show that it may have use in tumor therapy within a certain concentration range. (15 refs)

78-4256 **Role of Lipopolysaccharide in the Production of Plasma Cell Tumors in Mice Given Mineral Oil Injections.** (Eng) Platica, M. (Res. Inst. Hosp. Joint Diseases, Mount Sinai Sch. Medicine, New York, NY, 10035); Hollander, V. P. *Cancer Res* 38(3): 703-705; 1978.

The lipopolysaccharide (LPS) content of peritoneal fluids of female BALB/c mice given ip mineral oil or pristane (2,6,10,-14-tetramethylpentadecane) injections at 2, 4, and 6 mo and of normal mice was measured. Peritoneal fluids were collected from 1 to 23 mo after the beginning of treatment. They were passed through DEAE-Bio-Gel columns to remove an inhibitor to the *Limulus* amebocyte lysate (LAL) reaction and then were assayed for LPS by a spectrophotometric LAL test. The mean LPS content of control mice was 0.09 nanogram/ml. In the treated mice, the concentration of LPS correlated with time after the first oil injection. P-200 gel chromatography and heat stability of the active material were characteristic of LPS. The low concentrations of LPS found in the peritoneal fluids from mice given oil injections less than 2 mo before sampling suggest that LPS is not simply a contaminant from injection but arises from the chronic peritonitis that develops after oil treatment. These results are consistent with but do not prove the hypothesis that LPS plays an intermediate role in the pathogenesis of plasma cell tumors in the BALB/c mouse given oil injections. (21 refs)

78-4257 **Co-carcinogens from *Croton flavens* L. and the High Incidence of Esophageal Cancer in Curacao.** (Eng) Hecker, E. (Deutsches Krebsforschungszentrum, Institut fur Biochemie, Heidelberg, W. Germany); Weber, J. *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 72-75; 1977.

Several cocarcinogenic factors were isolated from the leaves and roots of the shrub *Croton flavens* L. on the island of Curacao. These findings strongly suggest that the exceptionally high rate of esophageal cancer in Curacao may be causally related to the cocarcinogenic tigliane derivatives found, because of the widespread local habits of brewing a tea from the leaves of this plant and chewing the roots. (13 refs.)

78-4258 **Aflatoxin B$_1$-2,3-dichloride as a Model of the Active Metabolite of Aflatoxin B$_1$ in Mutagenesis and Carcinogenesis.** (Eng) Fahmy, M. J. (Dept. Genetics, Inst. Cancer Res., Pollards Wood Res. Station, Nightingale Lane, Chalfont St. Giles, Buckinghamshire HP8 4SP, England); Fahmy, O. G.; Swenson, D. H. *Cancer Res* 38(8): 2608-2616; 1978.

The biological activities of aflatoxin B$_1$ 2,3-dichloride (AFB$_1$-Cl$_2$) and the parent mycotoxin aflatoxin B$_1$ (AFB$_1$) were compared in *Drosophila melanogaster* males. Mutagenicity was assayed for the whole X chromosome (recessive lethals and visibles), at specific euchromatic loci, and at representatives of the RNA genes, especially the ribosomal DNA sites. Survival studies of the males treated by injection showed that LD$_{50}$ levels of both compounds were reached at virtually the same molar doses and times after treatment, which indicates that chlorination of the mycotoxin did not alter its general toxicity to the whole organism. In contrast, the cytotoxic and mutagenic efficiencies of AFB$_1$-Cl$_2$ were considerably higher than those of AFB$_1$, and the differentials in these activities were more marked on the metabolically inert sperm than on the metabolizing earlier germ cell stages. The mutational spectra of AFB$_1$-Cl$_2$ and AFB$_1$ in vivo were identical to those previously ascertained after monofunctional alkylation, particularly with respect to the induction of mosaicism and selectivity for ribosomal DNA. These genetic results were consistent with the conclusion that AFB$_1$-Cl$_2$ is a model of the oxidative metabolite AFB$_1$ 2,3-oxide. Both induce their mutagenicity through reactions between C-2 of the AFB$_1$ residues and the bases of genetic DNA, especially the N-7 of guanine moieties. With AFB$_1$-Cl$_2$, the ribosomal DNA mutagenic selectivity index was of the same order as that of other direct and indirect alkylating carcinogens, but its absolute genetic activity, on a molar dose basis, was orders of magnitude higher than even the more genetically potent alkylating agents. (22 refs)

78-4259 **Fluorescence Studies of the Interactions of Serum Albumin and Rat Alpha$_1$-Fetoprotein with Aflatoxin B$_1$: Specificity and Binding Parameters.** (Eng) Evrain, C. (L.A. 87 C.N.R.S., U.E.R. des Saints-Peres, 45, rue des Saints-Peres, 75006 Paris, France); Cittanova, N.; Jayle, M. F. *Biochim Biophys Acta* 533(2): 408-414; 1978.

The interaction of bovine serum albumin (BSA) and rat α_1-fetoprotein (AFP) with aflatoxin B$_1$ (AFB$_1$) was followed by the fluorescence quenching of the protein in the presence of the ligand. In the BSA-AFB$_1$ system, the number of binding sites (n) wa 3.5 and the dissociation constant (K-d) was 3.1 ± 0.5 10^{-5} M; in the AFP-AFB$_1$ and anilinonaphthalen sulfonate competed for the same hydrophobic sites on BSA Fluorescence emission spectra of lysozymes, egg albumin and gamma globulin in the presence of AFB$_1$ indicated n specificity of the mycotoxin for albumin. (7 refs)

78-4260 **Aflatoxin Induced Inhibition of Protein Synthesis.** (Eng) Ramachandra Pai, M. (Dept. Biochemistry, Vallabhbhai Patel Chest Inst., Univ. Delhi, Delhi 110 007, India); Jayanthi Bai, N.; Venkitasubramanian, T. A. *Toxicon* 16(3): 283-287; 1978.

The effects of aflatoxin B$_1$ (AFB), aflatoxin G$_1$ (AFG), and aflatoxin M$_1$ (AFM) on hepatic protein synthesis were investigated in male Wistar rats. AFB, AFG, and AFM were injected ip in single doses of 6, 12, and 6 mg/kg, respectively. For in vivo studies, the rats received ^{14}C-leucine ip 24 hr after dosing, and they were sacrificed 3 hr later; for in vitro studies, the rats were sacrificed 24 hr after toxin administration. The protein level was lowest in AFB-treated rats. Decreases in the glycogen level were observed in AFB- and AFM-treated rats. All three aflatoxins markedly increased the amount of lipids, with AFB producing the max increase and AFG the minimum. In vivo protein synthesis in all three hepatic subcellular fractions (nuclear, mitochondrial, cytoplasmic) was inhibited significantly by AFM; that in the mitochondrial and cytoplasmic fractions was inhibited significantly by AFB. No inhibition was observed with AFG, in spite of its higher dose. The inhibitory effect of AFM was greater than that of AFB in all three fractions. In vitro, mitochondrial protein synthesis was inhibited significantly by AFG and AFM, but not by AFB; however, AFM and AFB, but not AFG, significantly inhibited cytoplasmic protein synthesis. The in vivo but not in vitro inhibition of mitochondrial protein synthesis by AFB could be due to its metabolic conversion in vivo to AFM. (25 refs)

78-4261 **In Vitro Conversion of Aflatoxin B$_1$ to Bacterial Mutagen(s) by Hepatic Microsomes of Rats on a Deficient Diet (Meeting Abstract).** (Eng) Suit, J. L. (Dept. Nutrition and Food Science, Center Cancer Res., Massachusetts Inst. Technology, Cambridge, MA, 02139); Rogers, A. E.; Jetten, M. E.; Luria, S. E. *Mutat Res* 53(1): 68; 1978. (no refs)

78-4262 **Comparative Study of the Effects of Aflatoxin B$_1$ Metabolites and α-Amanitin on Rat Liver RNA Polymerase and Chromatin Template Activities.** (Eng) Prasanna, H. R. (Dept. Molecular Biology, State Univ. New York at Buffalo, Buffalo, NY); Gupta, S. R.; Viswanathan, L.; Venkitasubramanian, T. A. *Toxicon* 16(3): 289-294; 1978.

The effects of aflatoxin B_1 (AFB) metabolites on rat liver RNA polymerase and chromatin template activities were compared with those of α-amanitin. AFB was administered to male albino rats at a dose of 1 mg/kg ip, and the rats were sacrificed 4 hr later. Liver slices from these rats were treated with 3.12 μg/ml AFB or 0.1 μg/ml α-amanitin. Both compounds inhibited nucleoplasmic RNA synthesis but had very little effect on nucleolar RNA synthesis. AFB strongly suppressed RNA synthesis by liver nuclei in vitro, but had no effect on the protein content of the nuclear fraction. α-Amanitin strongly inhibited RNA synthesis by hepatic nuclei from control rats but had no effect on hepatic nuclei from AFB-treated rats. Addition of 0.001 μg α-amanitin in vitro inhibited RNA polymerase activity of control rats, but 0.01 μg inhibited enzyme activity of both control and treated rats. At low and high α-amanitin concentrations, enzyme activity was the same in control and treated rats, indicating that this compound did not exert an additive inhibitory effect on enzyme activity in AFB-treated rats. Further experiments indicated that AFB administration reduced RNA polymerase activity by 35% and the template activity of chromatin by 31%. When both enzyme and chromatin were obtained from treated rats, a 72% inhibition was observed, indicating that these two effects were additive. α-Amanitin inhibited RNA synthesis by preparations from control rats by 23%, but had no effect on preparations from treated rats. (21 refs)

78-4263 **Aflatoxin B_1 Induction of Hepatocellular Carcinoma in the Embryos of Rainbow Trout (*Salmo gairdneri*).** (Eng) Wales, J. H. (Dept. Food Science and Technology, Oregon State Univ., Corvallis, OR, 97331); Sinnhuber, R. O.; Hendricks, J. D.; Nixon, J. E.; Eisele, T. A. *J Natl Cancer Inst* 60(5): 1133-1139; 1978.

Liver cancer was induced in rainbow trout by the exposure of fertile eggs to an aqueous soln of aflatoxin B_1 (AFB_1: 0.5 μg/ml) for 1 hr. Single treatments, given on alternate days during the embryonic period, produced a low hepatocellular carcinoma incidence (< 20%) prior to formation of the embryonic liver on day 14, but a steadily increasing incidence from day 15 (31.7%) until day 23 (58.3%), in fish examined 1 yr later. Treatment of trout eggs with ^{14}C-AFB_1 was used to quantitate the amount of AFB_1 absorbed by the eggs. Trout eggs treated 21 days after fertilization absorbed approx 30 nanograms of ^{14}C-AFB_1 during a 1-hr exposure to 0.5 ppm aqueous ^{14}C-AFB_1. After 1 day, 85%-90% of the ^{14}C-AFB_1 was metabolized and excreted or it was leached from the egg. The residual ^{14}C-AFB_1 remained constant until hatching, when an additional 50% was lost. Comparison of the amount of AFB_1 absorbed by the eggs with the amount of AFB_1 consumed per fish during a 1-yr feeding trial (at 4 ppb in the diet) indicates that the trout embryo is even more sensitive than juvenile trout to the carcinogenicity of AFB_1. (12 refs)

78-4264 **Reduced Nicotinamide Adenine Dinucleotide Phosphate-dependent Formation of 2,3-Dihydro-2,3-dihydroxyaflatoxin B_1 from Aflatoxin B_1 by Hepatic Microsomes.** (Eng) Lin, J. K. (Dept. Biochemistry, Coll. Medicine, Natl. Taiwan Univ., Taipei, Taiwan, China); Kennan, K. A.; Miller, E. C.; Miller, J. A. *Cancer Res* 38(8): 2424-2428; 1978.

2,3-Dihydro-2,3-dihydroxyaflatoxin B_1 (AFB_1 dihydrodriol) was formed as a major metabolite during the incubation of AFB_1 with rat and hamster liver microsomes. The yield of the dihydrodiol was max at pH 6.5, and its formation was dependent on NADPH and cytochrome P-450. Phenobarbital pretreatment of the animals resulted in a two- to fourfold increase in dihydrodiol formation by liver microsomes. However, 3-methylcholanthrene pretreatment did not alter the activity of rat liver microsomes. Inhibitors of epoxide hydrase did not lower the dihydrodiol yield in these systems. Dihydrodiol formation from AFB_1 and rat liver microsomes was negligible in the presence of DNA. Little or no formation was noted with microsomes from rat intestinal mucosa, kidney, or lung. These results further support the formation of AFB_1 2,3-oxide as a major electrophilic metabolite of AFB_1 in rat and hamster liver microsomal systems, since this highly reactive epoxide would be expected to hydrolyze readily to form the dihydrodiol. (29 refs)

78-4265 **Beta-Propiolactone Forms Phosphodiesters with Thymidine-5′-Monophosphoric Acid (Meeting Abstract).** (Eng) Segal, A. (Lab. Organic Chemistry and Carcinogenesis, Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY, 10016); Mate, U.; Wortman, M. *Proc Am Assoc Cancer Res* 19: 4; 1978. (no refs)

78-4266 **Mutagenicity of Inorganic Compounds in *Salmonella typhimurium*: Arsenic, Chromium and Selenium (Meeting Abstract).** (Eng) Lofroth, G. (Biochemistry Dept., Univ. California, Berkeley, CA, 94720); Ames, B. N. *Mutat Res* 53(1): 65-66; 1978. (no refs)

78-4267 **Metabolism of the Carcinogen Chromate by Rat Liver Microsomes.** (Eng) Gruber, J. E. (Dept. Chemistry, Dartmouth Coll., Hanover, NH, 03755); Jennette, K. W. *Biochem Biophys Res Commun* 82(2): 700-706; 1978.

The in vitro metabolism of chromate by rat liver microsomes was studied to determine whether the oxidation state of chromium would be affected. Incubation of chromate with rat liver microsomes and NADPH resulted in the reduction of hexavalent chromium to trivalent chromium, the amount of chromate reduced depending on the concentration of microsomes and NADPH. Max (complete) conversion to chromium(III) occurred with a microsome concentration of 1.5 mg/ml and an NADPH concentration of 1.44×10^{-3} M for 6 hr. Incubation of chromate with microsomes in the absence of NADPH led to complete recovery of chromate even after 24 hr incubation. In the absence of microsomes, $<20\%$ of the chromate was reduced by NADPH after 6 hr. These results suggest that an NADPH-requiring enzyme or enzyme system is involved in the in vitro metabolism of chromate. These data plus the results of earlier studies indicate that the metabolism of chromate by microsomal systems occurs in vitro and that it may be an important factor in determining the mutagenicity and carcinogenicity of chromium(VI) compounds in vivo. (16 refs.)

78-4268 **Cytogenetic Effects of Food Preservatives Sodium Sulphite and Sodium Benzoate (Meeting Abstract).** (Eng) Gopalan, H. N. (Botany Dept., Kenyatta Univ. Coll., Box 43844, Nairobi, Kenya); Njagi, G. D. *Genetics* 88(4, part 2, Suppl): s32-s33; 1978. (no refs)

78-4269 **Chromosomal Damage and Hair Dyes (2 Letters to Editor).** (Eng) Van Abbe, N. J. (British Industrial and Biological Res. Assoc., Carshalton, Surrey, England); Price, D. J. *Lancet* 2(8085): 368; 1978.

A three-way contingency analysis of the effect of hair dyes on chromosomes indicated that the damage associated with hair dyeing alone was not as prominent as that induced by high-dose radiography or the more serious viral infections. In another analysis of the data using the G-test, the excess chromosomal damage induced by dyes was found to be statistically significant, as was that induced by radiation and recent virus infections. However, there was no interaction between the dye and the other factors. (1 ref.)

78-4270 **Study of the Mutagenic Potential of Three GRAS Chemicals in Mice by the Heritable Translocation Test (Meeting Abstract).** (Eng) Jorgenson, T. A. (Stanford Res. Inst., Menlo Park, CA); Rushbrook, C. J.; Newell, G. W.; Green, S. *Mutat Res* 53(1): 124-125; 1978. (no refs)

78-4271 **Growth and Cytogenetic Characteristics of Nickel Sulphide-induced Rhabdomyosarcomas in Rats.** (Eng) Yamashiro, S. (Dept. Biomedical Sciences, Univ. Guelph, Guelph, Ontario, Canada N1G 2W1); Gilman, J. P.; Basrur, P. K.; Abandowitz, H. M. *Acta Pathol Jpn* 28(3): 435-444; 1978.

Rhabdomyosarcomas induced by single im injections of nickel sulfide (Ni_3S_2) in Fischer and Hooded rats were cultured in vivo and in vitro to study their growth characteristics and chromosome constitution. Tumor cell suspensions cultured in vitro exhibited more myogenic differentiation on the coverslips than cells grown in vivo in diffusion chambers. A characteristic feature of in vivo cultures was the appearance of microclusters that resembled the primary tumors. Chromosome analyses of the primary tumors revealed that a majority had a modal number in the diploid or near-diploid range. Fischer rat primary tumor cells exhibited abnormal configurations, including rings, dicentrics and triradials. A comparison of the chromosome makeup of the primary tumors and their metastases was performed on four sets of tumors. Three out of four metastases examined showed the diploid chromosome makeup characteristic of the primary tumors, suggesting that tumors with a diploid or near-diploid chromosome constitution are more likely to produce metastases. (19 refs)

78-4272 **Unscheduled DNA Synthesis Induced in Mouse Spermatids after Combined Treatment with Methyl Methanesulfonate and X-Rays.** (Eng) Carpenter, D. A. (Univ. Tennessee-Oak Ridge Graduate Sch. Biomedical Sciences, Oak Ridge, TN, 37830); Sega, G. A. *Mutat Res* 50(2): 219-228; 1978.

The combined effects of methyl methanesulfonate (MMS) and x-rays on (C3Hf x 101)F_1 mouse spermatids were determined. Mice were given 75 mg/kg MMS ip and a concurrent testicular injection of ^{3}H-thymidine (^{3}H-TdR) either immediately before or up to 20 hr after x-ray treatment (50-600 R). X-rays and MMS induced unscheduled DNA synthesis (UDS) in the spermatids. However, there was a statistically significant reduction in the amount of UDS when x-ray exposures of 200-600 R were given 4 hr before the MMS + (^{3}H)dThd injection. This reduction was greater than that which could be explained by the completion of repair of x-ray-induced lesions. It is suggested that the reduction in UDS was the result of an x-ray-produced impairment of at least a part of the repair mechanism involved in correcting MMS-induced DNA lesions. When the time interval between a 600-R x-ray exposure and MMS treatment was between 3 and 20 hr, there was a statistically significant reduction in UDS in the spermatids. No significant decrease in the UDS response occurred when the time interval between radiation expsure and MMS treatment was approx $<$ 3 hr. (10 refs)

78-4273 **The Metabolism of Lithocholic Acid-3α-sulfate by Human Intestinal Microflora.** (Eng) Kelsey,

M. I. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, P.O. Box B, Frederick, MD, 21501); Muschik, G. M.; Sexton, S. A. *Lipids* 13(2): 152-157; 1978.

Human intestinal microflora from freshly collected human feces were incubated with lithocholic acid 3α-sulfate, and the metabolites were analyzed by chromatography and mass spectrometry. The chromatographic properties of one of the metabolites were consistent with those described for a Δ^2- or Δ^3-cholenate. The formation of cholenates by the microflora may represent a retoxification of the sulfate ester of lithocholic acid. Since the compound is a major bile salt in the colon, microbial metabolism of this substrate may result in toxic or mutagenic products in patients at risk for colon cancer. (29 refs)

78-4274 **Malignant Mesothelioma with Minimal Asbestos Exposure.** (Eng) Chen, W. (Dept. Pathology, Univ. Washington Sch. Medicine, Seattle, WA, 98195); Mottet, N. K. *Hum Pathol* 9(3): 253-258; 1978.

The occurrence of a malignant mesothelioma in a 59-yr-old nonsmoker with minimal asbestos exposure is reported. The extent of exposure was documented by thorough examination of the patient's occupational history and personal hobbies and by counting the number of asbestos fibers in the lungs. There was an av of 168 fibers per 5 g wet tissue sample. Electron probe x-ray dispersion analysis of the fibers revealed the characteristic chemical components of asbestos – Si, Mg, Ca, and Fe. (16 refs)

78-4275 **Colon Cancer, Bile-Salt Binding, and Aluminium Hydroxide (Letter to Editor).** (Eng) Cruse, J. P. (Surgical Unit, Rayne Inst., Univ. Coll. Hosp. Medical Sch., London WC1E 6JJ, England); Lewin, M. R.; Clark, C. G. *Lancet* 1(8076): 1261; 1978.

The antacid aluminum hydroxide (AH), a bile salt-binding compound, may indirectly promote colon cancer by increasing the delivery of bile salts to the large intestine. A study of dimethylhydrazine-treated Wistar rats showed that AH increased the mortality over that due to the carcinogen alone. AH may be harmful to patients who have, or have had, carcinoma of the colon. (9 refs)

78-4276 **DNA Alkylation in Mice with Genetically Different Susceptibility to 1,2-Dimethylhydrazine-induced Colon Carcinogenesis.** (Eng) Cooper, H. K. (Abteilung Chemie, Ruhr-Universitat, 4630 Bochum, W. Germany); Buecheler, J.; Kleihues, P. *Cancer Res* 38(9): 3063-3065; 1978.

The role of methylation of DNA bases in the initiation of colon tumors by 1,2-dimethylhydrazine (DMH) was studied in male mice with a high (ICR/Ha) and low (C57BL/Ha) susceptibility to DMH-induced colon carcinogenesis. The mice were given a single sc injection of 15 mg/kg ^{14}C-DMH and sacrificed either 12 or 60 hr later. The concentrations of 7-methylguanine and O^6-methylguanine in the DNA of the colon, ileum, and kidney were 40%-60% lower in C57BL/Ha mice than in ICR/Ha mice. In hepatic DNA, the extent of methylation was higher in C57BL/Ha than in ICR/Ha mice. The rate of loss of methylated purines from colon DNA was similar in both strains. In all organs investigated, the incorporation of label into normal DNA bases was lower in C57BL/Ha mice. It was concluded that the low carcinogenic response of C57BL/Ha mice was due to the smaller extent of initial alkylation of colon DNA, which probably reflects differences in the enzymic metabolism of the parent carcinogen. (17 refs.)

78-4277 **DNA Damage in Liver, Colon, Stomach, Lung and Kidney of BALB/c Mice Treated with 1,2-Dimethylhydrazine.** (Eng) Brambilla, G. (Dept. Pharmacology, Univ. Genoa, I-16132 Genoa, Italy); Cavanna, M.; Parodi, S.; Sciaba, L.; Pino, A.; Robbiano, L. *Int J Cancer* 22(2): 174-180; 1978.

DNA single-strand breaks induced in various organs of BALB/c mice by treatment with a single dose of 1,2-dimethylhydrazine [DMH: 12.5-200 mg/kg were studied po (gastric intubation) or sc] were studied by a modified alkaline elution method. The modification allowed the evaluation of DNA damage in vivo without the use of radioactive prelabeling; DNA damage was detected in liver, lung, kidney, stomach and colon mucosa, with the liver showing the greatest amount of damage. The degree of damage was dependent on the dose and route of administration, being greater by the sc route. A differential effect was evident in colon mucosa from Swiss and C57BL/6 mice, which are respectively susceptible and resistant to the induction of bowel tumors by DMH. The higher degree of DNA damage found in liver in comparison with colon mucosa is consistent with the previously reported higher degree of DNA methylation in the former, but it does not correlate with the specificity of this carcinogen in inducing large intestinal tumors in mice given repeated sc injections. (62 refs.)

78-4278 **The Influence of Serotonin on the Mitotic Rate in the Colonic Crypt Epithelium and in Colonic Adenocarcinoma in Rats.** (Eng) Tutton, P. J. (Dept. Anatomy, Monash Univ., Clayton, Victoria, Australia); Barkla, D. H. *Clin Exp Pharmacol Physiol* 5(1): 91-94; 1978.

The influence of serotonin on the mitotic rate in colonic crypt epithelium and in chemically induced colonic adenocarcinomas was studied in male Sprague-Dawley rats. The rats were given 21 weekly injections of 1,2-dimethylhydrazine (21 mg/kg, sc) and then studied 2-8 wk later. In animals partially depleted of serotonin following an injection of 6-fluorotryptophan and in those whose serotonin receptors were blocked by treatment with bromolysergic acid diethylamide, cell proliferation proceeded at a normal rate in the crypt epithelium while essentially ceasing in the colonic tumors. Injection of 10 μg/kg serotonin caused an increase in the mitotic rate in the tumor tissue but not in the crypt epithelium. Blocking α-adrenoreceptors, while simultaneously stimulating serotonin receptors, might, by transiently decreasing the ratio between cell proliferation rates in normal and neoplastic cells, increase the efficacy of antineoplastic drugs whose specificity is for rapidly dividing cells. (14 refs)

78-4279 **Evidence of DNA Repair in the Guinea Pig Pancreas, In Vivo and In Vitro, Following Exposure to N-Methyl-N-Nitrosourethane.** (Eng) Iqbal, Z. M. (Sch. Public Health, Univ. Illinois Medical Center, P.O. Box 6998, Chicago, IL, 60680); Epstein, S. S. *Chem Biol Interact* 20(1): 77-87; 1978.

The DNA damage induced by N-methyl-N-nitrosourethane (NMUT) in the guinea pig pancreas and its subsequent repair was investigated in vitro and in vivo by alkaline sucrose density gradient analysis. A nonradioactive fluorimetric procedure was used to determine the DNA in gradient fractions. Following 30 min in vitro exposure of pancreatic slices to 20 mM NMUT, 65.5% of the DNA sedimented near the top of the gradient as $< 2.24 \times 10^6$-dalton fragments. However, incubation of carcinogen-treated slices for 3 hr in a fresh medium resulted in the repair of most DNA damage, as indicated by conversion of the low-mol-wt DNA fragments to DNA of mol wt comparable to that of control slices. A single ip administration of NMUT (30 mg/kg) to guinea pigs also induced extensive DNA damage ($< 2.24\ 10^6$-dalton fragments) in the pancreas within 4 hr; similar DNA damage was observed in the liver. However, in the pancreas and liver of animals sacrificed at increasing intervals after NMUT administration, a gradual conversion of shortened DNA fragments to high-mol-wt DNA indicated repair of DNA damage. Most of the DNA damage in the pancreas and liver was repaired by 14 and 7 days, respectively. (24 refs)

78-4280 **Mutagenicity Testing of Benomyl, Methyl-2-benzimidazole Carbamate, Streptozotocin and N-Methyl-N′-nitro-N-nitrosoguanidine in *Salmonella typhimurium* In Vitro and in Rodent Host-mediated Assays.** (Eng) Ficsor, G. (Dept. Biomedical Sciences, Western Michigan Univ., Kalamazoo, MI); Bordas, S.; Stewart, S. J. *Mutat Res* 51(2): 151-164; 1978.

In vitro spot tests, microsomal plate assays, liquid culture treatments, and rodent host-mediated assays were used to test the mutagenicity of benomyl, its commercial preparations Fundazol 50WP and Benlate 50WP, the benomyl metabolite methyl-2-benzimidazole carbamate (MBC), and its commercial preparation MBC 50WP. *Salmonella typhimurium* strains *his*G46, TA100, TA1530, TA1535 and TA1950 were used. In nonoverlay spot tests, Benlate 50WP was nonmutagenic to *his*G46 and TA1535 over a dose range of 50-5,000 θg/spot. In overlay spot tests of all five compounds (50-100 μg/spot) with *his*G46, TA1530, and TA1950, only MBC (100 μg/spot) showed weak mutagenicity in *his*G46. Positive controls were mutagenic in each strain. Toxicity was not observed. In the microsomal activation plate assay, MBC, benomyl, Fundazol 50WP and Benlate 50WP (50-2,000 μg/plate) were tested with TA100, and all were found to be nonmutagenic. Fundazol 50WP (10, 100, 1,000, and 10,000 μg/ml) was nonmutagenic to TA1530 in a 20 hr liquid culture treatment. In 1-hr liquid culture treatments, benomyl, Benlate 50WP, and Fundazol 50WP (0.25-1,000 μg/ml) failed to produce mutation in *his*G46, TA100 or TA1950. Appropriate positive controls were mutagenic in each test. In the rodent host-mediated assay, Fundazol 50WP was not mutagenic in mice [sc (500 mg/kg at 0, 1, 2 hr) with *his*G46 or po (4000 mg) with TA1950] but the positive control streptozotocin was. The consistently negative results in this study with commercial preparations of MBC and benomyl are contrary to positive findings reported previously with similar methods and similar preparations. Possible reasons for these differences are presented. (14 refs.)

78-4281 **Comparisons of the Fidelity of Transcription of RNA Polymerase I and II Following N-Hydroxy-2-acetylaminofluorene Treatment.** (Eng) Glazer, R. I. (Lab. Medicinal Chemistry and Biology, NCI, Room 6A01, Building 37, Bethesda, MD, 20014). *Nucleic Acids Res* 5(7): 2607-2616; 1978.

An attempt was made to determine if misincorporation is responsible for changes in the nucleotide distribution of the RNA transcribed by carcinogen-modified enzymes. Hepatic RNA polymerase I and II from normal or N-2-hydroxy-2-acetylaminofluorene (OH-AAF)-treated Sprague-Dawley rats or *Escherichia coli* polymerase and the synthetic templates poly(dG-dC)-poly(dG-dC) (A) and poly(dA-dT)-poly(dA-dT) (B) were used With template A, the predominant base-pair transformation that was transcribed in the presence of $Mg+^{2}$ or $Mn+^{2}$ by RNA polymerase I from control or OH-AAF-treated animals or by *E. coli* RNA polymerase was the transversion dG-rC to dG-rA. However, transcription in the presence of $Mg+^{2}$ by RN polymerase II from carcinogen-treated animals showed a statistically greater degree of the base-pair transformation dG-rC to dG-rU. In contrast, RNA polymerase I and II from control or OH-AAF-treated rats transcribed the base-pair transformation dA-rU to dA-rG, dA-rU to dA-rC, and dA-rU to dA-rA to equal extents with template B. *E. coli* RNA polymerase transcribed template B to produce only the transversion dA-rU to dA-rG. The results show that there is an inherently high degree of misincorporation by both rat liver enzymes and the bacterial polymerase when transcribing template A, compared with the low error rate that occurs with template B. Thus, RNA polymerases are capable of miscopying synthetic DNA templates. The consequences of base-pair transformations on the fidelity of transcription after carcinogen treatment are explored. (21 refs.)

78-4282 **Photolysis of 2-Azidofluorene In Situ as a Probe in Chemical Carcinogenesis: Bypass of Requirement for Metabolic Activation.** (Eng) Sarrif, A. M. (Dept. Pharmacology, Univ. Alabama in Birmingham Medical Center, Birmingham, AL, 35294); White, W. E.; DiVito, N. *Biochem Biophys Res Commun* 83(2): 506-512; 1978.

Photoaffinity labeling was used as a probe for studying carcinogenesis in C3H 10T1/2 CL8 mouse embryo fibroblasts. 2-Azidofluorene (2-AzF) was synthesized to serve as a specific photoaffinity label. Nonirradiated 2-AzF exhibited little toxicity and no transforming activity in the C3H cells. When 2-AzF was photolyzed by UV light (360 nanometers) in water to generate N-hydroxy-2-aminofluorene and then added to the cell, little toxicity again was noted, but the drug was weakly transforming. In both experiments, cell survival was 75%. However, when 2-AzF was incubated with the cells for 10 min and then photolyzed for 15 min, cell survival was reduced to 11%. The drug plus UV light also caused significant transformation, with both Types II and III foci being observed. The cytotoxicity profile and the amount of transformation induced were comparable with those obtained with N-acetoxy-2-acetylaminofluorene. Irradiation of the cells in the absence of the drug was virtually nontoxic and nontransforming. These findings indicate that photoaffinity labeling may be used as a specific probe in cell transformation studies. (13 refs.)

78-4283 **Effects of Acetylaminofluorene on Transfer RNA Methyltransferases of Rat Organs.** (Eng) Wainfan, E. (Lindsley F. Kimball Res. Inst. New York Blood Center, New York, NY); Randle, S. S.; Relyea, N. M.; Balis, M. E. *Cancer Res* 38(7): 1852-1856; 1978.

The activity of transfer RNA (tRNA) methyltransferases and levels of polyamines in the liver and other organs were measured in male and female CFN rats during the administration of acetylaminofluorene (AAF, 0.06% in the diet). Both the specific activity and max capacity of the tRNA-methylating enzymes in male rats that had received AAF for 3 wk were about double control levels. When HN_4+ was added to the assay mixture, there was an almost three-fold stimulation of enzyme activity in both groups. In AAF-treated males, the activity of liver tRNA methylase rose sharply within 5 days and remained elevated. In the treated females, liver tRNA methylase activity was only slightly increased after 5 days and was within control limits after 3 wk. There were no sex-related differences in enzyme activity in the controls. The increased enzyme activity in the males was still evident after 3 and 6 mo of AAF treatment. The tRNA-methylating enzyme activities in the spleen and colonic mucosal cells of treated males did not differ significantly from control levels. No significant differences in liver polyamine levels were observed between treated males and controls. These findings suggest that there may be a correlation between AAF-induced qualitative and quantitative changes in tRNA methylase activity and the initiation of steps leading to neoplasia. (no refs.)

78-4284 **Identification of Epoxide Hydrase as the Preneoplastic Antigen in Rat Liver Hyperplastic Nodules.** (Eng) Levin, W. (Dept. Biochemistry and Drug Metabolism, Hoffmann-La Roche Inc., Nutley, NJ, 07110); Lu, A. Y.; Thomas, P. E.; Ryan, D.; Kizer, D. E.; Griffin, M. J. *Proc Natl Acad Sci USA* 75(7): 3240-3243; 1978.

A liver microsomal protein, termed preneoplastic antigen, was detected in the hyperplastic nodules of male Holtzman rats fed a diet containing 2-acetylaminofluorene and was identified as the enzyme epoxide hydrase [glycol hydro-lyase (epoxide-forming)]. Purified preneoplastic antigen from hyperplastic nodules and purified rat liver microsomal epoxide hydrase were immunochemically identical on the basis of Ouchterlony double-diffusion analysis. In addition, the purified proteins had identical minimum mol wts in sodium dodecyl sulfate/polyacrylamide gels and both catalyzed the hydration of arene oxides to dihydrodiols. Chronic feeding of 2-acetylaminofluorene (0.05% of diet) to rats resulted in a five- to sevenfold increase in epoxide hydrase activity in the liver. The induced level of the enzyme was maintained in developing hyperplastic nodules and hepatomas, but not in the nontumor tissue after the carcinogen was removed from the diet. (24 refs.)

78-4285 **Unscheduled DNA Synthesis Induced by Procarcinogens in Suspensions and Primary Cultures of Hepatocytes on Collagen Membranes.** (Eng) Michalopoulos, G. (Jones Lab. Cancer Res., Dept. Pathology, Duke Univ. Medical Center, Durham, NC, 27710); Sattler, G. L.; O'Connor, L.; Pitot, H. C. *Cancer Res* 38(7): 1866-1871; 1978.

The feasibility of using suspensions of freshly isolated hepatocytes as a system for the rapid in vitro detection of potential chemical carcinogens was studied. The amounts of unscheduled DNA synthesis (UDS) in hepatocellular suspensions were measured after isolation of the DNA by two methods. With the trichloroacetic acid (TCA) precipitation method, insignificant (10%-20%) UDS was observed when high concentrations of the procarcinogen N-2-fluorenylacetamide (AAF) were used. When the cell DNA was isolated on cesium chloride gradients, much higher levels of UDS were measured at the same AAF concentrations. Suspensions of freshly isolated hepatocytes were incubated with various procarcinogens and ^{3}H-thymidine, and the DNA was isolated on cesium chloride gradients. Dimethylnitrosamine (DMN) elicited measureable UDS at high concentrations. 3′-Methyl-4-dimethylaminoazobenzene (3′MeDAB) also elicited measurable UDS at 2×10^{-5} M, but higher concentrations resulted in less UDS, possibly because of the toxicity of the compound. Similar toxicity effects were seen with benzo(a)pyrene (BP), which elicited max UDS at 8×10^{-6} M. UDS increased with increasing AAF concentration and with increasing aflatoxin B_1 concentration up to 10^{-6} M. Of the five carcinogens tested, BP and aflatoxin were the most effective in eliciting UDS. BP was also the most effective in eliciting UDS in 6-day-old cultures of hepatocytes on collagen membranes. UDS caused by the addition of AAF, 3′MeDAB, and aflatoxin B_1 to these cultures was less than that observed with similar concentrations of these carcinogens in suspensions of freshly isolated hepatocytes. (23 refs.)

78-4286 **Different Deviation Patterns of Carbohydrate-metabolizing Enzymes in Primary Rat Hepatomas Induced by Different Chemical Carcinogens.** (Eng) Sato, K. (Second Dept. Biochemistry, Hirosaki Univ. Sch. Medicine, Hirosaki 036, Japan); Takaya, S.; Imai, F.; Hatayama, I.; Ito, N. *Cancer Res* 38(9): 3086-3093; 1978.

The deviation patterns of nine carbohydrate metabolizing enzymes were examined simultaneously in the individual hyperplastic liver nodules and primary hepatomas induced in Sprague-Dawley rats by N-2-fluorenylacetamide (2-FAA), diethylnitrosamine (DENA), or 3′-methyl-4-dimethylaminoazobenzene (3′-Me-DAB). Rats were fed a diet containing 0.025% 2-FAA for 20 wk and then a normal diet for 4-12 wk, or 0.01% DENA in the drinking water for 3 wk, or a diet containing 0.06% 3′-Me-DAB for 25-29 wk. With decreased histological differentiation of the tumor, the liver-specific marker enzymes of gluconeogenesis, glucose-6-phosphatase and fructose-1,6-bis-phosphatase, as well as the hepatic isozymes of glucose-adenosine 5′-triphosphate phosphotransferase (GATP), pyruvate kinase, aldolase, and glycogen phosphorylase, decreased gradually and were replaced with the respective nonhepatic fetal or prototypic isozymes of undifferentiated tissues. In poorly differentiated hepatomas, glycogen synthase was also decreased and phosphofructokinase was increased. Glucose-6-phosphate dehydrogenase increased remarkably from nodular hyperplasia with no relation to differentiation. The degree of enzyme deviation from adult male liver differed among hepatomas induced by the different carcinogens, especially between those induced by 2-FAA and 3′-Me-DAB. These differences were most evident in the isozyme deviation patterns of GATP and pyruvate kinase. Some of the nodules and hepatomas retained exceptionally high activities of some hepatic isozymes, especially of the liver type of pyruvate kinase. These findings indicate that the enzyme deviation patterns in the hyperplastic liver nodules and primary hepatomas are similar to those found in transplantable hepatomas. (50 refs.)

78-4287 **Aldehyde Dehydrogenase in 2-Acetylaminofluorene-induced Rat Hepatomas. Characterization of Antigens Recognized by Anti-hepatoma Aldehyde Dehydrogenase Sera.** (Eng) Lindahl, R. (Developmental Biology Section, Dept. Biology, Univ. Alabama, University, AL, 35486). *Biochim Biophys Acta* 525(1): 9-17; 1978.

The material in normal rat liver that cross-reacts with anti-hepatoma aldehyde dehydrogenase (ADH) sera has been partially purified. This material is a series of proteins electrophoretically identical to the series of hepatoma-specific ADH's [aldehyde:NAD(P)+ oxidoreductase] induced by 2-acetylaminofluorene. The mol wts (100,000) and the isoelectric points (pI: 6.8-7.2) of the cross-reacting material are identical to the mol wts and p values of the hepatoma-specific isozymes. Absorption of anti-hepatoma ADH sera with the cross-reacting material removes an antibody population that recognizes a normal liver ADH. This isozyme accounts for 35% of the total normal liver ADH. A second antibody population recognizes an antigen that accounts for 10% of the total normal liver ADH. Approx 55% of the normal liver ADH activity is not recognized by any antibody population. In hepatomas, 15% of the total ADH activity is immunochemically identical to the normal liver cross-reacting material. The remaining 85% of the tumor ADH is characterizable as hepatoma-specific on the basis of these immunochemical studies. (12 refs.)

78-4288 **Mutagenic Activation of N-Hydroxy-2-acetylaminofluorene in the *Salmonella* Test System: The Role of Deacetylation by Liver and Kidney Fractions from Mouse and Rat.** (Eng) Schut, H. A. (Biochemical Pharmacology Section, Lab. Chemical Pharmacology, Developmental Therapeutics Program, Div. Cancer Treatment, NCI, NIH, Bethesda, MD, 20014); Wirth, P. J.; Thorgeirsson, S. S. *Mol Pharmacol* 14(4): 682-692; 1978.

An attempt was made to relate liver (L) and kidney (K) microsomal deacetylases, and their possible induction, to the mutagenic activation of N-hydroxy-2-acetylaminofluorene (OH-AAF) in vitro, using C57BL/6N and DBA/2N mice and Sprague-Dawley rats and the Salmonella (strain TA1538) mutagenesis assay. The mutagenicity of OH-AAF mediated by L or K microsomes was independent of treatment of mice or rats with microsomal enzyme inducers (3-methylcholanthrene, phenobarbital, Arochlor) and, at equivalent protein concentrations, was the same whether mouse L or mouse K microsomes were used. Mutagenesis with L or K microsomes from both species was inhibited completely by 0.1 mM paraoxon and partially by 3′-phosphoadenosine 5′-phosphosulfate (25-75 times the molar concentration of OH-AAF) when the rat $< 9{,}000 \times g$ supernatant was used. Although the rates of deacetylation of 2-acetylaminofluorene and OH-AAF were lower in K microsomes than in L microsomes of either species, both reactions were also inhibited completely by paraoxon. Ascorbic acid (10 mM) approx doubled the mutagenicity of OH-AAF in the presence of mouse K microsomes, but it had no effect in their absence. Butylated hydroxytoluene (10 mM) had very little or no effect, with or without ascorbic acid, on OH-AAF mutagenicity in the presence of L or K microsomes. It is concluded that in L and K fractions from the mouse and rat, the most important enzymatic step in the metabolic activation of OH-AAF to a mutagen in the Salmonella test system is deacetylation. Apparently, the OH-AAF-derived nitrenium ion, rather than the nitroxyl free radical, is the more important electrophilic species interacting with the bacterial DNA, resulting in frameshift mutations. (46 refs.)

78-4289 **Mutagenesis by N-Acetoxy-2-acetyl Aminofluorene of Chinese Hamster V79 Cells is Unaffected by Caffeine.** (Eng) Myhr, B. C. (Biology Branch, Somatic Cell Genetics Section, NCI, Bethesda, MD, 20014); DiPaolo, J. A. *Chem Biol Interact* 21(1): 1-18; 1978.

The effect of caffeine (CF) on the mutagenicity of N-acetoxy-2-acetylaminofluorene (AcAAF) was determined in Chinese hamster V79 cells using a reseeding assay in which the cells were reseeded prior to selection to achieve max expression without interference from metabolic crossfeeding. An in situ assay was also used for comparison. AcAAF (0.1-2.5 μg/ml) induced 8-azaguanine (AZG)- and 6-thioguanine (TG)-resistant cells in clone V79-4 cells in the presence of 5% fetal bovine serum. The number of mutants increased from 1 to $47/10^5$ survivors at 55-130 hr after treatment. The effect of CF (50-200 μg/ml) on mutation frequency was determined by adding CF 1-24 hr after AcAAF. The medium was replaced after 48 hr so that CF was absent during subsequent selection with AZG or TG. There was no significant change in AcAAF mutagenesis with any treatment combination, although CF greatly enhanced the lethality of AcAAF. CF appears to inhibit a postreplication repair process in the V79-4 cells, causing potentiation of AcAAF lethality without altering the error probability of the remaining postreplication repair activity. The in situ assay was subject to serious interference from crossfeeding, and it yielded an artifactual tenfold enhancement of AcAAF mutagenesis by CF. (32 refs)

78-4290 **A Comparative Study of Cyclophosphamide Using In Vivo and In Vitro Metabolic Activating Systems and *Salmonella typhimurium* Indicators** (Meeting Abstract). (Eng) Steelman, J. R. (McNeil Lab., Inc., Fort Washington, PA, 19034); Schreiner, C. A. *Mutat Res* 53(1): 72-73; 1978. (no refs)

78-4291 **Testing In Vitro of an Indirect Mutagen (Cyclophosphamide) with Human Leukocyte Cultures. Activation by Liver Perfusion and by Incubation with Crude Liver Homogenate.** (Eng) Madle, S. (Institut fur Genetik, Freie Universitat Berlin, Arnimallee 5-7, 1000 Berlin 33, W. Germany); Westphal, D.; Hilbig, V.; Obe, G. *Mutat Res* 54(1): 95-99; 1978.

Two possible methods for activating cyclophosphamide (CP) and testing its mutagenicity in human WBC in vitro are reported: (1) perfusion of rat liver in situ with CP and direct addition of the perfusate to human whole blood cultures and (2) activation of CP with a crude homogenate of mouse liver and addition of the mixture to the cultures by means of dialysis bags. Male Wistar rat livers were perfused with 0.63-20 mM CP for 5 min or 1 hr. Blood cultures (2.5 ml) were incubated for 80 hr (including an 8-hr colcemid treatment), treated with 1 ml perfusate for 3 hr, and allowed to recover for 29 hr. The final CP concentration in the cultures was 0.18-5.71 mM. The perfusate led to a dose-dependent suppression of the mitotic index and to the induction of chromatid translocations. The results were similar regardless of perfusion time, indicating that the metabolic transformation of CP was completed in 5 min or that a steady state was reached between the formation of chromosome-breaking products and their decomposition. In the second method, crude liver homogenates were obtained from phenobarbital-treated female NMRI mice. The homogenates were tested with and without 5 mM NADP and 10 mM glucose 6-phosphate. CP (0.16-20 mM) was added, 1 ml of the mixture was put into a dialysis bag, and the bag was submersed in colcemid-treated cultures for 3 hr at incubation hours 48-51. The final CP concentration was 0.05-5.71 mM. The results were similar to those of the perfusion experiments, except that the effects of the liver homogenates without cofactors were weaker. Mitotic indices were suppressed only at 10 and 20 mM CP, and chromatid translocations were induced only at 20 mM. (8 refs)

78-4292 **Carcinogenic Action of Low-Dose Cyclophosphamide (CP) in Rats (Meeting Abstract).** (Eng) Habs, M. (Inst. Toxicology and Chemotherapy, German Cancer Res. Center, Im Neuenheimer Feld 280, 69 Heidelberg, W. Germany); Schmahl, D. *Proc Am Assoc Cancer Res* 19: 14; 1978. (no refs)

78-4293 **Azaserine Induced Damage in DNA of Cultured Rat Pancreas (Meeting Abstract).** (Eng) Lilja, H. S. (Dartmouth Medical Sch., Hanover, NH, 03755); Kuhlmann, E. T.; Yager, J. D.; Longnecker, D. S. *Proc Am Assoc Cancer Res* 19: 174; 1978. (1 ref)

78-4294 **Acute Leukemia after Chemotherapy (Melphalan).** (Eng) Einhorn, N. (Dept. Gynecological Oncology, Radiumhemmet Karolinska Hosp. and Inst., Stockholm 60, Sweden). *Cancer* 41(2): 444-447; 1978.

The incidence of acute leukemia was investigated in 48 patients who were seen between 1966-1973, who received a minimum of 300 mg melphalan, and who survived at least 3 yr. Eleven of the patients had received melphalan only, 2 also had received other cytostatic agents, 30 also had been irradiated, and 5 had received melphalan, other cytostatics, and irradiation. The melphalan was usually given iv at a dose of 1 mg/kg. Of the 48, 4 developed acute leukemia 3-6.5 yr after initiation of treatment; no other new primary malignant tumors were noted. One of the four had also received cyclophosphamide, and one had also been irradiated. The 4 patients were among the 12 who had received at least 800 mg melphalan. Case reports of the four women are presented. It is suggested that if the melphalan did cause the leukemia, the latency period was remarkably short. In the evaluation of chemotherapy as adjuvant treatment, the risk of developing new primary tumors must be considered. (11 refs)

78-4295 **Levodopa Therapy and Malignant Melanoma.** (Eng) Sober, A. J. (Dept. Dermatology, Massachusetts General Hosp., Boston, MA, 02114); Wick, M. M. *JAMA* 240(6): 554-555; 1978.

A questionnaire/interview study was conducted to determine whether levodopa (L-3,4-dihydroxyphenylalanine) is involved in the induction or stimulation of growth of cutaneous malignant melanoma. Prospective query of 1,099 patients of the Melanoma Clinical Cooperative Group at the time of presentation of their primary melanoma showed only 1 patient who had been taking levodopa. It was concluded that if levodopa is a factor in the induction of melanoma, it must be playing an inconsequential role in the rapid rise in incidence observed for this tumor over the past decade. (13 refs.)

78-4296 **High Levels of Reduced Glutathione in Glandular Rat Stomach; Possible Implications for Gastric Carcinogenesis (Meeting Abstract).** (Eng) Boyd, S. (Field Studies and Statistics, NCI, Bethesda, MD, 20014); Sasame, H. *Proc Am Assoc Cancer Res* 19: 121; 1978. (1 ref)

78-4297 **Determination of N-Nitrosamines in Water by Fluorescent Derivatives.** (Rus) Melamed, D. B. (Inst. Nutrition, Moscow, USSR); Kostiukoskii, Ia. L. *Gig Sanit* (5): 67-68; 1978.

A sensitive assay for determining the fluorescent derivatives of various N-nitrosamines in water is described. The derivatives were separated by thin-layer chromatography. The detection limit of the technique was 1 μg/liter. (3 refs)

78-4298 **Mutagenicity of Dimethylnitrosamine and Ethyl Methanesulfonate as Determined by the Host-Mediated CHO/HGPRT Assay.** (Eng) Hsie, A. W. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Machanoff, R.; Couch, D. B.; Holland, J. M. *Mutat Res* 51(1): 77-84; 1978.

The mutagenicity of the procarcinogen dimethylnitrosamine (DMN) was compared with that of the direct-acting alkylating agent ethyl methanesulfonate (EMS) using the host (mouse)-mediated Chinese hamster ovary/hypoxanthine-guanine phosphoribosyltransferase assay system. Both nude (athymic) and phenotypically normal BALB/c mice were used in the assays. The induced mutation frequency increased linearly with DMN dose up to 250 mg/kg using a 2-hr treatment time. For DMN doses of 100 and 500 mg/kg, the induced mutation frequency rose linearly with time up to 6 hr and continued to increase when cells were left in the peritoneal cavity for an additional 18 hr. DMN was not mutagenic when tested in vitro, but EMS-induced mutations increased in a time- and dose-dependent fashion both in vitro and in the host-mediated assay. (20 refs.)

78-4299 **Removal of Methylated Purines from Rat Liver DNA after Administration of Dimethylnitrosamine.** (Eng) Pegg, A. E. (Dept. Physiology, Milton S. Hershey Medical Center, Hershey, PA, 17033); Hui, G. *Cancer Res* 38(7): 2011-2017; 1978.

The removal of abnormal methylated purines induced by ip injection of various doses of dimethylnitrosamine (DMN) from female Sprague-Dawley rat liver DNA was investigated. All methylated purines formed in the DNA were lost to some extent over a 24-hr period, but the rate of removal varied from purine to purine. The rates of loss of 1-methyladenine, 3-methyladenine, 7-methyladenine, 3-methylguanine, and 7-methylguanine (7-MG) were not changed significantly when the extent of methylation was increased $>$20-fold by increasing the dose of DMN from 0.75 to 20 mg/kg. However, O^6-methylguanine (O^6-MG) was lost rapidly from the DNA after the lower DMN dose, but at a much slower rate after the higher dose. When chromatin was prepared from livers of rats treated with labeled DMN and partially digested with DNase I or micrococcal I or micrococcal nuclease, no difference in the level of 7-MG or O^6-MG between the nuclease-resistant and nuclease-sensitive portions of the chromatin DNA was seen. Changing the dose of DMN from 25 μg/kg to 20 mg/kg did not alter the relative distribution of these methylated guanines. Although a substantial amount of O^6-MG was lost from the chromatin DNA within 24 hr at the lower doses, there was no indication that this purine was lost more readily from either fraction of the chromatin DNA. When labeled O^6-MG, produced in hepatic DNA by administration of 0.75 mg/kg ^{14}C-DMN, was diluted by unlabeled O^6-MG, formed by a 20-mg/kg DMN dose given 1 hr later, the rate of loss of labeled O^6-MG was greatly reduced. It was concluded that O^6-MG is removed from hepatic DNA by a mechanism that is more efficient after lower DMN doses either because the removal system becomes saturated at higher levels of DNA methylation or because it is inhibited by he larger doses of this carcinogen. (49 refs)

78-4300 ***In Vivo* Reaction of Dimethylnitrosamine with Nucleic Acids.** (Eng) Grilli, S. (Istituto di Cancerologia, Universita degli Studi di Bologna, via S. Giacomo 14, 40126 Bologna, Italy); De Giovanni, C.; Prodi, G.; Giamanini, A. G. *Gann* 69(1): 39-45; 1978.

The in vivo reaction of dimethylnitrosamine (DMNA) with nucleic acids was studied to determine whether the methylamine nitrogen formed was derived from the DMNA. ^{14}C-^{15}N-DMNA (30 mg/kg) was injected ip into female Wistar rats, and the animals were killed 12 hr later. Their livers were extracted and pooled; DNA, ribosomal RNA, nuclear proteins, and cytoplasmic proteins were extracted; and the amount of radioactivity bound to the macromolecules was measured. Following strong acid hydrolysis, 7-methylguanine was the main compound resulting from the interaction of DMNA with the nucleic acids. Enzymatic and alkaline hydrolysis, however, resulted in the quantitative liberation of methylamine. Methylamine isolated from the liver nucleic acids of ^{15}N-DMNA-treated rats had a mol wt of 31, indicating that DMNA nitrogen is not involved in the binding. Since methylamine nitrogen cannot be derived from 7-methylguanosine fission, these findings disagree with the generally proposed mechanism of alkylation. (16 refs)

78-4301 **Urinary Excretion of N-Nitrosodiethanolamine Administered Orally to Rats.** (Eng) Preussmann, R. (Inst. Toxicology and Chemotherapy, German Cancer Res. Center, D-6900 Heidelberg, W. Germany); Wurtele, G.; Eisenbrand, G.; Spiegelhalder, B. *Cancer Lett* 4(4): 207-209; 1978.

N-Nitrosoethanolamine (NDE1A) was administered to male BD-VI rats, and its urinary excretion rate was measured to determine if a high rate could account for the low carcinogenicity of this compound. The rats received single doses of 1,000, 500, or 100 mg/kg NDE1A by gavage. Approx 70% of the dose was excreted unchanged in the urine within 24 hr. This percentage appeared to be independent of the absolute dose, at least within the range 10-1,000 mg/kg. However, since some of the urine may have been absorbed by the feces, the real excretion values may be somewhat higher. The amount collected within 24 hr represented 95% of the excreted dose; no urinary elimination of NDE1A occurred after 48 hr. These findings suggest that the low carcinogenicity of NDE1A is the result of a high and fast excretion rate, probably because of its low resorption through cellular membranes. (8 refs)

78-4302 **The Acute Effects of the Carcinogen DIPN on Pancreatic Secretion in Hamsters (Meeting Abstract).** (Eng) Roberts, C. (VA Hosp., San Francisco, CA); Reber, H. A. *Gastroenterology* 74(5, part 2): 1142; 1978. (no refs)

78-4303 **Chromosomal Aberrations in Rat Lymphocytes Treated In Vivo with 1-Phenyl-3,3-dimethyltriazene and N-Nitrosomorpholine. A Further Report on a Possible Method for Carcinogenicity Screening.** (Eng) Newton, M. F. (Dept. Biology as Applied to Medicine, Middlesex Hosp. Medical Sch., London W1P 6DB, England); Bahner, B.; Lilly, L. J. *Mutat Res* 56(1): 39-46; 1977.

The carcinogens N-nitrosomorpholine (NM) and 1-phenyl-3,3-dimethyltriazene (PDT) were screened for their ability to cause an increase in chromosomal aberrations in rat lymphocytes in vitro after treatment in vivo. NM was injected ip in doses of 200, 250, 300, and 400 mg/kg, and the rats were sacrificed 3 or 20 hr later. PDT was injected ip in doses of 3.5, 7.0, 35, and 50 mg/kg, and the animals were sacrificed 6 hr later. Lymphocyte preparations were cultured for 48 hr in the presence of phytohemagglutinin, and colchicine was added 1.5 hr before harvesting. NM caused a significant increase in the number of chromosomal aberrations after a single dose of 250 mg/kg or higher. These aberrations were mainly of the isolocus type. Significant increases in the number of isolocus- and chromatid-type aberrations were found after single doses of 35 and 50 mg/kg PDT, which are about half the amounts found to cause revertants of *Saccharomyces cerevisiae* in the host-mediated assay in mice. The positive results with PDT, which causes tumors mainly of the central and peripheral nervous systems, could imply the existence of an active circulating metabolite of PDT detectable by the chromosomal damage it causes to rat lymphocytes. The existence of such a long-lived metabolite may be necessary for the induction of tumors in the central and peripheral nervous systems. (18 refs)

78-4304 **Differential Metabolism of Geometrical Isomers of N-Nitroso-2,6-dimethylmorpholine in the Hamster.** (Eng) Gingell, R. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB, 68105); Nagel, D.; Kupper, R. *Xenobiotica* 8(7): 439-443; 1978.

The metabolism of the cis and trans isomers of the pancreatic carcinogen N-nitroso-2,6-dimethylmorpholine (NDMM) was studied in randomly bred male Syrian golden hamsters. The animals were inoculated ip with 100 mg/kg of either isomer as a 10-mg/ml soln in saline-propylene glycol (2:1). Both isomers were metabolized by B-oxidation and ring scission to N-nitroso-(2-hydroxypropyl)(2-oxopropyl)amine (HPOP), the suggested proximate pancreatic carcinogen, and to N-nitrosobis(2-hydroxypropyl)amine (BHP). Although the cis isomer produced slightly higher blood levels of NDMM and HPOP than the trans isomer, the differences are probably not biologically significant. The amounts of metabolites in the 24-hr urine were similar, and there was no unchanged NDMM, indicating little difference in the overall amount of metabolism of each isomer by β-oxidation. However, the initial rate of β-oxidation and urinary excretion of the cis isomer was much greater than that for the trans isomer, probably because a relative ease of enzymatic axial attack. Alternatively, the trans isomer may bind more weakly to the hepatic mixed-function oxidase, diminishing the rate of metabolism. However, since the accumulated 24-hr urine metabolites were similar, any anticipated difference in the carcinogenic potency of cis- and trans-NDMM in the hamster pancreas is likely to be minimal. (13 refs.)

78-4305 **Mutagenicity Screening on Pesticides and Modification Products: A Basis of Carcinogenicity Evaluation.** (Eng) Shirasu, Y. (Toxicology Div., Inst. Environmental Toxicology, Kodaira-shi, Tokyo 187, Japan); Moriya, M.; Kato, K.; Lienard, F.; Tezuka, H.; Teramoto, S.; Kada, T. *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 267-285; 1977.

The results of microbial mutagenicity screening studies, cytogenetic and dominant lethal studies of Captan [N-(trichloromethylthio)-4-cyclohexene-1,2-dicarboximide] and ethylenethiourea (ETU), and mutagenicity studies of the nitrosation products of pesticides are presented. The rec and reversion assays were used to screen 193 pesticides for mutagenicity in two strains of *Escherichia coli* and four strains of *Salmonella typhimurium* without metabolic activation. Fifteen pesticides were mutagenic: Captafol, Captan, Dexon, Dichlorvos, Folpet, N-(2-hydroxyethyl)hydrazine, 2,4-dinitrophenyl thiocyanate, 5-nitro-1-naphthonitrile, Vamidothion, Ferbam, Ziram, arsenic dimethyldithiocarbamate, bis(dimethylthiocarbamoyl)disulfide, N,N′-bis(dimethyldithiocarbamoyl)ethylenediamine, and Dichlofluanid. When liver homogenates from phenobarbital- or polychlorinated biphenyl-treated rats were used, the mutagenicity of some of the pesticides was either lost or enhanced. In cytogenetic studies of Captan, the incidence of chromosome aberrations in human diploid fibroblast cells treated in vitro (1.5, 3.0, or 4.0 μg/ml for 4 or 24 hr) was not increased significantly. Similar findings were obtained in vivo with male rats. Captan did not increase the rate of embryonic death at any spermatogenesis stage tested. Negative results were also obtained in in vivo cytogenetic and dominant lethal studies of ETU, which might be due to its organ affinity: metabolic studies revealed that ETU accumulated in the thyroid. Preliminary results of the nitrosation and mutagenicity testing of 120 pesticides that were negative in the rec and reversion assays are given. (42 refs.)

78-4306 **Testing of Known Carcinogens and Noncarcinogens for Their Ability to Induce Unscheduled DNA Synthesis in HeLa Cells.** (Eng) Martin, C. N. (Cancer Res. Unit, Univ. York, Heslington, York, Y01 5DD, England); McDermid, A. C.; Garner, R. C. *Cancer Res* 38(8): 2621-2627; 1978.

The ability of 51 chemical carcinogens and noncarcinogens to induce unscheduled DNA synthesis (UDS) in HeLa cells was tested in the presence or absence of a rat liver mixed function oxidase preparation. The chemicals tested included those giving erroneous results in bacteria mutagenicity assays and representative compounds from four classes of chemical carcinogens: nitrosamines polycyclic aromatic hydrocarbons, aromatic amines and mycotoxins. None of the noncarcinogens induced UDS. Of 38 documented carcinogens, 34 were active safrole, N-propyl-N-nitrosourea, aflatoxin B_2, and N butyl-1-N-nitrosourea were inactive. Six compounds for which carcinogenicity data are incomplete were active: 4-nitro-o-phenylenediamine, 2-nitro-p-phenylene diamine, formaldehyde, 2,2′-dichlorobenzidine, 3,3′, 5,5′-tetrafluorobenzidine, and 3,3′,5,5′-tetrachlorobenzi dine. Urethan, N-dimethyl-p-aminoazobenzene, and di ethylstilbestrol, carcinogens that are weakly active or inactive in bacterial mutagenicity assays, were active in the UDS assay. The bacterial mutagens sodium azide and 9-aminoacridine were inactive. The use of this assay in a tier scheme for the short-term testing of potential chemical carcinogens is discussed. (38 refs)

78-4307 **Mutagenicity and Cytotoxicity of Congeners of Two Classes of Nitroso Compounds in Chinese Hamster Ovary Cells.** (Eng) Couch, D. B. (Chemical Industry Inst. Toxicology, Research Triangle Park, NC, 27709); Hsie, A. W. *Mutat Res* 57(2): 209-216; 1978.

The induction of mutation by certain nitrosamidines and nitrosamides was quantitated using the hypoxanthine-guanine phosphoribosyltransferase locus in Chinese hamster ovary (CHO-K_1-BH_4) cells. Dose-response relationships for cytotoxicity and mutagenicity were observed for N-methyl-N-nitrosourea (MNU), N-ethyl-N-nitrosourea (ENU), N-butyl-N-nitrosourea (BNU), N-methyl-N′-nitro-N-nitrosoguanidine (MNNG), and N-ethyl-N′-nitro-N-nitrosoguanidine (ENNG). Although all the agents produced an exponential loss of clonal growth with increasing dose, nitrosoguanidines were more toxic than nitrosoureas, and within each class of compound, cytotoxicity declined with increasing size of the alkyl group. Based on the concentration of each agent required to kill 90% of the cells, the following order of cytotoxicity was observed: MNNG > ENNG > MNU > ENU > BNU. This is the same order of potency observed for mutation induction per unit concentration of mutagen. (14 refs.)

78-4308 **Fibrinolytic Activity of Cultured Cells Derived During Ethylnitrosourea-induced Carcinogene-**

sis of Rat Brain. (Eng) Hince, T. A. (Dept. Cell Pathology, Sch. Pathology, Middlesex Hosp. Medical Sch., Riding House St., London, W1P 7LD, England); Roscoe, J. P. *Br J Cancer* 37(3): 424-433; 1978.

Using a sequential in vivo/in vitro cell culturing approach, the fibrinolytic activity of cell lines derived from an ethylnitrosourea (ENU)-induced glioma, and cell lines derived during the latent period was determined. The intermediate cell lines were derived from fetuses or young rats 2-145 days after administration of 40 mg/kg ENU to the mothers after day 12 of gestation. High levels of plasminogen-dependent fibrinolytic activity were demonstrated in lines derived from the ENU-induced gliomas, but lines from normal adult rat brain showed only low levels of activity. The degree of lysis produced by a given cell line was dependent on the av number of cells per colony, and a different pattern of response was observed for tumor and normal cell lines. A positive correlation existed among the level of fibrinolytic activity, growth in agar, and tumorigenicity of a cell line. Fibrinolytic activity was associated with cell lines derived at various times in the latent period, before the appearance of a visible tumor. Many cell lines derived from rat brains at 57-60 (E7), 90-91 (E8), and 111-112 (E6) days after transplacental ENU exposure showed fibrinolytic activity, and in the latter group the close association with growth in agar and tumorigenicity was also demonstrated. With lines E7 and E8, the possession of fibrinolytic activity preceded the ability of cells to form colonies in agar. (26 refs)

78-4309 **Effect of Chloramphenicol on the Carcinogenicity of N-Nitrosomethylurea.** (Rus) Bogush, T. A. (Dept. Carcinogenic Agents, Cancer Res. Center, Moscow, USSR); Belitskii, G. A.; Shabad, L. M. *Biull Eksp Biol Med* 86(7): 61-63; 1978.

The effect of the antibiotic chloramphenicol (CA) on the carcinogenicity of N-nitrosomethylurea (NMU) was studied in CBA mice. Animals received CA (1 mg/g/day, intragastrically) for 5 days; 2 hr after the last CA administration, mice were inoculated sc with NMU (0.025-0.07 mg/g). NMU induced lymphomas, leukemias, hepatomas, renal cystadenomas, solid carcinomas, and adenocarcinomas of the uterus, ovaries, and prestomach. The av latent period of tumor development in mice exposed to 0.07 mg/g NMU was 158 days, compared with 208 days in mice treated with NMU + CA (incidence of tumor-bearing animals was 36.5% and 27.5%, respectively). The av latent period of tumor development in mice exposed to 0.025 mg/g NMU was 35.8 wk, compared with 54 wk in animals exposed to NMU + CA (incidence of tumor-bearing animals was 20% and 12% respectively). (8 refs.)

78-4310 **Human Exposure to N-Nitroso Compounds in the Environment.** (Eng) Fine, D. H. (Cancer Res. Dept., Thermo Electron Res. Center, Waltham, MA, 02154); Rounbehler, D. P.; Fan, T.; Ross, R. *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 293-307; 1977.

Two new aspects of human exposure to N-nitroso compounds are reported: in vivo nitrosation in humans due to ingestion of nitrite-preserved foodstuffs and the presence of excessive N-nitrosamine contamination in widely used herbicides. Chromatographic analysis of blood samples taken before and after ingestion of a midday meal consisting of 310 g spinach, 170 g cooked bacon, 200 g tomatoes, 120 g bread, and 460 g beer indicated that about 7 μg of nitrosamine [both N-nitrosodimethylamine (NDMA) and N-nitrosodiethylamine] were formed in vivo. However, the total amount of nitrosamine formation could have been greater, because the calculations did not take into account nitrosamine loss by metabolic processes or exhalation or the absorption of nitrosamine by fat and other tissues. Analysis of various herbicides formulated for home use revealed the presence of NDMA at levels as high as 0.06%. On the basis of these findings, it is suggested that epidemiologic studies be initiated to follow persons who may have been exposed to high levels of nitrosamine-containing formulations in the working environment (agricultural as well as manufacturing and formulating workers). Literature data on the presence of N-nitroso compounds in food, tobacco, air, water, and soil are reviewed. (31 refs.)

78-4311 **Oncogenicity of Purine 3-Oxide and Unsubstituted Purine in Rats.** (Eng) Teller, M. N. (Walker Lab., Sloan-Kettering Inst. Cancer Res., Rye, NY, 10580); Giner-Sorolla, A.; Stohrer, G.; Budinger, J. M.; Brown, G. B. *Cancer Res* 38(8): 2229-2232; 1978.

Sc injection of purine 3-oxide (7.5 or 0.75 mg 3x/wk for 8 wk) into Wistar rats resulted in the appearance of sarcomas and fibromas at the intercapsular site of administration, hepatocellular carcinomas, and a high incidence of sc fibromas in the hip area. A small number of liver tumors but no tumors at the injection site appeared in rats that received 6.5 or 0.65 mg of the parent compound, purine. Oxidation of purine 3-oxide by xanthine oxidase occurred in two steps to yield the potent oncogen 3-hydroxyxanthine. A similar process may occur in vivo, since a protein preparation from rat sc tissue has similar oxidizing activity. (19 refs)

78-4312 **Effect of Phorbol Myristate Acetate on Cyclic Nucleotide Levels in Mouse Epidermis.** (Eng) Belman, S. (Dept. Environmental Medicine, New York Univ. Medical Center, New York, NY, 10016); Troll, W.; Garte, S. J. *Cancer Res* 38(9): 2978-2982; 1978.

The effect of a single topical administration of phorbol myristate acetate (PMA) on cyclic AMP (cAMP) and cyclic guanosine 3′:5′-monophosphate (cGMP) levels in the epidermis 0-72 hr after treatment were determined in female Swiss Ha/ICR mice. An acetone soln of PMA (10 μg/0.2 ml) was applied to the depilated backs of the test animals. No changes in cAMP levels were found 0-72 hr after treatment, but a twofold increase in cGMP levels was apparent by 36 hr. However, this increase had subsided by 72 hr. PMA treatment stimulated DNA synthesis in a cyclic manner. There was a possible slight depression in thymidine incorporation into DNA at 3 hr followed by three peaks at 18, 42, and 60 hr. Incorporation of cytidine into RNA was slightly depressed at 1 hr, followed by two peaks at 3 and 18 hr. Values declined to control levels by 30 hr and remained unchanged. Protein synthesis was increased by 1 hr after PMA application, and it reached a max at 24 hr before declining to control values by 72 hr. (17 refs.)

78-4313 **Induction of Preneoplastic Hyperplasia and Carcinoma by N-Methyl-N′-nitro-N-nitrosoguanidine from Regenerated Mucosa of Ulcers Induced by Iodoacetamide in Fundus of Rat Stomach.** (Eng) Shirai, T. (First Dept. Pathology, Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467, Japan); Takahashi, M.; Fukushima, S.; Tatematsu, M.; Hirose, M.; Ito, N. *Gann* 69(3): 361-366; 1978.

N-Methyl-N′-nitro-N-nitrosoguanidine (MNNG)-induced carcinogenesis in the regenerating mucosa of iodoacetamide (IAA)-induced stomach ulcers was studied in male Wistar rats. Both IAA (0.01%-0.1%) and MNNG (50 mg/liter) were administered in the drinking water. There were five groups of 25 animals. Group 1 was given IAA during weeks 1-12 and MNNG during weeks 16-28. Group 2 was given IAA and MNNG during weeks 16-28. Group 3 was given MNNG during weeks 16-28 and IAA during weeks 32-44 Group 4 was given MNNG during weeks 16-28, and Group 5 was given IAA during weeks 1-12. All animals were sacrificed at 60 wk. Pyloric gland metaplasia was found in the fundic ulcers of 15/17 Group 1 animals, 12/17 Group 2 animals, 10/15 Group 3 animals, and 15/18 Group 5 animals. Proliferative lesions, (atypical metaplasia) in the pyloric gland metaplasias were found in 10/17 Group 1 animals, 5/17 Group 2 animals, and 1/15 Group 3 animals. An adenocarcinoma was observed in the ulcerative region of two Group 1 animals. There was no significant difference in the incidence of hyperplasia, adenoma, and adenocarcinoma in the pyloric region between Groups 1, 2, and 3 animals and those in Group 4. An adenocarcinoma was also observed in the duodenum of a Group 2 rat. These findings indicate that gastric cancers can develop from ulcerative lesions and that pyloric gland metaplasia is more susceptible to MNNG than the rapidly regenerating mucosa at the margin of an ulcer. (10 refs.)

78-4314 **Quantification of Chemical Mutagenesis in Diploid Human Fibroblasts: Induction of Azaguanine-resistant Mutants by N-Methyl-N′-nitro-N-nitrosoguanidine.** (Eng) Jacobs, L. (Dept. Cytogenetics, Inst. Medical Res., Camden, NJ, 08103); Demars, R. *Mutat Res* 53(1): 29-53; 1978.

The toxic and mutagenic effects of N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) were studied using cultured diploid human fibroblasts. When 10^5 cells were exposed to $<1 \times 10^{-6}$ M MNNG for 4 hr. cell survivals of almost 100% were observed; concentrations $>5 \times 10^{-6}$ M resulted in av cell survivals of <1%. Cells were then treated with 3.0 x 10^{-6} M MNNG and selected for resistance to azaguanine and thioguanine. The induced mutant frequency was maximized by allowing a 5-7-day posttreatment interval for the expression of the mutant phenotype and by replating the cells at the beginning of selection at a population density of <450 cells/cm^2. Independent dose-response experiments gave induced mutant frequencies as high as 7.0 x 10^{-4} to 8.8 x 10^{-4} mutants per viable cell at about 5% survival, compared with a spontaneous mutation rate of 3.7 x 10^{-6} to 7.2 x 10^{-6} mutants per generation. The azaguanine-resistant mutants observed after MNNG treatment were phenotypically stable, and they closely resembled prototype azaguanine-resistant cultures from humans with inherited mutant X-chromosomal alleles for hypoxanthine-guanine phosphoribosyltransferase. (55 refs)

78-4315 **Base and Base Sequence Specificity of the Binding of 4-Hydroxyaminoquinoline 1-Oxide to DNA.** (Eng) Gotoh, O. (Dept. Physics, Faculty Science, Univ. Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113, Japan); Wada, A.; Tada, M.; Tada, M. *Gann* 69(1): 61-66; 1978.

The binding of 4-hydroxyaminoquinoline 1-oxide (4-HAQO), a metabolite of 4-nitroquinoline 1-oxide, to synthetic and natural DNA's was examined in vitro. With a standard enzyme concentration, the binding of 4-HAQO to poly(deoxyguanosine-deoxycytosine) [poly(dG-dC)]-poly(dG-dC), poly(dG)-poly(dC), poly(deoxyadenine-deoxythymidine) [poly(dA-dT)]-poly(dA-dT), λ-DNA, and poly(dA)-poly(dT) decreased in this order. These findings also indicate that a difference in base sequence leads to a different amount of binding: only a very small amount of 4-HAQO could bind to poly(dA)-poly(dT), but >10 times this amount could bind to poly(dA-dT)-poly(dA-dT). The optical melting profiles of the 4-HAQO-modified DNA's were examined. Decreases in the melting temperature and broadening of the transition width were commonly observed. The melting fine structure of the λ-phage DNA became less clear with modification. The shape of the melting curves of synthetic polynucleotides was not changed much by binding, suggesting that the binding sites are distributed randomly along a DNA molecule. The binding of 4-HAQO to a purine base may distort the secondary structure of the neighboring base pairs. The degree of distortion, estimated as a free energy increment associated with binding, differed considerably mong polynucleotides with different base sequences. (18 efs)

8-4316 **Microsomal N-Hydroxylation of trans-4′-Alkoxy-4-acetamidostilbenes.** (Eng) Gammans, R. . (USV Pharmaceutical Corp., Tuckahoe, NY); Sehon, R. .; Anders, M. W.; Hanna, P. E. *Drug Metab Dispos* 6(3): 26-231; 1978.

he influence of 4′-alkoxy substituents on the in vitro metab-ism of a series of trans-4-acetamidostilbenes (AAS's) by amster liver microsomes was studied. 4′-methoxy-AAS was onverted to a single ether-extractable metabolite, 4′-me-oxy-4-(N-hydroxy-AA)S, and 4′-ethoxy-AAS was convert-l to 4′-ethoxy-4-(N-hydroxy-AA)S, 4′-hydroxy-AAS, and -ethoxy-AAS α,β-epoxide. 4′-Butoxy-AAS was metabol-ed to the hydroxamic acid, 4′-butoxy-4-(N-hydroxy-AA)S, id it underwent (ω-1)-hydroxylation of the aliphatic chain, oducing 4′-(3-hydroxybutoxy)-AAS. 4′-Propoxy-AAS was onverted to 4′-propoxy-4-(N-hydroxy-AA)S and 4′ ropoxy-AAS α,β-epoxide. Attempts to examine the kinet-s of metabolism of 4′-methoxy-AAS and 4′-ethoxy-AAS oved unsuccessful. In contrast to these analogs, the metab-ism of 4′-butoxy-AAS followed Michaelis-Menten kinetics. -Alkoxy-AAS's were not metabolized when heat-inactivat-l microsomes were used or when the NADPH-generating stem was omitted. Although each substrate was converted a potentially toxic or carcinogenic arylhydroxamic acid, e rates of both bioactivation and the formation of presuma-y nonhazardous carbon oxidation products may be signifi-ntly influenced by suitable modification of the physico-emical properties of the substrates. (20 refs.)

3-4317 **Bilary Excretion of N,N-Dimethyl-4-aminoazobenzene (DAB) in the Rat. Effects of Pretreat-ent with Inducers and Inhibitors of the Mixed-Function xidase System and with Agents That Deplete Liver Glu-thione.** (Eng) Levine, W. G. (Dept. Molecular Pharmacolo-, Albert Einstein Coll. Medicine, Yeshiva Univ., Bronx, Y, 10461); Finkelstein, T. T. *Drug Metab Dispos* 6(3): 55-272; 1978.

ne biliary excretion of N,N-dimethyl-4-aminoazobenzene)AB) metabolites following a single iv injection of the C-labeled form of this dye was studied in male Wistar rats. ne biliary excretion of amaranth, an azo dye excreted almost tirely in nonmetabolized form, was also studied. After in-ction of ^{14}C-DAB in control animals, about 20%-25% of e radioactivity (RA) was excreted in 1 hr. In animals treat-l with 3-methylcholanthrene or phenobarbital to induce the ixed-function oxidase (MFO) system, the biliary excretion RA was markedly enhanced, especially in the early phase the collection period. In rats treated with the MFO inhibi-rs piperonyl butoxide or SKF 525-A, the biliary excretion RA was markedly suppressed throughout the collection period. Biliary excretion of RA was also suppressed after pretreatment with methyl iodide or diethyl maleate, which decrease liver glutathione (GSH) levels. Injection of a mixture of ^{14}C-DAB metabolites from animals treated with ^{14}C-DAB stimulated the excretion of biliary RA to a greater extent than did injection of ^{14}C-DAB. GSH-depleting agents and MFO inducers and inhibitors had no effect on the rate of excretion of injected metabolites. Pretreatment with nafenopin did not affect the excretion of ^{14}C-DAB metabolites, and the binding of DAB or its metabolites to Z protein was limited by the concentration of Z in the liver. The excretion of RA was as rapid after the injection of unconjugated metabolites as after the injection of conjugated metabolites. Free 4-aminoazobenzene was excreted relatively slowly. The results indicate that metabolism is the rate-determining step in the biliary excretion of DAB and that liver GSH is involved in the biliary fate of DAB. (45 refs.)

78-4318 **Studies in Rats of the Mechanism of Organotropy and Metabolism of the Colon Carcinogen Methylazoxymethanol Acetate (Meeting Abstract).** (Eng) Zedeck, M. S. (Sloan-Kettering Inst., New York, NY); Grab, D. J.; Frank, N.; Wiessler, M.; Bertram, B.; Schmahl, D. *Digestion* 16(3): 288; 1977. (1 ref)

78-4319 **Mutagenicity Testing of Certified Food Colors and Related Azo, Xanthene and Triphenylmethane Dyes with the Salmonella/Microsome System (Meeting Abstract).** (Eng) Brown, J. P. (Dynapol, Palo Alto, CA, 94304); Roehm, G. W.; Brown, R. J. *Mutat Res* 53(1): 70; 1978. (no refs)

78-4320 **Direct Black 38, Direct Blue 6, Direct Brown 95, Benzidine Derived Dyes.** (Eng) Thomas, A. W. (Natl. Inst. Occupational Safety and Health, 5600 Fishers Lane, Rockville, MD, 20852); Boeniger, M. F.; Weber, M. G.; Stein, H. P.; Upton, A. C.; Millar, J. D. *Am Ind Hyg Assoc J* 39(8): A18-A24; 1978.

Studies that led to the recommendation by the National Institute for Occupational Safety and Health that the three benzidine (BZD)-derived dyes Direct Black 38, Direct Blue 6, and Direct Brown 95 be handled as if they were human carcinogens are summarized. All three dyes produced significant incidences of cancerous and precancerous changes in the livers of male and female rats given these compounds in the feed for 90 days. The damage was similar to that caused by BZD. In the same feeding test with mice, all three dyes were harmful to the liver but no cancerous changes were found. This finding is compatible with the interpretation that the toxic effects may be related to BZD, as this compound is more likely to produce cancer in rats than mice. Although the dyes were BZD-free when fed to the animals, substantial amounts of BZD were found in the urine of both rats and mice. The urine of workers at two BZD dye manufacturing plants and two textile finishing companies contained BZD or its metabolite monacetybenzidine (MAB). In one of the dye manufacturing plants, workers exposed only to the finished dyes had about 20 times more BZD and up to 200 times more MAB in their urine than if they had been exposed only to the residual BZD content in the dyes. It is suggested that preventing the worker inhalation of the dye particles would greatly reduce BZD absorption by the body. (18 refs.)

78-4321 **Kidney and Liver Tumors in Rats from 4,4′-Methylene Bis(2-carbomethoxyaniline).** (Eng) Stula, E. F. (Haskell Lab., Elkton Road, Newark, DE, 19711); Sherman, H.; Barnes, J. R.; Aftosmis, J. G. *J Environ Pathol Toxicol* 1(3): 199-209; 1978.

The carcinogenicity of 4,4′-methylene bis(2-carbomethoxyaniline), also known as methylene-bis methyl anthranilate (MBMA), was investigated in ChR-CD weanling rats (Charles River cesarean-derived, Sprague-Dawley origin, barrier-sustained, random-bred albino). Groups of 50 rats of each sex were fed diets containing 0, 1,500, or 15,000 ppm MBMA for up to 2 yr. At both dietary levels, MBMA caused slight degenerative histologic changes in the liver and kidney of both sexes. Male rats receiving 15,000 ppm MBMA showed a statistically significant increase ($p < 0.05$) in kidney and liver tumors compared with controls, and females on the same dose had an elevated kidney tumor incidence compared with controls ($p < 0.10$). There was a statistically significant lower incidence of pituitary tumors in males ($p < 0.05$) and in females ($p < 0.10$) receiving 15,000 ppm MBMA compared with controls; the females also showed a lower incidence of mammary tumors ($p < 0.05$) compared with controls. The lower incidence of pituitary and mammary tumors in MBMA-fed rats than in controls suggests a possible endocrine effect produced by MBMA. Since MBMA-related tumors were not found until 18 mo after the initiation of a relatively large MBMA dose (1.5% of the diet), the compound is considered to be a carcinogen of moderate potency. (7 refs)

78-4322 **Methemoglobin Formation and Binding to Blood Constituents as Indicators for the Formation, Availability and Reactivity of Activated Metabolites Derived from trans-4-Aminostilbene and Related Aromatic Amines.** (Eng) Wieland, E. (Institut fur Pharmakologie und Toxikologie, Universitat Wurzburg, Versbacher Landstrasse 9, D-8700 Wurzburg, W. Germany); Neumann, H. G. *Arch Toxicol (Berl)* 40(1): 17-35; 1978.

Female Wistar rats were used to study the interactions of aminostilbene derivatives with blood constituents. The compounds were administered by stomach tube or iv injection, and methemoglobin (MHb) formation was measured for up to 4 hr. Interactions with blood constituents were also studie in vitro. After po administration, neither trans-4-dimethylaminostilbene nor the less toxic 4-dimethylaminodiben zyl raised the MHb level appreciably. After iv injection of th amino or the nitroso derivatives, however, considerable MH induction was observed. The less toxic dibenzyl compound produced more MHb than the trans-stilbene analogs. The results were confirmed by in vitro experiments with suspensions of rat RBC. The respective amines were inactive, an p-tolylhydroxylamine was as active as the nitroso compoun in this system. After the addition of labeled compounds t whole blood, 60%-70% of trans-4-nitrosostilbene and 4-nitrosodibenzyl was found in the RBC, and most of the radioactivity was bound to Hb. trans-4-Aminostilbene also exhibite affinity for the RBC (65%), but the radioactivity was no bound to Hb; aminodibenzyl did not enter the RBC to a appreciable extent. In vivo, trans-4-aminostilbene an 4-aminodibenzyl were N-oxidized to a similar extent, and th primary activation products of the former appeared even faster in the blood. However, intermediates originating durin MHb formation were more reactive and the trans-stilben derivatives bound two to three times more to Hb than th dibenzyl derivatives. The results indicate that binding to H and not the level of MHb is an indicator of the availabilit and reactivity of some activated aromatic amine metabolites (47 refs)

78-4323 **Identification and Quantitation of 3-Hydroxy-N-nitrosopyrrolidine in Fried Bacon** (Eng) Lee, J. S. (Dept. Food Science and Technology, Orego State Univ., Corvallis, OR, 97331); Libbey, L. M.; Scanla R. A.; Bills, D. D. *Bull Environ Contam Toxicol* 19(5 511-517; 1978.

3-Hydroxy-N-nitrosopyrrolidine (HNPYR) was identifie and quantitated in commercial fried bacon by gas chromatography and mass spectrum analysis of its trimethylsilyl derivative. HNPYR levels, relative to bacon wt, ranged from 0. to 3.9 ppb. (10 refs.)

78-4324 **Stereochemical Assignment of (E)- an (Z)-2-(1-Naphthyl)-1-phenylpropene and Thei Photocyclization to 5-Methylchrysene.** (Eng) Browne, C. E (Dept. Chemistry, Oklahoma State Univ., Stillwater, OK 74074); Dobbs, T. K.; Hecht, S. S.; Eisenbraun, E. J. *J Org Chem* 43(9): 1656-1660; 1978.

A shorter and improved synthesis (20% yield) of the carcinogen 5-methylchrysene (5MC) via UV irradiation of (E)- o (Z)-2-(1-naphthyl)-1-phenylpropene (ENP or ZNP), wa developed. Dehydration of 2-(1-naphthyl)-1-phenyl-2-propanol gave varying ratios of ENP, ZNP, and 2-(1-naphthyl)-3-phenylpropene, depending on the choice of reagent and photocyclization of ENP and ZNP gave 5MC. (23 refs)

8-4325 **Mutagenicity of Anthraquinones in the Salmonella/Microsome System: Activation of Anhraquinone Glucosides by Gut Bacterial Enzymes (Meeting bstract).** (Eng) Brown, J. P. (Dynapol, Palo Alto, CA, 4304); Roehm, G. W.; Brown, R. J. *Mutat Res* 53(1): 69-70; 978. (no refs)

8-4326 **Relationships Between Carcinogenicity and Theoretical Reactivity Indices in Polycyclic romatic Hydrocarbons.** (Eng) Smith, I. A. (Dept. Chemisy, Univ. California at San Diego, La Jolla, CA, 92093); erger, G. D.; Seybold, P. G.; Serve, M. P. *Cancer Res* 38(9): 968-2977; 1978.

heoretical reactivity indices were used to examine the metaolic reactions presumed responsible for the transformation f polycyclic aromatic hydrocarbons (PAH'S) to ultimate arcinogens. Twenty-five representative PAH's and their preumptive metabolites were analyzed. Of a large number of ndices examined, several showed a strong correlation with he carcinogenic activity of the test compounds. These findngs support the belief that specific transformations involving ihydrodiol, "bay-region" epoxide, and carbonium ion interediates are responsible for the carcinogenic activity of these ompounds. This type of analysis may provide a rapid and imple means for prescreening compounds for potential carinogenicity. (40 refs.)

78-4327 **Changes of the Cell Cycle During Carcinogenesis of Hamster Cheek Pouch Epithelium by ,12-Dimethylbenz(a)anthracene.** (Eng) Nagamine, Y. Dept. Pathology, Yamaguchi Univ. Sch. Medicine, 1144 Kogushi, Ube 755, Japan). *Gann* 69(3): 317-322; 1978.

he kinetic characteristics of 7,12-dimethylbenz(a)anthraene (DMBA)-induced hyperplastic and papillomatous epihelium in the cheek pouches of female Syrian hamsters were etermined. One cheek pouch was painted with 0.1 ml of a .5% soln of DMBA three times a week for 5 wk; in some nimals, the treatment was continued for >8 wk. Small tuors developed in several hamsters by day 47 and in all animals by day 67. DMBA treatment resulted in a progressive cceleration in the cell cycle, with the mean cycle times in he hyperplastic and neoplastic stages being approx wo-thirds and one-third of the control value, respectively. he G_1 phase was the one that was reduced the most. Most umor doubling times ranged from 100 to 150 hr, but the edian was between 200 and 250 hr. A nominal growth fracion exceeded unity in both the hyperplastic and neoplastic pithelium, indicating that a considerable number of cells in hich DMBA stimulated DNA synthesis failed to enter itosis. The inability of repair synthesis to restore DMBA-induced damage completely could also account for he failure of stimulated cells to enter mitosis. (25 refs.)

78-4328 **Biotransformation and Bioactivation of 7,12-Dimethylbenz(a)anthracene in Human Fetal and Placental Tissues. Analyses of HPLC Profiles and Studies with *Salmonella typhimurium*.** (Eng) Juchau, M. R. (Dept. Pharmacology, Sch. Medicine SJ-30, Univ. Washington, Seattle, WA, 98195); Namkung, M. J.; Jones, A. H.; DiGiovanni, J. *Drug Metab Dispos* 6(3): 273-281; 1978.

The capacity of human fetal and placental enzymes to catalyze the mixed-function oxygenation of 7,12-dimethylbenz(a)anthracene (DMBA) was studied. The metabolites formed in vitro were separated by high-pressure liquid chromatography (HPLC) and tentatively identified based on comparative HPLC retention times of known standard compounds. Comparisons were made with the commonly utilized substrate benzo(a)pyrene (BP), and the capacity of human fetal and placental tissues to generate mutagenic metabolites from DMBA was assessed with *Salmonella typhimurium* auxotrophs. The profiles of metabolites formed in incubation flasks containing fetal livers, lungs, kidneys, or adrenals were in general qualitatively similar to those observed using preparations of adult rat or rhesus monkey livers. The kidney exhibited the highest activities in each of the fetuses investigated. Profiles of metabolites from the placentas of women who smoked were markedly different from those of other tissues investigated, with large concentrations of metabolites appearing in the diol region. With BP as substrate, the metabolites cochromatographed largely with phenols and quinones. Placental microsomes from nonsmokers produced DMBA and BP profiles with minimal or no metabolites. The total metabolites formed from DMBA and BP were generally similar. Human fetal and placental tissue enzymes also catalyzed the formation of hydrocarbon metabolites mutagenic to various tester strains of *S. typhimurium*. (42 refs.)

78-4329 **Statistical Evaluation of Pienta's In Vitro Carcinogenesis Assay.** (Eng) Umezawa, K. (Dept. Molecular Oncology, Inst. Medical Science, Univ. Tokyo, 4-6-1, Shirogaendai, Minato-ku, Tokyo 108, Japan); Hirakawa, T.; Tanaka, M.; Katoh, Y.; Takayama, S. *Toxicol Lett* 2(1): 23-27; 1978.

The in vitro carcinogenesis assay developed by Pienta was evaluated statistically on the basis of eight experiments in which hamster embryo cells were exposed to 3-methylcholanthrene (3-MC: 0.1, 0.5, or 1.0 μg/ml) dissolved in dimethyl sulfoxide (DMSO: 0.2%) or to DMSO alone. No transformation was detected in DMSO controls or tissue culture controls, but 3-MC induced an av of 7.38 transformed colonies. The biomodal distribution of 3-MC transformation was calculated to determine the risk of a false negative. The results indicate that a positive result in the assay is reliable, but that there is a high risk of a false-negative result with chemicals possessing low transformation efficiency. This risk can be reduced by repeating the experiments. (5 refs)

78-4330 **Enhancement of Carcinogenesis by Prostaglandins.** (Eng) Lupulescu, A. (Medical Res. Building, Wayne State Univ., Detroit, MI, 48201). *Nature* 272(5654): 634-636; 1978.

The effects of prostaglandin E$_2$ (PGE$_2$) and prostaglandin F-2α (PGF-2α) on skin tumors induced by 3-methylcholanthrene (MCA) were examined in male albino Swiss mice. Mice were treated (1) topically with 0.2 ml of a 0.4% soln of MCA 3x/wk for 2 mo, (2) with MCA and, concomitantly, 10 μg PGE$_2$ im 3x/wk for 2 mo, or (3) with MCA and, concomitantly, 10 μg PGF-α im 3x/wk for 2 mo. Two additional groups were treated with each PG alone. At the end of 2 mo and 2 hr before they were sacrificed, mice from each group received an im injection of ^{3}H-thymidine, ^{3}H-uridine, ^{3}H-leucine, or ^{3}H-proline. Multiple and large tumors occurred after 2 mo in 90% of both the MCA plus PGE$_2$- and MCA plus PGF$_2$-treated mice at the treatment site. No tumors were observed in mice treated with MCA, PGE$_2$, or PGF-2α alone. There was a marked increase (15 to 20-fold) of ^{3}H-thymidine, followed by ^{3}H-uridine, ^{3}H-proline, and ^{3}H-leucine, in homogenized material from tumors of MCA plus PGE$_2$- and MCA plus PGF-2α-treated mice compared with those from MCA- or PG-treated or control epidermis. In investigations using MCA alone, tumors occurred only after 4 mo and usually after 5-6 mo; the PG's therefore, decreased the latent period by at least 65%. The PGE$_2$ and PGF-2α stimulation of DNA, RNA, and protein synthesis in hyperplastic epidermal cells following MCA application could explain the enhancement of cutaneous carcinogenesis and shortening of the latent period. (10 refs.)

78-4331 **Activities of 3,4-Benzpyrene Hydroxylase Systems Reconstituted by Means of Self-Assembly from Control and 3-Methylcholanthrene-induced Liver Microsomes of "Susceptible" and "Unsusceptible" Mice.** (Eng) Lyakhovich, V. V. (Dept. Biochemistry, Acad. Medical Sciences USSR, Novosibirsk 630091, USSR); Tsyrlov, I. B.; Gromova, O. A.; Rivkind, N. B. *Biochem Biophys Res Commun* 81(4): 1329-1335; 1978.

The self-assembly of 3,4-benzpyrene (3,4-BP) hydroxylase from solubilized proteins and lipids of control and 3-methyl-cholanthrene (MCA)-induced liver microsomes from mouse strains differing in susceptibility to 3,4-BP hydroxylase induction by polycyclic hydrocarbons was compared. The self-assembly was much more efficient in MCA-induced microsomes from induction-susceptible C57B1/6 mice and (C57B1/6 x DBA/2)F$_1$ hybrids than in control microsomes of these mice. In contrast, the self-assembly efficiency of MCA-treated microsomes from DBA/2 mice was no higher than that of the control microsomes of this strain. These data suggest that the regulation of the aggregation process during the molecular morphogenesis of endoplasmic membranes in mice is inherited as a dominant characteristic. (16 refs)

78-4332 **Rejection of Dinitrochlorobenzene-conjugated Syngeneic Tumor Cells by Dinitrochlorobenzene-Sensitized Guinea Pigs.** (Eng) Roth, J. A. (Dept. Surgical Services, Veterans Hosp., Sepulveda, CA, 91343); Morton, D. L.; Holmes, E. C. *J Surg Res* 25(1): 1-7; 1978.

The effect of dinitrochlorobenzene (DNCB), which can be easily conjugated to cells as dinitrophenyl (DNP), on the growth of a methylcholanthrene-induced sarcoma (MCA-25) in inbred female strain-2 guinea pigs was studied. MCA-25 tumor was conjugated to DNCB without significant loss of viability. Half of the animals were presensitized with 0.1 ml of a 20-mg/ml soln of DNCB injected id. All animals received 10^7 conjugated or unconjugated tumor cells injected id into both flanks. There was no significnat difference in tumor growth between sensitized and unsensitized animals. DNCB-conjugated tumor cells grew well in 17/22 animals not sensitized to DNCB, compared with only 8/21 sensitized animals. In addition, the DNCB-conjugated tumor cells grew significantly more slowly in the latter. Unconjugated tumor cells grew well in 18/20 unsensitized animals, even though they received a simultaneous injection of DNCB-conjugated cells. However, the growth of unconjugated tumor cells was inhibited by the simultaneous injection of conjugated tumor cells in sensitized animals. The results indicate that animals sensitized to hapten will not only reject hapten-conjugated tumor cells, but will also reject simultaneously injected unconjugated tumor cells. (15 refs.)

78-4333 **Competitive Binding and Metabolism of Several Carcinogenic Polycyclic Aromatic Hydrocarbons by AKR Mouse Embryo Cells in Culture.** (Eng) Zytkovich, T. H. (Dept. Molecular Medicine, Mayo Clinic, Rochester, MN, 55901); Moses, H. L.; Spelsberg, T. C. *Cancer Res* 38(8): 2307-2313; 1978.

The binding of ^{3}H-benzo(a)pyrene (BP), ^{3}H-benzo(a)anthracene [DB(a,h)A], and ^{3}H-dibenz(a,c)anthracene [DB(a,c)A] to nuclear chromatin was examined in cultured AKR mouse embryo cells. The covalent binding of the tritiated carcinogens to the nuclear substrates was inhibited effectively by 7,8-benzoflavone and by four unlabeled potent carcinogens: BP, DB(a,h)A, DB(a,c)A, and 3-methylcholanthrene. The weak carcinogens pyrene and anthracene had much less of an effect on this binding. Because covalent binding is a metabolically dependent event, the effects of the unlabeled compounds on the metabolism of the three tritiated compounds were also evaluated. The potent carcinogens inhibited the metabolism of the labeled compounds, causing decreased nuclear binding of the latter. Anthracene and pyrene stimulated the metabolism and had a variable effect on nuclear binding. The ability of the unlabeled compounds to compete with the metabolism and nuclear binding of the labeled carcinogens was apparently related to their carcinogenic potency. The data suggest the existence of a common metabolic pathway for potent carcinogens that differs, at least in part, from that of weak carcinogens. (43 refs)

78-4334 **Fine Structural Alterations Induced in Erythrocytes by Phorbol Myristate Acetate.** (Eng)

Vhite, J. G. (Dept. Pediatrics, Univ. Minnesota Medical ich., Box 479 Mayo Memorial Building, Minneapolis, MN, 5455); Repine, J. E. *Am J Pathol* 91(3): 571-580; 1978.

Previous studies have demonstrated that 12-O-tetradecanoyl-phorbol-13-acetate (phorbol myristate acetate, PMA) is a po-ent membrane-active agent causing cell wall-derived vacuole ormation in neutrophils and granule labilization in platelets. The present investigation demonstrates that PMA (2 x 10^{-6}, x 10^{-5}, or 1 x 10^{-4} M) also has marked effects on normal uman RBC. RBC exposed to PMA were converted into tomatocytes and stomatospherocytes. The effects of PMA vere concentration-dependent, required removal of plasma, nd were max at 37 C. Although the response of RBC to PMA was not identical to that of other blood cells tested previously, the similarities suggest that the capacity of the gent to produce membrane invagination may be fundamen-al to its mode of action. (23 refs.)

8-4335 **Plasma Membrane Alterations in Cultured Myogenic Cells Treated with the Tumor Pro-noter Tetradecanoylphorbol-13-Acetate (Meeting Abstract).** Eng) Schimmel, S. D. (Dept. Biochemistry, Univ. South Florida Coll Medicine, Tampa, FL, 33612); Keldie, V. C. *Fed Proc* 37(6): 1817; 1978. (no refs)

8-4336 **A Cell Culture Bioassay to Analyze Metabolism of Phorbol Diester Tumor Promoters.** (Eng) O'-Brien, T. G. (Wistar Inst. Anatomy and Biology, Phila-delphia, PA, 19104); Diamond, L. *Cancer Res* 38(8): 2567-2572; 1978.

The tumor promoter 12-O-tetradecanoylphorbol-13-acetate TPA) induces ornithine decarboxylase (ODC) in confluent monolayers of transformed hamster cells and potentiates the nduction of ODC in these cells by fresh medium. This obser-vation is the basis of a method that assays the metabolism of phorbol diester tumor promoters by the loss of their ODC-inducing activity. TPA-containing medium exposed to growing hamster embryo fibroblasts (HEF) for various lengths of time showed a rapid and progressive loss of the ability to potentiate ODC induction in transformed hamster cells, suggesting that the TPA had been metabolized. No loss of ODC-inducing activity occurred when TPA was exposed to cultures of growing or confluent human fibroblasts for 7 days. Mixing experiments with TPA-containing 'conditioned' medium from cultures of HEF and human fibroblasts indicated that HEF treated with TPA did not elaborate an inhibitor of ODC induction. This bioassay procedure was compared with the direct analysis of ^{3}H-TPA metabolism by thin-layer chromatography; the striking differences between hamster and human fibroblasts in their ability to metabolize TPA were confirmed. Cells from several other rodent species also metabolized TPA, but none of the four human cell lines tested, including SV40-transformed lines, metabolized the compound. The bioassay procedure was also used to measure metabolism of other phorbol diesters. Phorbol-12,13-didecanoate exposed to HEF did not lose its ability to induce ODC but phorbol-12,13-dibenzoate did. These results indicate that its metabolism renders TPA biologically inactive, as measured by ODC-inducing activity. However, not all phorbol diesters are metabolized by a given cell type, nor does their metabolism always lead to loss of biological activity. It is concluded that the chromatographic procedure plus the bioassay can be used to analyze the relationship between the chemical structure and biological activity of phorbol diester tumor promoters. (23 refs)

78-4337 **Effects of Phorbol-12-myristate-13-acetate on the Phenotypic Program of Cultured Chondroblasts and Fibroblasts.** (Eng) Lowe, M. E. (Dept. Anatomy, Univ. Pennsylvania Medical Sch., Philadelphia, PA, 19104); Pacifici, M.; Holtzer, H. *Cancer Res* 38(8): 2350-2356; 1978.

Phorbol-12-myristate-13-acetate (PMA: 5 x 10^{-8} M) had a prompt, differential, and partially reversible effect on cultured chick chondroblasts. Within 36 hr, PMA transformed sessile, polygonal, epithelioid chondroblasts into motile, multilayered, fibroblastic cells. By 48 hr, the chondroblasts no longer synthesized their type IV sulfated proteoglycan or a 180,000-dalton glycosylated protein that constitutes approx 5% of the total protein in normal chondroblasts. The effect of PMA on chondroblast morphology and protein synthesis was reversible after 4 days in PMA-containing medium but not after 12 days. PMA altered the morphology of chick fibroblasts, but it did not block the synthesis of their characteristic type III sulfated proteoglycan. PMA was mitogenic for the chondroblasts but not for the fibroblasts, in spite of the phenotypic similarities of these two cell types. (37 refs)

78-4338 **The Microsomal Metabolism of Benzo(a)pyrene Phenols.** (Eng) Capdevila, J. (Dept. Biochemistry, Univ. Texas Health Science Center at Dallas, 5323 Harry Hines Blvd., Dallas, TX, 75235); Estabrook, R. W.; Prough, R. A. *Biochem Biophys Res Commun* 82(2): 518-525; 1978.

Difference spectrophotometry coupled with high-performance liquid chromatography (HPLC) was used to monitor the steady-state metabolism of 3- and 9-hydroxybenzo(a)pyrene (9-OHBP) by liver microsomes from 3-methylcholanthrene-pretreated rats. The microsomal metabolism of 9-OHBP was accompanied by a progressive loss of absorbance at approx 421 nanometers (nm), concomitant with increases in absorbance at 409 and 436 nm. The two major 9-phenol metabolites had absorbance bands at 427 and 435 nm. Appreciable absorbance was also generated above 450 nm, suggesting that a small amount of BP quinones may exist during the steady state of the reaction. Experiments using 3-OHBP yielded difference spectra similar to those obtained with the 9-phenol. HPLC analysis revealed two major polar metabolites of 9-OHBP whose retention times relative to the 3-phenol were 0.59 and 0.68 min. These compounds had chromatographic properties similar to those of the 7,8-dihydrodiol of BP, and the optical and fluorescence emission spectra were similar to those of 9-OHBP. The effect of base titration on the fluorescence emission spectra was similar to the effect seen with the 9-phenol. HPLC also revealed two metabolites of ^{14}C-3-OHBP: the major metabolite had a retention time (0.83 min) and absorbance spectrum identical to those of BP 3,6-quinone; the second metabolite had a retention time of 0.59 min, similar to that of 3-OHBP, and an absorbance spectrum identical to that of the first metabolite of 9-OHBP. The results indicate that the liver microsomal fraction contains an oxygenase capable of metabolizing BP phenols to diphenols. (15 refs.)

78-4339 **Regio- and Stereoselectivity of Various Forms of Purified Cytochrome P-450 in the Metabolism of Benzo(a)pyrene and (-)trans-7,8-Dihydroxy-7,8-dihydrobenzo(a)pyrene as Shown by Product Formation and Binding to DNA.** (Eng) Deutsch, J. (Chemistry Branch, NCI, NIH, Bethesda, MD, 20014); Leutz, J. C.; Yang, S. K.; Gelboin, H. V.; Chiang, Y. L.; Vatsis, K. P.; Coon, M. J. *Proc Natl Acad Sci USA* 75(7): 3123-3127; 1978.

Highly purified cytochromes P-450-LM2 and P-450-LM4 and partially purified P-450-LM1, P-450-LM3b, and P-450-LM7 from rabbit liver microsomes exhibited different catalytic activities in the metabolism of benzo(a)pyrene (BP) and (-)trans-7,8-dihydroxy-7,8-dihydrobenzo(a)pyrene [(-)trans-7,8-diol] in a reconstituted enzyme system. The two highly purified cytochromes also exhibited differences in the activation of BP and (-)trans-7,8-diol to intermediates that bind to DNA, as well as in the stereoselective conversion of (-)trans-7,8-diol to the highly mutagenic and carcinogenic diol-epoxides r-7,t-8-dihydroxy-t-9,10-oxy-7,8,9,10-tetrahydro-BP (diol-epoxide I) r-7,t-8-dihydroxy-t-9,10-oxy-7,8,9,10-tetrahydro-BP (diol-epoxide II). P-450-LM2 was more active than P-450-LM4 in the metabolism of BP and in its conversion to products that bind to DNA. In contrast, P-450-LM4 is more active than P-450-LM2 in the metabolism of (-)trans-7,8-diol and in its conversion to products that bind to DNA. The radio of activity (percent substrate metabolized) with BP relative to that with (-)trans-7,8-diol is 21 for P-450-LM2 and 0.3 for P-450-LM4; P-450-LM1, P-450-LM3b, and P-450-LM7 gave intermediate ratios. Marked stereoselectivity in the oxygenation of the *trans*-7,8-diol to the highly mutagenic and putatively carcinogenic diol-epoxides I and II was observed with P-450-LM4, whereas the other preparations showed less selectively. The ratio of diol-epoxide I to diol-epoxide II ranged from 0.3 for P-450-LM7 to 11 for P-450-LM4. The substrate specificity and regio- and stereoselectivity of the different forms of cytochrome P-450 may regulate the balance between the activation and detoxification pathways of BP and, therefore, determine the susceptibility of individual tissues, strains, anc species to the carcinogenic action of BP. (43 refs.)

78-4340 **Tumorigenicity Studies with Diol-Epoxides o Benzo(a)pyrene Which Indicate that (±)-trans 7β,8α-Dihydroxy- 9α,10α-epoxy- 7,8,9,10 tetrahydrobenzo(a)pyrene is an Ultimate Carcinogen in Newborn Mice.** (Eng) Kapitulnik, J. (Dept. Pharmacology Hadassah-Hebrew Univ. Medical Sch., Jerusalem, Israel) Wislocki, P. G.; Levin, W.; Yagi, H.; Jerina, D. M.; Conney A. H. *Cancer Res* 38(2): 354-358; 1978.

The tumorigenicity of low equimolar doses of benzo(a)pyren (BP), (±)- trans -7β,8αdihydroxy-9β,10β-epoxy-7,8,9,1 tetrahydrobenzo(a)pyrene (diol-epoxide 1), (±)-tran 7β,8α- dihydroxy -9α,10α- epoxy-7,8,9,10-tetrahydr benzo(a)pyrene (diol-epoxide 2), (±)-trans- 7,8 dihydroxy- 7,8-dihydrobenzo(a)pyrene (BP 7,8-dihydrodiol) and the tetraols derived from the spontaneous hydrolysis o diol-epoxide 2 were evaluated in newborn Swiss-Webste mice. The mice were injected ip with 4, 8 and 16 nmoles o one of the test compounds on the 1st, 8th, and 15th days o life, and were sacrificed at 28 wk. Diol-epoxide 1 was highl toxic in newborn mice, and most of the animals given thi compound died before weaning. Diol-epoxide 2 and BP 7,8-dihydrodiol were about 40- and 15-fold more active, respectively, than BP in causing pulmonary adenomas. Diol-epoxide 1 and the tetraols derived from diol-epoxide 2 di not induce pulmonary adenomas. These results indicate tha diol-epoxide 2 is an ultimate carcinogenic metabolite and tha BP 7,8-dihydrodiol is a proximate carcinogenic metabolite o BP in newborn mice. The results also provide support for th proposal that diol epoxides, in which the epoxide ring form part of a bay region of the hydrocarbon, are of key importance for the carcinogenicity of BP and benz(a)anthracene (47 refs.)

78-4341 **Relationship Between Somatic Mutation an Neoplastic Transformation.** (Eng) Barrett, J. C (Div. Biophysics, Sch. Hygiene and Public Health, Johns Hopkins Univ., Baltimore, MD, 21205); Ts'o, P. O. *Proc Nat Acad Sci USA* 75(7): 3297-3301; 1978.

The somatic mutation (SM) and neoplastic transformatio (NT) of diploid Syrian hamster embryo cells were examine concomitantly. Mutations induced by benzo(a)pyrene an N-methyl-N'-nitro-N-nitrosoguanidine were quantitated a the hypoxanthine phosphoribosyltransferase and Na+/K+ ATPase loci and compared to phenotypic transformation measured by changes in cellular morphology and colony formation in agar. Both cellular transformations had characteristics distinct from the SM's observed at the two loci. Morphological transformation was observed after a tim comparable to that of SM but at a frequency that was 25- to 540-fold higher. Transformants capable of colony formation n agar were detected at a frequency of 10^{-5}-10^{-6}, but not until 32-75 population doublings after carcinogen treatment. Although this frequency of transformation is comparable to that of SM, the detection time required is much longer than the optimal expression time of conventionally studied SM's. NT of hamster embryo cells has been described as a multistep, progressive process. Various phenotypic transformations of cells after carcinogen treatment may represent different stages in this progressive transformation. The results are discussed in this context, and the role of mutagenesis in the transition between various stages is considered. NT may be nitiated by a mutational change, but it cannot be described completely by a single gene mutational event involving a dominant, codominant, or X-linked recessive locus. NT induced by chemical carcinogens is more complex than a single gene mutational process. Thus, this comparative study does not give experimental support to predictions of the carcinogenic potential of chemicals based on a simple extrapolation of the results obtained from conventional SM assays. (28 refs.)

78-4342 **Metabolism of Benzo(a)pyrene and Its Major Metabolites to Ethyl Acetate-soluble and Water-soluble Metabolites by Cultured Rodent Trachea.** (Eng) Moore, B. P. (Natl. Inst. Res. Dairying, Shinfield, Reading RG2 9AT, England); Cohen, G. M. *Cancer Res* 38(9): 3066-3075; 1978.

The metabolism of benzo(a)pyrene (BP) and its primary metabolites to their major ethyl acetate- and water-soluble (eas and ws) metabolites by cultured rat and hamster trachea was investigated. The major eas metabolites of BP cochromatographed with and had fluorescence excitation and emission spectra similar to 9,10-dihydrodiol, 7,8-dihydrodiol, and benzo(a)pyren-3-yl hydrogen sulfate. Enzymic hydrolysis of the ws metabolites suggested that the two major ws glucuronide conjugates were those of the monohydroxy-BP's and 4,5-dihydrodiol. Studies of the metabolism of the individual metabolites indicated that 3-hydroxy-BP was metabolized to an eas product that cochromatographed with and had fluorescence excitation and emission spectra similar to benzo(a)pyrene-3-yl hydrogen sulfate and to a ws glucuronide conjugate. 4,5-Dihydrodiol was metabolized to an unidentified eas metabolite and to a ws glucuronic acid conjugate. Both 7,8-dihydrodiol and, to a smaller extent, 9,10-dihydrodiol were metabolized, presumably via their respective diol-epoxides, to an eas metabolite that cochromatographed with and had fluorescence excitation and emission spectra similar to 7,8,9,10-tetrahydro-7,8,9,10-tetrahydroxy-BP. However, significant differences were observed in the ws metabolites formed from these metabolites. A major pathway of 7,8-dihydrodiol metabolism was to its ws glucuronide conjugate, but little or no 9,10-dihydrodiol was conjugated directly with uridine diphosphate glucuronic acid (UDGA). Small amounts of 9,10-dihydrodiol were metabolized to the catechol 9,10-dihydroxy-BP, which was rapidly conjugated with UDGA. These findings indicate that the rodent trachea, an area susceptible to hydrocarbon carcinogenesis, can metabolize BP to a wide range of oxidative and conjugative products. Factors affecting the balance and activities of these enzyme systems may be important in determining the susceptibility of the respiratory tract to carcinogenesis. (59 refs.)

78-4343 **Toxicity and Carcinogeniciy of Benzo(a)pyrene on Lung Cell Cultures and Cultivated Tracheal Tissue of Fetal Syrian Gold Hamsters. The Development and First Experiences with an in Vitro Test System.** (Ger) Richter-Reichhelm, H. B. (Hannover, W. Germany); Emura, M. *Staub-Reinhalt Luft* 38(6): 247-250; 1978.

Mixed lung cell cultures from Syrian gold hamster embryos (15 days gestation) were treated with varying concentrations of benzo(a)pyrene (BP). A dose-dependent toxicity was observed in the colony-forming assay. The addition of insulin (0.2 IU/ml)to the culture medium increased the plating efficiency of the cells from 1% to 3.9%. The colonies were divided into fibroblast, epi-, and endotheliallike cells. The toxicity of BP for these cells was also dose-dependent. BP-induced transformation rates were determined in the fibroblastic cells and were shown to be dose-dependent. At 0.05 and 0.25 μg BP the transformation rate was 1%, but at other doses 0.5% was never attained. Increases in insulin content increased BP toxicity in transformed fibroblasts. In soft-agar tests performed to detect the transformation of the epi- and endotheliallike cells, 4/6 cell lines had colony-forming ability. Sc implantation of 5 x 10^6 of these cells into syngenetic neonates resulted in walnut-sized sarcomas in 4/5 animals over a 3-mo period. The histological evaluation of BP treated in vitro explants of tracheal tissue from the same fetuses is in progress. Observations on 66% of the explants treated with 0.75 μg BP indicate that approx 15% show squamous metaplasia and other epithelial alterations. (6 refs.)

78-4344 **In Vitro Cell Transformation of Hamster Renal Cells Under the Influence of Atmospheric Fine-dust Extracts and the Papovavirus SV40.** (Ger) Seemayer, N. (Dusseldorf, W. Germany). *Staub-Reinhalt Luft* 38(6): 254-258; 1978.

The carcinogenic effects of fine atmospheric dust extracts were examined in Syrian gold hamster renal cells that were vaccinated with simian virus 40 (SV40) (Line Rh 911). Carcinogenic polycyclic hydrocarbons including: benzo(a)pyrene, 9,10-dimethylbenzanthracene, and dibenz(a,h)anthracene, each dissolved in a 0.5-1 mg/ml soln of dimethyl sulfoxide, were tested on the same system. The fine atmospheric dust extracts caused a significant increase in the transformation rate of cultures of SV40-inoculated hamster renal cells. There was also a significant increase in the transformation rate after exposure to the carcinogenic polycyclic hydrocarbons. The test system using hamster renal cells enables a clear differentiation of normal cells from transformed cells due to typical morphology and newly acquired immunologic characteristics. The atmospheric dust extracts seem to contain substances that increase the SV40 induced transformation rate. The mechanism for this increased rate remains unclear, but the polycyclic hydrocarbons contained in the extracts are implicated. (20 refs.)

78-4345 **Carcinogenic Atmospheric Pollutants and the Nature of Low-Level Risks.** (Eng) Albert, R. E. (Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY, 10016); Burns, F. J. *In: Incidence of Cancer in Humans. Proceedings of the Cold Spring Harbor Conferences on Cell Proliferation.* Hiatt, H. H.; Watson, J. D.; Winsten, J. A., eds. (Cold Spring Harbor: Cold Spring Harbor Laboratory): Origins of Human Cancer. Vol. 4, 602 pp.; 289-292; 1977.

Air pollution carcinogenesis studies suggest that extrapolation of dose-tumor incidence curves to define a probability of developing cancer from a given low-level of carcinogen exposure are inaccurate in the absence of knowledge about concomitant exposure to promoting agents. Low-level carcinogen exposure may heighten the susceptibility to cancer induction by otherwise noncarcinogenic substances. (8 refs.)

78-4346 **Study on Some Aspects of Exhaust Emissions from Diesel Engines.** (Eng) Srinivasa Rao, P. S. (I.C. Engines Lab. Dept. Mechanical Engineering, Indian Inst. Technology, Madras-600 036, India); Gopalakrishnan, K. V.; Murthy, B. S. *Chem Age India* 28(12): 1143-1145; 1978.

Studies were made of the cause of excessive smoke emissions from diesel-powered vehicles, effectiveness of antismoke fuel additives, effect of a dual fuel system using ethylalcohol and diesel fuel on pollution from diesel engines, and the effect of operating conditions on exhaust levels of nitric oxide and benzo(a)pyrene. (2 refs)

78-4347 **Differences in Effects of Norharman with Various Classes of Chemical Mutagens and Amounts of S-9.** (Eng) Nagao, M. (Biochemistry Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104, Japan); Yahagi, T.; Sugimura, T. *Biochem Biophys Res Commun* 83(2): 373-378; 1978.

The effect of norharman on the mutagenicity of various chemicals depends on the class of chemical and on the amount of S-9 mix (metabolic activation system prepared from rat liver). For example, aniline was mutagenic only in the presence of norharman, and mutagenicity increased linearly with the amount of S-9 mix. Norharman also increased the mutagenicity of benzo(a)pyrene in the presence of a large amount of S-9 mix but suppressed its mutagenicity when small amount was used. (17 refs.)

78-4348 **The Relationship Between Carcinogenicity an Mutagenicity of Some Polynuclear Hydrocar bons.** (Eng) Andrews, A. W. (Chemical Carcinogenesis Pro gram, Frederick Cancer Res. Center, Frederick, MD, 21501) Thibault, L. H.; Lijinsky, W. *Mutat Res* 51(3): 311-318; 1978

Ten polynuclear hydrocarbons and 15 of their partially hydrogenated derivatives were tested for mutagenicity using th Ames *Salmonella typhimurium* assay, and the results wer compared with existing carcinogenicity data. Only 1/25 compounds was mutagenic when tested in the absence of the Aroclor 1254-stimulated liver microsome S-9 mix. The remainde were mutagenic in at least one strain of *Salmonella* whe activated by a crude microsomal preparation from rat liver Of the 24 compounds that have been tested for carcinogenicity, there was a correlation between mutagenicity in the Ame system and carcinogenicity for 14. Eight of the remainin compounds were false positives and two were false negative in the Ames assay. This does not preclude acceptance of th basic precept of the Ames test as a good one, and its effectiveness in predicting carcinogenicity might be increased in th future. (14 refs.)

78-4349 **Seasonal Variation of Aryl Hydrocarbon Hydroxylase Inducibility in Human Lymphocytes in Culture.** (Eng) Richter, A. (Dept. Pharmacology, Univ. Toronto, Toronto, Ontario, Canada, M5S 1A8); Kadar, D.; Liszka-Hagmajer, E.; Kalow, W. *Res Commun Chem Pathol Pharmacol* 19(3): 453-475; 1978.

The aryl hydrocarbon hydroxylase (AHH) inducibility in lymphocytes from the peripheral blood of 92 healthy subjects (19-39 yr old) was tested during an 8-mo period (November-June). The av AHH inducibility was higher from March to June than from November to February, indicating a seasonal variation. The absolute number of lymphocytes was generally increased during May and June, resulting in an increased number of cells started in culture. However, a qualitative change within the lymphocyte population appeared to have taken place earlier (in March and April), resulting in an exceptionally high recovery rate of cells after 96 hr in culture; this change was associated with a higher av AHH inducibility in these months than in other months. The increased number of lymphocytes in blood observed in May and June was associated with only a minor increase in their AHH inducibility, compared with AHH inducibility during late fall and winter. Regardless of the cause of the change in AHH inducibility during different periods of the year, ignoring the change may produce an error in assessing the distribution of AHH inducibility among the population. (46 refs)

78-4350 **Aryl Hydrocarbon Hydroxylase Inducibility and Carcinoma of the Urinary Bladder.** (Eng) Trell, E. (Section Preventive Medicine, Univ. Lund, Lund, Sweden); Oldbring, J.; Korsgaard, R.; Hellsten, S.; Mattiasson, I.; Telhammar, E. *IARC Med Sci [Cancer]* 6(4): 138; 1978.

Aryl hydrocarbon hydroxylase (AHH) inducibility was assessed in 46 patients with carcinoma of the urinary bladder. Six had high inducibility levels, 18 intermediate, and 22 low. In the male patients, but not in the female patients, there was an overrepresentation of tobacco consumers. The AHH inducibility distribution did not differ from that of normal populations. There were no differences according to sex and age of the patients, smoking habits, type of cancer (recidivating or nonrecidivating, transitional or flat epithelial), or hereditary or occupational history. The findings support previous conclusions that high AHH inducibility plays no significant role in the pathogenesis of urinary bladder carcinoma. (16 refs)

78-4351 **Embryopathies Due to Spermatozoal Impairment in Xenopus Laevis.** (Eng) Hemsworth, B. N. (Life Science Lab., Teesside Polytechnic, Middlesbrough, Cleveland, England); Wardhaugh, A. A. *Mutat Res* 51(1): 45-53; 1978.

The effects of γ-radiation and various chemicals, including a range of contraceptive steroids, on the spermatozoa and developing embryos of the toad *Xenopus laevis* were studied. Sperm motility and the number of ova cleaved were reduced by exposure of the sperm to dimethylmyleran, trimethylphosphate (TMP), α-chlorohydrin, various progestational steroids, and the estrogenic steroids ethinyl estradiol and mestranol. Methyl methanesulfonate (MMS) had no apparent effect on sperm motility of fertilizing capacity, but it was cytotoxic to sperm. γ-Rays had no adverse effects on sperm motility or fertilizing ability. Exposure to either of the isomeric forms of dimethylmyleran, MMS, or γ-radiation led to a significant production of deformed tadpoles, whereas TMP, α-chlorohydrin, the progestational steroids, and the estrogenic steroids had no embryopathic effects. These findings provide some reassurance regarding the possible mutagenic action of oral contraceptives. (19 refs.)

78-4352 **Hepatocellular Carcinoma in a Young Woman with Prolonged Exposure to Oral Contraceptives.** (Eng) Gattanell, P. N. (Dept. Neoplastic Diseases, Mount Sinai Sch. Medicine, 100th St. and Fifth Ave., New York, NY, 10029); Perloff, M.; Holland, J. F. *Med Pediatr Oncol* 4(2): 99-103; 1978.

A case of hepatocellular carcinoma in a 28-yr-old woman is presented in which the apparent etiology was the use of oral contraceptives for 5.5 yr. Her total calculated dose was 1,365 mg of norethindrone, the 17,α-ethinyl-substituted derivative of 19-nortestosterone, and 68 mg of mestranol, the 3-methyl ether of ethinylestradiol. There was no history of hepatitis or alcoholism. Sequential therapeutic trials with ia 5-fluorouracil, ia adriamycin, and iv 1,3-bis(2-chloroethyl)-1-nitrosourea were unsuccessful. The patient died in hepatic coma 8 mo after diagnosis. She never became jaundiced or developed ascites, and the bilirubin level never rose above 2 mg/dl. This is the eighth report of hepatocellular carcinoma in women of childbearing age treated with oral contraceptives. (28 refs.)

78-4353 **Hormonal Factors Related to the Incidence of Testicular Tumors in Male Pseudohermaphrodite Rats.** (Eng) Allison, J. E. (P.O. Box 26901, Univ. Oklahoma Health Sciences Center, Oklahoma City, OK, 73190); Weidenbach, P. W.; Becker, R. R.; Hollander, R. *Fertil Steril* 30(1): 91-95; 1978.

A study was undertaken to determine if testicular tumors in male pseudohermaphrodite (tfm) rats were hormone-dependent and, if so, which hormone(s) was involved. Pieces of testicular tumor from tfm rats were transplanted into the connective tissue between the skin and the body wall musculature of the groins of normal males and females and tfm males. Pieces were also transplanted to the anterior chambers of the eyes of intact and gonadectomized normal males and females, and intact and gonadectomized tfm males. The transplants grew in all hosts except normal males. Levels of circulating gonadotropins were measured in all host types. All hosts except normal males had high serum levels of luteinizing hormone (LH), indicating that testicular tumors in tfm rats are LH-dependent. Since the anatomical, genetic, endocrinologic conditions in tfm rats are similar to those in human patients with testicular feminization, this LH dependence may also be true in man. (13 refs.)

See also:
*(Rev.): 78-4203, 78-4205, 78-4206, 78-4209, 78-4210, 78-4218, 78-4220, 78-4221, 78-4222, 78-4223, 78-4227, 78-4228, 78-4229, 78-4230, 78-4232, 78-4233, 78-4234, 78-4236, 78-4237, 78-4238, 78-4239, 78-4240, 78-4241, 78-4242, 78-4243, 78-4244, 78-4245, 78-4246.
*(Phys.): 78-4360, 78-4366, 78-4376, 78-4381.
*(Viral): 78-4460, 78-4502, 78-4504, 78-4530, 78-4540.
*(Immun.): 78-4544, 78-4559, 78-4560, 78-4563, 78-4566, 78-4569, 78-4575, 78-4576, 78-4579, 78-4581, 78-4582, 78-4586, 78-4588, 78-4589, 78-4591, 78-4593, 78-4597, 78-4599, 78-4600, 78-4601, 78-4602, 78-4604, 78-4606, 78-4613, 78-4617, 78-4622.
*(Path.): 78-4643, 78-4659, 78-4661, 78-4663, 78-4665.
*(Epid.-Biom.): 78-4671, 78-4677, 78-4680, 78-4689, 78-4691, 78-4696, 78-4698, 78-4703, 78-4708, 78-4710, 78-4711, 78-4712, 78-4713, 78-4714, 78-4715, 78-4716, 78-4717, 78-4722, 78-4730, 78-4736, 78-4737, 78-4738, 78-4752.

78-4354 **Plutonium-239 Deposition in the Skeleton of the Mouse.** (Eng) Green, D. (MRC Radiobiology Unit, Harwell, Didcot, Oxfordshire, OX11 0RD, England); Howells, G. R.; Thorne, M. C. *Int J Radiat Biol* 34(1): 27-36; 1978.

The distribution, 24 hr later, of iv injected plutonium-239 in the skeleton of the female CBA mouse was investigated by neutron-induced autoradiography together with computer-based methods of data reduction. With these techniques, it was possible to measure the localization of ^{239}Pu on the endosteal and periosteal surfaces of the bone to an accuracy of approx ± 2.5 μm. At 24 hr after injection, distribution of ^{239}Pu about the endosteal and periosteal surfaces of the third lumbar vertebra, a central caudal verebra, the right ilium, and the right femur was similar except for the endosteal surfaces of the ilium. Except for the latter, $>$ 60% of the ^{239}Pu lay within the bone, some penetrating as fas as 20 μm into the bone. In the ilium, the distribution was more strongly peaked about the edge of the bone image. Although the distribution on various bone surfaces appeared similar, the absolute magnitude of deposition varied from one bone to another, as shown by radiochemical analyses of ^{239}Pu in other comparable bones of the skeleton. It is assumed that ^{239}Pu deposition in bone is influenced more by extrinsic features such as blood supply than by the particular nature of the local bone surface. (14 refs.)

78-4355 **The Uptake and Redistribution of ^{241}Pu Within the Gonads.** (Eng) Priest, N. D. (Natl. Radiological Protection Board, Harwell, Didcot, Oxfordshire, England); Jackson, S. *Int J Radiat Biol* 34(1): 49-65; 1978.

The distribution pattern and binding sites of plutonium in the testes and ovaries were studied following the iv administration of ^{241}Pu citrate soln to male and female hamsters and a female rabbit. Within the first 2 hr after injection, marked concentrations of β tracks were associated with a small number of atretic graafian follicles and with the follicular fluid in the thecal rings of the ovaries. One day after injection, most of the plutonium in the atretic follicles was phagocytosed by macrophages, and after 10 days, β tracks were most conspicuous over macrophages in the stroma and ovarian medulla, with little plutonium remaining in the atretic follicles. β Tracks were seen throughout the testes 15 min and 2 hr after injection, possibly because of an artifact of tissue processing. After 1 day and, to a greater extent, after 10 days, there was a focalization of β tracks in the cells comprising the interstitial tissue. It appeared that the macrophages had accumulated the plutonium in this tissue. No migration of macrophages was seen in the testes. Histochemical staining revealed the presence of acid protoglycans, including chondroitin sulfate and glycoproteins at the sites of plutonium concentration i the ovary. These molecules were regarded as likely recepto sites for plutonium. No acidic carbohydrates were found i the testes, in which the initial binding site for plutonium ma be a compound lipid. This was deduced from the apparen inability of the interstitial tissue of the testes to bind plutoni um in situ. (17 refs.)

78-4356 **A Mathematical Model for Estimation o Plutonium in the Human Body from Urine Dat Influenced by DTPA Therapy.** (Eng) Hall, R. M. (E. I. du Pont Co., Savannah River Plant, Aiken, SC, 29801); Poda G. A.; Fleming, R. R.; Smith, J. A. *Health Phys* 34(5) 419-431; 1978.

An empirical model for the urinary excretion of plutoniu (Pu) was developed using bioassay data from accidentally exposed patients (two puncture wound cases and several inhalation cases) treated with diethylenetriaminepentaaceti acid (DTPA). The mathematical expressions derived included functions relating the fraction of systemic Pu available fo chelation to the time of the initial deposition and the interva between successive treatments. These empirical expression can be used to provide an early assessment of systemic burdens for DTPA-treated patients, for determining the optima DTPA dosage regimen, and for evaluating the effectivenes of DTPA therapy. DTPA therapy initiated immediately afte Pu contamination will reduce the body burden by a facto of about 30. A larger fraction of Pu loosely deposited in bod tissues can be removed by prolonged therapy. A recent P inhalation incident was used to demonstrate the usefulnes of this model for early assessment of a worker's systemi burden from urinary excretion data during extended DTPA therapy. Pu urinary excretion data for six workers were compared with the excretion rates predicted by the Pu-DTPA model and a reasonably good correlation was obtained. (2 refs.)

78-4357 **^{32}P and Acute Leukemia: Development of Leukemia in a Patient with Hemoglobin Yakima** (Eng) Bagby, G. C. (Leukemia Res. Lab., VA Hosp., Portland, OR, 97207); Richert-Boe, K.; Koler, R. D. *Blood* 52(2) 350-354; 1978.

Acute nonlymphocytic leukemia (ANLL) developed in 197 in a 54-yr-old man who had been treated with ^{32}P for polycythemia vera (PV), which was diagnosed in 1954. He had re ceived ^{32}P at intervals of 6-24 mo over a period of 10 yr (1955-1965) to a cumulative dose of 72 mCi. In 1964, his daughters were found to have erythrocytosis. Investigation led to the discovery in all three patients of a new Hb variant, Hb Yakima, with high oxygen affinity. After ^{32}P therapy was discontinued, the man remained stable until 1975, when he developed pancytopenia and a progressive increase in HbF. In 1976, peripheral blood and bone marrow samples supported the diagnosis of the preleukemic syndrome. One year later, the patient developed ANLL accompanied by fetal erythropoiesis. Because this type of hemoglobinopathy was discovered 27 yr after the introduction of ^{32}P for the treatment of PV and because there are now > 39 high-oxygen-affinity Hb's, it is anticipated that additional patients similar to the one reported have been exposed to ^{32}P. These patients may be the first group to permit assessment of the risk of ^{32}P-induced leukemia in a non-neoplastic disorder. (20 refs.)

78-4358 **Investigation of Tritium Incorporation by Means of Excreted Metabolites.** (Eng) Biro, T. (Inst. Isotopes, 1525 Budapest, pf. 77, Hungary); Szilagyi, M. *Curr Top Radiat Res* 12(1): 313-321; 1977.

Daily urine samples from workers repeatedly exposed to ^{3}H during the chemical processing of various labeled compounds were analyzed by liquid scintillation counting. ^{3}H concentrations were determined in native or treated (charcoal and/or acid) urine and in the urea, creatinine, protein, and centrifuge residue fractions. The time dependence of ^{3}H concentrations in the urea and in the centrifuge residue differed significantly from that of the ^{3}H in native or treated urine. In the urea fraction, the ^{3}H half-life was about 20 hr. (16 refs)

78-4359 **Unusual Dose-Response of Chromosome Aberrations Induced in Human Lymphocytes by Very Low Dose Exposures to Tritium.** (Eng) Hori, T. A. (Div. Genetics, Natl. Inst. Radiological Sciences, 9-1, 4-Anagawa, Chiba 280, Japan); Nakai, S. *Mutat Res* 50(1): 101-110; 1978.

WBC cultures prepared from the peripheral blood of a healthy adult man were exposed for 48 hr to tritiated water or ^{3}H-thymidine (^{3}H-TdR), at doses ranging from 1×10^{-3} to 100 μCi/ml. Aberrations in the lymphocyte chromosomes were examined in the metaphase figures accumulated during a 5-hr treatment with colcemid. A low level of DNA synthesis was found as early as 24 hr after exposure to tritiated water or ^{3}H-TdR. However, the DNA labeling index increased continuously, and it reached a level of 20% at 48 hr. The aberrations induced by the radiation from both sources were mostly chromatid types, such as chromatid gaps and deletions. The dose-response relationships produced unusual curves in both cases. At the higher dose range, the yields of chromatid breaks increased linearly with dose, but those at the lower dose range were significantly higher than would be expected by downward extrapolation from the linear relation. Partial-hit or partial-target kinetic events occurred at the very low dose levels (lower than 5 and 5×10^{-2} μCi/ml for tritiated water and ^{3}H-TdR, respectively). (24 refs)

78-4360 **Problems of Extrapolation from High Dose to Low Dose in *Tradescantia* Mutation Studies.** (Eng) Nauman, C. H. (Biology Dept., Brookhaven Natl. Lab., Upton, NY, 11971); Sparrow, A. H. *Environ Health Perspect* 22: 161-162; 1978.

Problems in extrapolating the effects of low-dose carcinogen exposure from those of high-dose exposure were studied by treating inflorescences of several clones of *Tradescantia* with ionizing radiation and with the gaseous form of several known or suspected chemical mutagens. The dose rate at which a radiation dose-response curve was determined was found to have a significant influence on any downward extrapolation: the lower the dose rate, the closer the curve would approach a +1 slope and the more accurately would an extrapolation to lower doses predict the mutagenicity in that low-dose region. Slopes of the exposure-response curves obtained after exposure of three clones to gaseous 1,2-dibromoethane (DBL) and ethyl methanesulfonate (EMS) varied from +0.6 to +2.4. In one clone, 250 ppm EMS was four times as mutagenic as 160 rads of acute x-rays. For DBE, slopes generally increased from +1 to +1.5 as exposure time increased from 2 hr to 6 days. Since there was some indication that the slope may decrease at low exposure, extrapolation of high exposure data to low-exposure situations might give an underestimation of the danger of a low exposure to DBE. (no refs)

78-4361 **Low-Dose Radiation (Letter to Editor).** (Eng) Kneale, G. (Regional Cancer Registry, Queen Elizabeth Hosp., Birmingham 15, England); Stewart, A. M. *Lancet* 2(8083): 262-263; 1978.

In 1944-1972 and 1944-1977 studies of cancer deaths among Hanford nuclear plant workers, the low risk estimates found, in the form of doubling doses (in rads) are defended. Questions are raised as to whether some widely held assumptions about the role of radiation in carcinogenesis can be applied to the Hanford data. It was found that all ionizing radiations are not sufficiently similar for similar values of doubling dose to apply, that all members of a population are not equally sensitive to radiation, that sensitivity to radiation varies with age, and that the dose-response relationship in this case would not be linear. Comparison of observed and expected cancer deaths can show if there is a cancer hazard for nuclear industry workers, but the dose levels should be higher than those in the Hanford study (mean cumulative dose was 1.38

rads for cancers, 0.99 for noncancers). However, it is significant that the mean cumulative dose was higher for 22 bone marrow cancers (4.49 rads) than for 42 other reticuloendothelial neoplasms (0.99 rads), even though these two groups accounted for only 3.3% and 6.3% of the fatal cancers (expected proportions, 2.0% and 8.6%). (5 refs.)

78-4362 **DNA-Repair after UV-Irradiation in Skin Fibroblasts from Patients with Actinic Keratosis.** (Eng) Sbano, E. (Dept. Dermatology, Univ. Siena, Siena, Italy); Andreassi, L.; Fimiani, M.; Valentino, A.; Baiocchi, R. *Arch Dermatol Res* 262(1): 55-61; 1978.

An autoradiographic counting technique was used to measure UV-induced unscheduled DNA synthesis in skin fibroblasts from 12 patients with chronic actinic keratosis (AK) and those from 12 aged-matched healthy controls. Fibroblast cell strains were obtained from the forehead skin and the abdominal or axillary skin of each subject. Skin fibroblasts from the AK patients showed a lower level of excision repair of UV-induced DNA damage than fibroblasts from normal subjects. In both normal subjects and AK patients, fibroblasts from skin ordinarily exposed to sunlight showed a higher DNA repair capacity than did fibroblasts from normally unexposed skin. DNA repair decreased as a function of aging in both groups of subjects. The data show that skin cancer DNA in AK patients may be promoted by a defect in DNA repair. The increased repair capacity of skin that is chronically exposed to sunlight is probably the consequence of a defensive process caused by enzymatic induction. (14 refs.)

78-4363 **Induction of Ouabain-resistant Mutations in C3H 10T1/2 Mouse Cells by Ultraviolet Light.** (Eng) Chan, G. L. (Lab. Radiobiology, Dept. Physiology, Harvard Univ. Sch. Public Health, Boston, MA, 02115); Little, J. B. *Proc Natl Acad Sci USA* 75(7): 3363-3366; 1978.

Ouabain-resistant mutants were induced in C3H mouse embryo 10T1/2 fibroblasts by exposing them to UV light, thus making available an in vitro system for studying mutagenesis and oncogenic transformation in parallel. ^{86}Rb uptake studies showed that biochemical mutants at the plasma membrane Na+,K+ transport ATPase locus were being selected for in this system. The optimal expression time for the mutants and the induced mutation frequency depended on the dose of UV light. The ratio of transformation to mutation frequencies was on the order of 10 for four different doses (25, 50, 75, and 100 ergs/mm^2, which suggests that the target size in the cellular genome for transformation may be approx 10 times the size of the Na+,K+ ATPase gene. It is proposed that both transformation and mutation induction can be quantitatively studied in this single system. (39 refs.)

78-4364 **Damage and Repair in Mammalian Cells after Exposure to Non-ionizing Radiations. I. Ultraviolet and Visible Light Irradiation of Cells of the Rat Kangaroo (*Potorous Tridactylus*) and Determination of Photorepairable Damage In Vitro.** (Eng) Harm, H. (Programs in Biology, Univ. Texas at Dallas, P. O. Box 688, Richardson, TX, 75080). *Mutat Res* 50(3): 353-366; 1978.

Cornea cells of the rat kangaroo (potoroo, *Potorous tridactylus*) were exposed to far-UV [254 or 302 nanometers (nm)] radiation with or without subsequent illumination by near-UV or visible light, and the cellular DNA was tested for the presence of photoproducts binding yeast photoreactivating enzyme (PRE). DNA from untreated control cells contained no binding sites for the yeast PRE. The DNA of far-UV-irradiated cells kept in the dark prior to DNA extraction showed considerable competitive inhibition (CI) of the in vitro photorepair system, which consisted of UV-irradiated transforming DNA of *Haemophilus influenzae* and an extract containing yeast PRE. When the irradiated cells were exposed to white light prior to DNA extraction, the CI by their DNA was much reduced. An initial irradiation of 254 or 302 nm produced similar results. In further experiments, potoroo cells were exposed for various time periods to 0.18% hydrogen peroxide. Increasing time of pretreatment with H_2O_2 was associated with increasing CI of the in vitro photorepair system by the DNA extracted from the cells; similar results were obtained when the extracted DNA only was treated with H_2O_2. CI by irradiated potoroo DNA was reduced by exposure to wavelengths $>$ 470 and $>$ 415 nm, but not to wavelengths $>$ 555 or $>$ 365 nm. Even the wavelengths with the best photorepair effectiveness caused some damage, interacting with the yeast PRE in vitro. For unknown reasons, one particular strain of potoroo cells lost its potential for photorepair. The data suggest that the expression of photorepair in mammalian cells depends on a number of experimental parameters, including the spectral composition of the photoreactivating light. Superposition of damage by the photoreactivating treatment appears to be the critical factor. (21 refs.)

78-4365 **Toxicity and Mutagenicity of Radiation from Fluorescent Lamps and a Sunlamp in L5178Y Mouse Lymphoma Cells.** (Eng) Jacobson, E. D. (Bureau Radiological Health, HFX 120, 5600 Fishers Lane, Rockville, MD, 20857); Krell, K.; Dempsey, M. J.; Lugo, M. H.; Ellingson, O.; Hench, C. W. *Mutat Res* 51(1): 61-75; 1978.

The toxic and mutagenic effects of the broad-spectrum light from several types of fluorescent lamps and from a mercury vapor sunlamp were studied in L5178Y mouse lymphoma cells. To facilitate comparison of the different rates of mutagenesis derived from exposure-reponse curves obtained for each light source, a parameter, joule-equivalent mutagenesis (jem), equal to mutants per 10^5 survivors per joule per square meter, was defined. Jem values were calculated using the integrated irradiance of each lamp. Unfiltered broad-spectrum radiation emitted by germicidal, mercury vapor, black light blue, black light, and cool white lamps was both toxic and mutagenic to L5178Y cells when the cells were irradiated in phosphate-buffered saline. Based on jem values, the relative mutagenicities of the lamps (compared with the germicidal UV lamp) were 3×10^{-3} for the sunlamp, 1×10^{-4} for the black light and cool white lamps, and 3×10^{-5} for the black light blue lamp. The mutagenic effect was linear throughout the range of exposures tested, and the toxic and mutagenic effects of the lamps were in general agreement with their relative spectral outputs from 290 to 330 nanometers. (19 refs.)

78-4366 **Induction of Cancer by Photodynamic Action and the Problem of Benefits and Potential Risk in Photochemotherapy in Humans.** (Eng) Santamaria, L. (Camillo Golgi Inst. General Pathology, Univ. Pavia, Pavia, Italy); Giordano, G. G. *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 146-150; 1978.

The hypothesis that a photodynamic reaction can be carcinogenic whenever the damage at the molecular level is still compatible with cellular life but is able to bring about neoplastic transformation was investigated by exposing 955 Swiss albino mice to benzo(a)pyrene (BP) and Wood's 125-watt light. In the first experiment, groups of mice were exposed to Wood's light (2x/wk for different lengths of time) and/or painted with BP. A tumor incidence of 4% was noted in BP-treated mice, but the highest incidence (58%) was noted in mice treated with BP and exposed to the light (8×10^{10} ergs/cm^2, not the highest dose used). In another experiment, two groups of mice were exposed to Wood's light and two groups were kept in rooms illuminated by daylight or kept in darkness. Mice receiving Wood's light developed carcinomas earlier than those kept in the dark, but continued irradiation inhibited carcinoma formation. Daylight accelerated carcinoma onset, which led to an increase in carcinoma incidence in these mice over that in mice kept in the dark. A third experiment was conducted along the lines of the first using a light dosage of 8×10^{10} ergs/cm^2. Light exposure resulted in a max 70% increase in benign and approx 100% increase in malignant neoplasms. At the molecular level, it is assumed that the induction of cancer by BP and light is due to a mutagenic effect. The observation of enhancement and inhibition of carcinogenesis, depending on the extent of light exposure (also noted in in vitro experiments), suggests that carcinogenesis and its enhancement by photodynamic action might be consistent with a cellular damage compatible with cellular life, whereas inhibition of carcinogenesis by photodynamic action should occur when the damaging effects kill normal as well as malignant or potentially malignant cells. Although the long-term effects of photodynamic treatment in humans are not known, the clinical benefits appear to depend on the death of the affected cells rather than their recovery after inactivation of the infectious agents. (45 refs.)

78-4367 **A Vitamin A Analogue Inhibits Radiation-induced Oncogenic Transformation.** (Eng) Harisiadis, L. (Radiological Res. Lab., Coll. Physicians and Surgeons of Columbia Univ., New York, NY, 10032); Miller, R. C.; Hall, E. J.; Borek, C. *Nature* 274(5670): 486-487; 1978.

The possibility that retinoids might inhibit radiation-induced oncogenic transformation was studied using cultured C3H mouse embryo fibroblasts. The transformation frequency following a single dose of ^{60}Co γ rays (400 rads) was determined in the presence and absence of the trimethylmethoxyphenyl analog of N-ethylretinamide (Ro-11-1430: 7.1 μM. The retinoid was not toxic at the concentrations used, and it significantly reduced the transformation rate from 9.04×10^{-4} transformants/surviving cell to 2.67×10^{-4}. Preliminary experiments indicated that much higher retinoid concentrations reduced the transformation rate near zero; however, some toxicity was observed at these concentrations. Ongoing experiments with other vitamin A analogs suggest that the ability to inhibit radiation-induced transformation is not confined to Ro-11-1430. Retinoids could have a role in the prevention of neoplasms induced by radiation as well as by chemical carcinogens. (18 refs.)

78-4368 **Occurrence of Carcinogenesis and Leukemogenesis Following Diagnostic and Therapeutic Exposure to Ionizing Radiation.** (Ger) Neumeister, K. (Staatliches Amt fur Atomsicherheit und Strahlenschutz der DDR, Waldowallee 115, DDR-1157 Berlin, E. Germany). *Dtsch Gesundheitsw* 32(38): 1793-1796; 1977.

Two men and two women who had been exposed to diagnostic, therapeutic, or occupational ionizing radiation developed squamous epithelial carcinomas at the irradiation site. The first patient had received repeated x-radiation for tinea barbae 42 yr before he developed keratinizing squamous epithelial carcinoma of the cheek. One irradiation session was longer than planned, but the total dose could not be established. Another patient received pelvic x-ray therapy (total dose 3,000 rads) after ovariectomy for benign cystadenoma of the ovaries. She developed a radiation ulcer and, finally, carcinoma 23 yr later. A third patient developed keratinizing squamous epithelial carcinoma 17 yr after removal of a broken needle from her hand with the aid of fluoroscopy. The dose rate was estimated to be 20 R/min. A surgeon who had been exposed to ionizing radiation occupationally prior to 1945 developed typical radiogenic lesions of his hands and ultimately, keratinizing squamous epithelial carcinoma of the index finger. The dose could not be estimated. A causative relationship between prior exposure to ionizing radiation and carcinoma is possible in all cases. (41 refs.)

78-4369 **Pituitary Fibrosarcoma Secondary to Radiation Therapy.** (Eng) Ahmad, K. (Dept. Radiology, Alice Crocker Lloyd Radiation Therapy Center, Univ. Hosp., 1405 E. Ann St., Ann Arbor, MI, 48109); Fayos, J. V. *Cancer* 42(1): 107-110; 1978.

A 56-yr-old man who received postoperative radiation therapy for a chromophobe adenoma developed a fibrosarcoma of the pituitary gland region 10 yr later. A total dose of 4,092 rads of cobalt-60 radiation was applied to the pituitary in a single course of 21 fractions over 29 days. (7 refs)

78-4370 **Hyperparathyroidism and Radiation Therapy.** (Eng) Christensson, T. (St. Eriksplan 2A, S-11320 Stockholm, Sweden). *Ann Intern Med* 89(2): 216-217; 1978.

The association of hyperparathyroidism (HPT) and previous radiation therapy to the neck was investigated among a group of 15,903 persons who entered a medical screening program in Stockholm, Sweden. Hypercalcemic HPT with parathyroid adenomas was discovered in 58 people (44 women, 14 men). The adenomas were single in all but a few patients. In no case was parathyroid hyperplasia found. All patients underwent exploration because of constant hypercalcemia upon repeated testing. Postoperative serum calcium values were normal. The 44 patients plus age- and sex-matched controls randomly chosen from the screening program group were investigated with respect to prior radiation therapy. Eight women in the hyperparathyroid group had received neck irradiation vs none of the controls. The eight patients had been irradiated at a mean age of 8.1 yr, and their mean age at diagnosis of HPT was 55.2 yr. All had been irradiated with x-rays (200-600 rads) for tuberculous adenitis. This study confirms that a relation exists between radiation therapy to the neck and the development of HPT. (14 refs.)

78-4371 **Synovial Sarcoma of the Neck Associated with Previous Head and Neck Radiation Therapy.** (Eng) Mischler, N. E. (Quisling Clinic, 2 W. Gorham St., Madison, WI, 53703); Chuprevich, T.; Tormey, D. C.; Ramirez, G.; Mack, E. A. *Arch Otolaryngol* 104(8): 482-483; 1978.

The case report of a 28-yr-old man who developed a synovial sarcoma in his neck 14 yr after receiving four facial and neck irradiation treatments for acne is presented. The total radiation dose that had been delivered was not known. Special stains and electron microscopy disclosed a characteristic synovial sarcoma. Ten months after surgery the lesion recurred, and the patient was treated with radiotherapy and chemotherapy. The histologic appearance of the tumor suggests a synovioblastic origin: it most likely arose from undifferentiated mesenchyme that retained the potential for synovioblastic differentiation. (10 refs.)

78-4372 **Thorium-induced Neoplasia of the Spleen.** (Ger) Schumacher, K. A. (Department fur Radiologie I, Universitat Ulm, Steinhovelstrasse 9, D-7900 Ulm/Donau, W. Germany); Bargon, G. *Soobshch Akad Nauk Gruz SSR* 31(8): 486-488; 1978.

The case report of a 58-yr-old man who presented with multiple circular foci in the spleen 25 yr after application of a thorium-containing contrast medium for vascular visualization is presented. In addition to these foci, ThO_2 deposits were present in the liver and spleen. The foci met the angiographic criteria of an angioblastic sarcoma. The problem of whether the lesions are thorium-induced is discussed. (12 refs.)

78-4373 **Thorotrast as an Etiological Factor in the Development of Malignant Bone Marrow Diseases.** (Ger) Fabricius, H. A. (Medizinische Klinik der Universitat, Hugstetter Strasse 55, 7800 Freiburg, W. Germany); Heni, N.; Mobius, W.; Henrichs, H.; Arnold, H. *Inn Med* 4(2): 76-80; 1977.

The literature contains 41 cases of leukemia (34 acute, 7 chronic) in patients previously given Thorotrast (TRT: thorium dioxide in a sugar soln). The first case was published in 1954. The frequency of leukemia in TRT-treated patients is about 8/10,000, or about 10 times the usual incidence. A case involving a 64-yr-old man is reported to illustrate the difficulty in diagnosing leukemia in TRT-treated patients. The patient presented with refractory anemia, but he was in good general condition and he had no other clinical findings. Blood and bone marrow studies led to diagnoses ranging from toxic damage to myelopoiesis or preleukemia. Two weeks later his condition had deteriorated and acute myeloid leukemia was confirmed. An x-ray-positive substance thought to be TRT was found in the spleen and later in the liver and in biopsy material from the iliac crest. No clear indication for the use of TRT emerged from the patient's history. It is assumed that it was used to treat a war-wound in 1942. Because of the age of the patient, he was treated conservatively with RBC and thrombocytes. He died 4 mo later. Thorium dioxide was identified in tissues obtained at autopsy. X-rays of the liver and spleen are helpful in determining the presence of TRT. (17 refs.)

78-4374 **Does Cholecystectomy Predispose to the Development of Colorectal Cancer?** (Fre) Capron, J. P. (Clinique Medicale, Hopital Beaujon, F 92118 Clichy Cedex, France); Delamarre, J.; Canarelli, J. P.; Brousse, N.; Dupas, J. L. *Gastroenterol Clin Biol* 2(4): 383-389; 1978.

The frequency of the association of colon cancer and chole-ystectomy was determined in a retrospective study of 237 atients (Group Ia, 129 men; Group Ib, 108 women) operated n for colon cancer and 2,458 subjects (Group IIa, 1,480 men; roup IIb, 978 women) free of colon cancer at autopsy. The ge range of the two groups of men was 50-79 yr and that f the two groups of women, 40-79 yr. The frequency of holecystectomy was significantly higher ($p < 0.001$) for the omen in Group Ib (13%) than for the women in Group IIb 3.3%); no significant difference was noted between the two roups of men. In Group Ib, the frequency of cholecystecto-ny was significantly higher in patients with cancer of the ight hemicolon ($p < 0.05$) and of the rectum ($p < 0.02$) than n those with cancer of the left hemicolon. The results indi-ate that cholecystectomy, which causes changes in bile acid netabolism, could be one of the factors predisposing to the evelopment of cancer of the rectum or colon. (31 refs)

8-4375 **The Development of Mucosal Changes After Gastric Surgery for Ulcer Disease.** (Eng) Ja-unger, K. G. (Depts. Surgery and Pathology, Univ. Hosp., Jmea, Sweden); Domellof, L.; Eriksson, S. *Scand J Gastroen-erol* 13(2): 217-223; 1978.

The development of morphological changes in the body nucosa after partial gastrectomy for ulcer disease was stud-ed early (within 3 yr) after Billroth I gastrectomy in 41 pa-ients [18 with gastric ulcer (GU), 23 with duodenal ulcer DU)], early after Billroth II gastrectomy in 14 patients (4 GU, 10 DU), late (10-24 yr) after Billroth I resection in 79 atients (44 GU, 35 DU), and late after Billroth II resection n 172 patients (41 GU, 131 DU). The morphological findings n the body mucosa of the surgical and biopsy specimens were ompared. Evaluation of the six forceps biopsies taken at gas-roscopy in all patients revealed great individual variation, ot only in the degree of atrophy and chronic gastritis, but lso with regard to the presence of intestinal metaplasia. Within 3 yr after partial gastrectomy, the mucosal changes ad become advanced in both DU and GU patients, and they vere almost as severe as those found late postoperatively. Although late postoperatively there was a higher incidence f cystic dilatation of the gastric glands after Billroth II resec-ion than after Billroth I resection ($p < 0.001$), there were o other correlations between the type of anastomotic proce-ure and the mucosal changes. In addition, there were no orrelations between the mucosal changes and factors such s age, sex, or vagotomy. The results show that the increased ncidence of mucosal changes caused by partial gastrectomy s of a similar magnitude in both DU and GU patients. If hese mucosal changes lead to malignant transformation, the results may explain why stump carcinoma has previously been found with the same frequency in patients who had undergone surgery for DU and for GU. (40 refs)

78-4376 **Proflavin Photosensitization of Double and Single Stranded DNA: EPR Study of the Effect of Various Sulphur-containing Substances.** (Eng) Piette, J (Lab. General and Medical Microbiology, Univ. Liege, Sart-Tilman, 4000 Liege, Belgium); Calberg-Bacq, C. M.; Van de Vorst, A. *Photochem Photobiol* 27(4): 457-464; 1978.

The proflavin (PF)-mediated photosensitization of double-stranded calf thymus DNA and single-stranded DNA from bacteriophage ϕ x 174 was followed by electron paramagnetic resonance measurements of free-radical induction in frozen soln in the presence of oxygen. The effect of the addition of various sulfur-containing substances to the PF-DNA mixtures was studied and quantitatively expressed for definite amounts of PF bound to both DNA's. Upon irradiation with visible light, radiosensitive radicals were observed in the presence of these sulfur-containing substances. Some of them caused a decrease in the amount of peroxide radicals normally induced in the photosensitized DNA. This decrease appeared to be linked to the ability of the compounds to interact with DNA in such a way that they modified the binding of PF to DNA. Cysteine, cysteamine, cystamine, and cystine protected calf thymus DNA against the PF and light free-radical induction by 55%, 58%, 64% and 62%, respectively. Bacteriophage single-stranded DNA is also protected by the addition of cysteamine and cystamine, although to a lesser extent than calf thymus DNA. These protective effects were observed under conditions in which strong binding of the dye to the DNA was predominant. (38 refs)

78-4377 **The Crucial Role of DNA Double-strand Breaks in Cellular Radiobiological Effects.** (Eng) Leenhouts, H. P. (Assoc. Euratom-ital, Wageningen, Netherlands); Chadwick, K. H. *Adv Radiat Biol* 7: 55-101; 1978.

A molecular theory was developed to explain the biological effects of ionizing radiation on living eukaryotic cells. An attempt was made to show that the dose relationships of the different radiation-induced effects in these cells can all be analyzed and related to the number of DNA double-strand (ds) breaks induced in the cell. It is proposed that each radiation-induced ds break has a probability of leading to cell death, chromosomal aberration, somatic mutation, malignancy, or hereditary effects. Linear-quadratic dose kinetics for the induction of ds breaks were combined with the random distribution of the number of ds breaks in a cell population to produce a series of relatively simple equations that describe the dose-response relationships for the various effects. The dose relationships are defined essentially by two coefficients, but these coefficients are made up of different parameters to take into account the complicated processes involved in the radiation interaction. The theoretical approach to mutations was extended to radiation-induced malignancy by using the results of previous investigators showing that the induction of malignancy is associated with a somatic mutation. They concluded that the factor controlling the malignant nature of a cell is contained in a chromosome and that this factor can be suppressed by the presence of a normal homologous chromosome; ie, it is recessive. In a cell carrying the recessive malignant genotype, a mutation or aberration in the normal homologous chromosome could remove the suppressive action of this chromosome and allow the expression of the malignant factor. (148 refs)

78-4378 **Xeroderma Pigmentosum Fibroblasts of the D Group Lack an Apurinic DNA Endonuclease Species with a Low Apparent Km.** (Eng) Kuhnlein, U. (Dept. Biophysics, B. C. Cancer Foundation, Vancouver, B. C., Canada V5Z 3K3); Lee, B.; Penhoet, E. E.; Linn, S. *Nucleic Acids Res* 5(3): 951-960; 1978.

Apurinic DNA endonuclease activity from cultured human fibroblasts was resolved into two species by phosphocellulose chromatography. The species had sedimentation coefficients of 3.38 and 2.88 and apparent Km's for apurinic sites of 5 and 44 nM, respectively. Cell lines from normal individuals, from a patient with ataxia telangiectasia, and from patients with the A and D complementation groups of xeroderma pigmentosum were compared. The low Km species was absent from extracts of the three cell lines of xeroderma pigmentosum complementation group D. The significance of this finding is not known. (21 refs)

78-4379 **Environmental Carcinogenesis and Imperfect Repair of Damaged DNA in *Homo Sapiens*: Causal Relation Shown by In Vitro Studies of Rare Inherited Disorders.** (Eng) Paterson, M. C. (Biology and Health Physics Div., Atomic Energy Canada Ltd., Chalk River, Ontario, Canada). *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 16-17 pp.; 1978. (no refs)

78-4380 **Selective Particle Deposition and Bronchogenic Carcinoma.** (Eng) Schlesinger, R. B. (Inst. Environmental Medicine, New York Univ. Medical Center, 550 First Ave., New York, NY, 10016); Lippmann, M. *Environ Res* 15(3): 424-431; 1978.

Regional and intrabronchial particle deposition patterns determined in experimental inhalation tests using a hollow cast of the human tracheobronchial tree, γ-tagged aerosols, and a collimated radiation detector were compared to the distribution of sites of origin of primary bronchogenic tumors in the upper bronchial tree obtained from published reports. The test aerosols consisted of tagged (^{99}Tc) ferric oxide microspheres under three conditions of constant inspiratory-type flow (15, 30, and 60 liters/min). These flow rates correspond to the av flow rates reported for rest, light work and heavy work, respectively. Particles covering a similar size range as those used in this test make up a large part of the mass in urban atmospheric aerosols and their surfaces could act as vectors for transport of carcinogens into the respiratory tract. It was estimated, from published reports, that 10% of all bronchogenic carcinomas arise in the main bronchi, 30% in the lobar bronchi, 30% in the segmental bronchi, and 30% in bronchi distal to the segmental. Particle deposition was calculated to be lowest in the main bronchi and greatest in the lobar and segmental bronchi. This pattern was similar for the range of particle sizes and exposure flow rates tested. The deposition data suggest that bifurcations in the lobar and segmental bronchi receive higher doses than those at other branching levels, which may partly explain the higher cancer incidences at these sites. The deposition characteristics of the airways may play a significant role in cancer pathogenesis. (31 refs)

78-4381 **Use of Parabiont Mice to Study the Histogenesis of Sarcomas Induced by Plastic Plates.** (Rus) Moizhess, T. G. (Lab. Mechanisms Carcinogenesis, Cancer Res. Center, Moscow, USSR). *Tsitologiia* 20(2): 215-217; 1978.

To determine whether sarcomas induced by plastic plates originate from local connective tissue cells or from cells migrating with the blood, a 22- x 15-mm polyvinyl chloride plate was implanted sc in parabiotic pairs of 3-mo-old male CBA mice (each pair had a common abdominal cavity). One of the mice in each pair had a marker T_6 chromosome. Cytogenetic analysis of the developed tumors showed that the karyotype of all tumors corresponded to that of the host mouse. (8 refs)

78-4382 **Mortality from Leukaemia and Cancer in Shipyard Nuclear Workers.** (Eng) Najarian, T. (193 Lewis Road, Belmont, MA, 02178); Colton, T. *Lancet* 1(8072): 1018-1020; 1978.

A review of death certificates in New Hampshire, Maine, and lassachusetts for 1959-1977 yielded a total of 1,722 deaths mong former workers at the Portsmouth Naval Shipyard, here nuclear submarines are repaired and refueled. ontact with the next of kin was possible for 592 ien. All deaths under age 80 were classified as being n former nuclear or non-nuclear workers depending n information supplied by the next of kin. With US ge-specific proportional cancer mortality for white ien as a standard, the observed vs expected ratio f leukemia deaths was 5.62 (6 observed, 1.1 expect- d) among the 146 former nuclear workers. For all cancer eaths, this ratio was 1.78. Among non-nuclear workers, iere was no statistically significant increase in proportional iortality from either leukemia or from all cancers. The ex- ess proportional leukemia and cancer mortality among nu- lear workers exceeds predictions based on previous data of idiation effects in man. (4 refs)

8-4383 **Dielectric Characterization of UICC Asbestos Samples.** (Eng) Fleming, D. W. (Dept. Physics, aurentian Univ., Sudbury, Ontario, Canada); Rubin, G. A. *owder Technol* 20(2): 261-271; 1978.

n attempt was made to determine the mobility of charges n Union Internationale Contre le Cancer (UICC) samples f Canadian chrysotile and on South African amosite asbes- s. The former showed relatively large energy dissipation iaxima and they were slightly thixotropic at volume concen- ations as low as 0.5%. The latter were dielectrically inactive and they did not show thixotropic behavior up to fiber concentrations of 1.0 volume %. (15 refs.)

78-4384 **Elimination of Asbestosis (Letter to Editor).** (Eng) Matalani, E. (Occupational Health Unit, 509 Pitt St., Sydney, New South Wales 2000, Australia). *Med J Aust* 1(3): 157; 1978.

The use of Kaowool, a mineral ceramic fiber made from kaolin, as a substitute for asbestos is proposed. However, since a crystalline form of silica (Cristobalite) can be detected in the fused material when Kaowool is heated to high temperatures (1,250 C for 500 hr), its potential health hazards, ie, mesothelioma production, should be investigated. It is suggested that a threshold limit value of 10 mg/m^3 be used until testing is complete. (2 refs)

See also:
*(Rev.): 78-4206, 78-4209, 78-4224, 78-4225, 78-4227, 78-4228.
*(Chem.): 78-4272, 78-4282, 78-4351.
*(Viral): 78-4414, 78-4415, 78-4502.
*(Immun.): 78-4546, 78-4550, 78-4568, 78-4576, 78-4592.
*(Path.): 78-4648, 78-4658.
*(Epid.-Biom.): 78-4717, 78-4719, 78-4722, 78-4724, 78-4725, 78-4754.

78-4385 **On Epizootiology of Lymphoid Leukosis in Chickens.** (Eng) De Boer, G. F. (Dept. Virology, Central Veterinary Inst., Houtribweg 39, Lelystad, Netherlands); Maas, H. J.; Van Vloten, J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 19-24; 1978.

The prevalence of lymphoid leukosis (LL) virus infections and clinical disease was studied among laying-type chickens of an egg production testing station in the Netherlands during 1972-1976. The horizontal transmission of LL was also studied in 1-day-old and 8-wk-old chicks with high titers of maternal antibody to LL viruses. Viremia was detected in 31.6% of the 3- to 6-wk-old cockerels from the testing station, and homogenates of the spleen and bursa of Fabricius were positive in 40.8% of the birds tested. Viremia was present in 11.3% of the female hatch mates tested at 20 mo of age. Using WBC preparations, 21.9% of the 19-mo-old birds tested were virus-positive. The mean mortality due to LL was 0.43% during 1973-1974 and 2.8% and 1.28%, respectively, during the following 2 yr. Overt clinical disease occurred in 152/9,790 chickens under observation. The LL virus isolated belonged to subgroup A in 97% of the cases and to subgroup B in 3%. Antibody to subgroup A was rare in the 3- to 6-wk-old viremic birds, but it was found in 32% of the 20-mo-old viremic birds. In the horizontal transmission study, the plasma from 7-mo-old birds that had been exposed to LL-infected hatch mates at 1 day or 8 wk of age was virus-negative. However, a number of WBC preparations from the same birds were virus-positive. The data suggest LL virus can be transmitted horizontally and that chickens develop a resistance to LL virus infection with increasing age. (14 refs.)

78-4386 **Transformation Parameters in Chicken Fibroblasts Transformed by AEV and MC29 Avian Leukemia Viruses.** (Eng) Royer-Pokora, B. (Biologisch-Medizinische Abteilung, Max-Planck-Institut fur Virusforschung, Spemannstrasse 35, 7400 Tubingen, W. Germany); Beug, H.; Claviez, M.; Winkhardt, H. J.; Friis, R. R.; Graf, T. *Cell* 13(4): 751-760; 1978.

The expression of transformation parameters was studied in chicken fibroblasts (derived from clonal strains of chicken embryo fibroblasts) transformed by two model-defective leukemia viruses, avian erythroblastosis virus (AEV) strain ES4 and avian myelocytomatosis virus strain MC29. Fibroblasts transformed by Rous sarcoma virus (RSV) were analyzed for comparison. Infection with AEV or MC29 led to a rapid morphological transformation of most cells. AEV- and RSV-transformed fibroblasts were similar in that they exhibited microvilli at their surface, showed a disappearance of actin cables, were agglutinable by lectins, and showed a decrease in large, external, transformation-sensitive (LETS) protein (a high mol-wt cell-surface glycoprotein) and an increase in the rate of hexose uptake. They also had elicited slightly increased levels of cell-associated proteolytic activity, but no increase in the fibrinolytic activity of the harvested fluids. They were capable of anchorage-independent growth and of sarcoma induction. MC29-transformed fibroblasts also showed a disappearance of actin cables, and they were agglutinable by lectins, formed surface alterations that consisted of bleblike protrusions, and were capable of anchorage-independent growth. However, MC29-transformed fibroblasts expressed normal levels of LETS protein, had no increase in the rate of hexose uptake or in proteolytic activity, and were not sarcomagenic, although they showed an accelerated growth rate in culture. It is possible that MC29 and, perhaps, AEV transform fibroblasts by a mechanism different from that of RSV. (38 refs)

78-4387 **Normal Chicken Cells (chf-) Express a Surface Antigen Which Cross-reacts with Determinants of the Major Envelope Glycoprotein (gp85) of Avian Myeloblastosis Virus.** (Eng) Collins, J. J. (Dept. Surgery, Duke Univ. Medical Center, Durham, NC, 27710); Montelaro, R. C.; Denny, T. P.; Ishizaki, R.; Langlois, A. J.; Bolognesi, D. P. *Virology* 86(1): 205-216; 1978.

Attempts were made to distinguish chick helper factor-positive (chf+) cells from chf- cells using rabbit antisera raised against the purified major envelope glycoprotein (gp85) of avian myeloblastosis virus (AMV) in several serological assays, including competition radioimmunoassay and membrane immunofluorescence. However, chf+ cells coul not be differentiated from chf- cells. Radioimmunoprecipitation analysis of material recognized in uninfecte chf(+) and chf(-) chicken cells by the rabbit anti-AM gp85 antisera revealed the presence, in immune precipitates of both cell types, of a high-mol-wt glycoprotei (approx 125,000 daltons); however, low amounts o gp85 were precipitated only from the chf(+) cells. I contrast, antisera raised against the gp85's of Pragu strain Rous sarcoma virus (RSV: subgroup C), Bratislava-77 strain RSV (subgroup C), or Rous-associated virus type O did not recognize the 125,000-dalton molecule in either cell type, although gp85 was again precipitated from the chf(+) cells. The high-mol-wt glycoprotein, designated normal chicken glycoprotein, appears to represent a

ormal chicken cell-surface glycoprotein that is antigenically
ross-reactive with unique determinants of AMV gp85 that
e presumably dependent on the carbohydrate portion of the
olecules. (32 refs)

3-4388 **Anomalous Behavior of the Major Avian
Myeloblastosis Virus Glycoprotein in the Pres-
ıce of Sodium Dodecyl Sulfate.** (Eng) Marciani, D. J. (Lab.
umor Virus Genetics, NCI, Bethesda, MD, 20014); Papa-
atheakis, J. D. *J Virol* 26(3): 825-827; 1978.

he major glycoprotein of avian myeloblastosis virus (AMV)
as characterized using polyacrylamide gel electrophoresis
AGE) and gel filtration in the presence of sodium dodecyl-
ılfate (SDS). The apparent mol wt of the glycoprotein de-
eased from 140,000 with 4% acrylamide to a limiting value
81,000 with 10% acrylamide. The use of retardation coeffi-
ents yielded a mol wt for the AMV glycoprotein of 64,000.
Vhen gel chromatography in SDS was used to determine the
pparent mol wt of the glycoprotein from its hydrodynamic
operties alone, the estimated value was 50,000. It is con-
uded that the generally assigned value of 80,000 daltons for
e AMV major glycoprotein, as determined by SDS electro-
ıoresis, may be an overestimate because of its relatively low
ee electrophoretic mobility and peculiar confirmation in
DS. The true mol wt of the AMV glycoprotein must conse-
uently be determined by alternative methods such as sedi-
entation analysis in the presence of detergents or amino
id analysis in combination with specific chemical cleavage
the polypeptide chain. (13 refs.)

8-4389 **Quantitative Analysis of MC29 Virus in Plasma
of Hepatoma-Bearing Turkey Poults by Deter-
ination of Reverse Transcriptase Activity and Radioim-
uno-Assay.** (Eng) Talas, M. (Microbiological Res. Group
Hungarian Acad. Sciences, Budapest and D. I. Ivanovsky
ıst. Virology, Moscow, USSR); Zakharova, L. G.; Stoger,
; Altstein, A. D.; Piker, E. G. *Acta Microbiol Acad Sci Hung*
(4): 323-329; 1977.

nalysis of reverse transcriptase (RT) activity was compared
ith radioimmunoassay (RIA) of specific viral proteins for
e quantitative determination of myelocytomatosis virus 29
4C29) in MC29 hepatoma-bearing turkey poults.
ne-day-old turkey poults were infected iv with the virus,
ıd plasma was collected from tumor bearers on day 12
ostinfection. Pellets of virus particles obtained by
entrifugation were resuspended in buffer soln. Max
ral RT activity was obtained after incubation of
e reaction mixture containing the virus preparation
r 180 min at 20 C or for 120 min at 37 C. Opti-
um conditions also included the presence of
025%-0.2% Triton X-100 and 6 mM $MgCl_2$ or 0.5 mM $MnCl_2$ in the mixture. The best exogenous template for the reaction was poly(rA)-oligo(dT). A direct relation existed between plasma dilution and RT activity. A mixture of labeled p19 and p27 of avian myeloblastosis virus (AMV) was used as a standard in the RIA. The number of virions in the AMV pool used was known; therefore, the number of MC29 virions in the plasma preparations could be calculated. The data indicated that RIA was more sensitive than analysis of RT activity for the quantitative determination of MC29 virus. With RIA, virus could be detected even in 10^{-4} dilutions of plasma, but only two- to fourfold dilutions could be used with the RT analyses. (14 refs)

78-4390 **Isolation of Lymphoid Leukemia Virus in Hens.** (Rus) Popova, L. S. (No affiliation given); Zelensky, V. P.; Zaretskaya, L. I. *Veterinariia* (7): 99-100; 1977.

The assay for routine isolation of chicken leukemia virus is described. Initial virus-containing specimens were obtained from the internal organs of 1 to 10-day-old chicken with histologically confirmed diagnosis of leukemia or from 8 to 11-day-old embryos of virus-bearing chicken. Tissue specimens were centrifuged, filtered and cell-free filtrate was tested in reaction of complement fixation (RCF) for the presence of antigen GS. RCF-positive filtrates were incubated with chicken embryo fibroblast (CEF) monolayer cultures. The third generation of CEF was tested for immune fluorescence (RIF). Virus-containing cultures (RIF-positive) were then inoculated into 1 to 3-day-old chicken (0.2-0.3 ml, ip); the bio-test was considered positive, if chicken developed histologically verified leukemia, or if their sera contained group-specific or type-specific antigen. (no refs)

78-4391 **Tumor Antigen Induction by the Envelope-defective Bryan Strain of Rous Sarcoma Viruses.** (Eng) Kurth, R. (Friedrich-Miescher-Laboratorium, Max-Planck-Institut, 74 Tubingen, W. Germany); Kitchener, G. *J Natl Cancer Inst* 60(6): 1365-1369; 1978.

The expression of tumor-specific cell-surface antigen(s) (TSSA) was studied in chicken, quail, and turkey cells infected and/or transformed by wild-type avian leukosis and sarcoma viruses (ALV, ASV) or by the envelope-defective Bryan high-titer strain of Rous sarcoma virus [BH-RSV(-)]. All fibroblast-transforming ASV, including BH-RSV(-), were able to induce the expression of TSSA. Thus, TSSA was group-specific for all transforming ASV. Since BH-RSV(-) is a stable deletion mutant lacking the capacity to code for the 85,000-dalton major virus envelope glycoprotein (gp85), it is unlikely that TSSA is antigenically related to the gp85 of endogenous or exogenous ALV and ASV. (29 refs)

78-4392 **Use of a Fluorescent Probe to Study the Plasma Membrane Properties of Transformed and Normal Cells (Meeting Abstract).** (Eng) Pessin, J. E. (Univ. Illinois, Urbana, IL, 61801); Salter, D. W.; Glaser, M. *Fed Proc* 37(6): 1597; 1978. (no refs)

78-4393 **Membranes and Cytochromes of Ethidium Bromide Resistant Cultured Cells (Meeting Abstract).** (Eng) Soslau, G. (Hahnemann Medical Coll., Philadelphia, PA, 19102); Zavodny, P. J. *Fed Proc* 37(6): 1725; 1978. (no refs)

78-4394 **Correlation of Procollagen mRNA Levels in Normal and Transformed Chick Embryo Fibroblasts with Different Rates of Procollagen Synthesis.** (Eng) Rowe, D. W. (Dept. Pediatrics, Univ. Connecticut Health Center, Farmington, CT, 06032); Moen, R. C.; Davidson, J. M.; Byers, P. H.; Bornstein, P.; Palmiter, R. D. *Biochemistry* 17(9): 1581-1590; 1978.

Procollagen (PC) messenger RNA (mRNA) obtained from chick embryonic tendon cells, cultured fibroblasts, and fibroblasts transformed by Rous sarcoma virus (RSV) was measured by translation in a modified reticulocyte lysate and by hybridization with a complementary DNA (cDNA). The lysate was treated with Staphylococcal nuclease to destroy endogenous mRNA and chromatographed on a Sephadex column to reduce the endogenous amino acid content. The optimal conditions for PC mRNA translation included 40 mM sodium, 6 mM phosphate, and 0.4 mM spermidine in addition to the usual reagents. With these methods, PC mRNA activity could be detected in cells synthesizing only 1%-2% PC. The immunoprecipitable product was degraded by collagenase, and it migrated slightly slower than authentic secreted PC upon polyacrylamide gel electrophoresis. PC mRNA was isolated from embryonic chick tendons and purified by oligo(dT)-cellulose chromatography and sucrose gradient centrifugation. The mRNA activity was located in the 27S region of the gradient; this RNA was used to synthesize PC cDNA. The purified cDNA was about 900 nucleotides long, it was specific for PC mRNA, and it hybridized to 75% with RNA from cells synthesizing PC. The PC mRNA concentration correlated well with procollagen synthesis rates in tendon fibroblasts in situ, fibroblasts in culture, and in RSV-transformed fibroblasts. When tendon fibroblasts were grown in culture, the PC mRNA concentration fell about threefold; the concentration was 1.5-fold lower at high cell density than at low density. RSV transformation lowered PC mRNA levels another four- to tenfold at both high and low density. These results indicate that the extent of PC synthesis is determined primarily by PC mRNA levels rather than by translational control. (43 refs)

78-4395 **Concordance of the RNA Termini of Recombinants from Crosses Between Avian Retroviruse with Different Termini.** (Eng) Joho, R. H. (Institut fu Molekularbiologie I, Universitat Zurich, Honggerberg, 809 Zurich, Switzerland); Billeter, M. A.; Weissmann, C. *Virology* 85(2): 364-377; 1978.

The 5′- and 3′-terminal RNase T_1-resistant oligonucleotide of avian retroviruses and recombinants arising from crosse between them were analyzed. The parent viruses were (1 ts337-C, a temperature-sensitive mutant of the Prague C strain of Rous sarcoma virus (Pr RSV-C), and Rous-associated virus 6 (RAV 6) and (2) Prague B strain RSV and RAV 1. In a previous study, several RNA recombinants appeare to contain the 5′-proximal part of one parent and the 3 proximal part of the other parent; however, the termin regions themselves were not analyzed. Of the 10 recombinants examined in this study, all contained 5′- and 3′-termina regions derived from only one of the parents. The structure of the termini were compatible with the existence of subterminal redundancy in all recombinant viral RNA's, as has bee demonstrated for the parental RNA's of Pr RSV-C and avia myeloblastosis virus. Six of the 10 recombinants had crossovers in the immediate vicinity of the viral termini, possibly a a consequence of proviral DNA synthesis. (31 refs)

78-4396 **The Distribution of Virus-Specific RNA i Rous Sarcoma Virus-induced Hamster Tumo Cells.** (Eng) Brugge, J. S. (Dept. Pathology, Univ. Colorad Medical Center, Denver, CO, 80262); Purchio, A. F.; Erikson, R. L. *Virology* 83(1): 27-33; 1977.

The size distribution of virus-specific RNA in the nucleus an cytoplasm of Rous sarcoma virus (RSV)-transformed hamster cells was studied. Cytoplasmic RNA from cloned cell derived from tumors induced in Syrian hamsters by th Schmidt-Ruppin strain of RSV was fractionated int polyadenylated and nonpolyadenylated RNA. The nonpolyadenylated RNA contained no detectable viral RNA sequences. When the polyadenylated RNA was fractionated b sucrose density gradient centrifugation and the RNA fron each fraction hybridized with ^{3}H-complementa DNA (^{3}H-cDNA), only one peak of viral RNA w detected. This species sedimented at the same rate the 21S class of virus-specific RNA found in RS infected chicken cells, and there was no change i size pattern in late-passage tumor cells that becam negative for group-specific antigens. Hybridizatio of fractionated nuclear RNA with ^{3}H-cDNA produce three classes of virus-specific nuclear RNA sedimenting a 47S, 35S, and 21S. Sedimentation of nuclear RNA unde denaturing conditions did not change this size distribution The data suggest that the nonpermissive behavior o RSV-transformed cells is related to improper processing o viral RNA in the cells. (19 refs)

3-4397 A Mutant of Rous Sarcoma Virus with a Thermolabile Defect in the Virus Envelope. (Eng)
ato, F. (Instituto Superiore di Sanita, 00161 Rome, Italy);
eamand, J. A.; Wyke, J. A. *Virology* 88(1): 71-81; 1978.

he temperature sensitive (*ts*) defect in *LA*30A, a mutant of
ie Prague strain of Rous sarcoma virus (RSV), was studied.
eplication of the mutant was studied by the formation of
ifective centers. The heat-labile virion component of *LA*30A
fected the initiation of transformation and replication, in-
icating that the lesion was of the early coordinate type. The
efective component of *LA*30A appeared to function before
rovirus synthesis, in that the time required for it to pass
ırough its *ts* stage was far shorter than for *LA*335, which
as a *ts* RNA-dependent DNA polymerase. Also, once *LA*
0A penetrated the cell, the infection was no longer *ts*.
seudotypes of both Bryan high-titer strain RSV and vesicu-
ır stomatitis virus with envelopes donated by *LA*30A
howed heat lability typical of the mutant. All these data
trongly suggest that *LA*30A possesses a *ts* defect in the viral
nvelope glycoproteins. When the mutant underwent pheno-
ypic mixing with Rous-associated viruses (RAV), the *ts* de-
ect was stabilized, even when the infected cells lacked recep-
ors for RAV envelope glycoproteins. The possible reasons
or this effect are discussed. (29 refs.)

**8-4398 Aggregation of Platelets and Cell Membrane Vesiculation by Rat Cells Transformed In Vitro
y Rous Sarcoma Virus.** (Eng) Gasic, G. J. (Dept. Pathology,
Jniv. Pennsylvania, Sch. Medicine, Philadelphia, PA,
9104); Boettiger, D.; Catalfamo, J. L.; Gasic, T. B.; Stewart,
3. J. *Cancer Res* 38(9): 2950-2955; 1978.

The acquisition of in vitro platelet-aggregating activity was
nvestigated in normal rat kidney cells transformed by the
377 strain of Rous sarcoma virus (RSV), and LR primary
at embryo cells transformed by the Schmidt-Ruppin strain
f RSV. Both types of transformed cells induced the aggrega-
ion of rat platelets in vitro. The aggregating activity was
pecific for the transformed cells and was absent in the nor-
nal parent cells. The aggregation reaction was accompanied
y the release of serotonin from the platelets. Further study
ınd purification of this activity from the transformed cells
lemonstrated that the activity was shed from the cells grow-
ng in culture and that it was associated with membrane vesi-
les of heterogeneous size. Normal cells also produced vesi-
les in culture, but the level of vesicle production was less
han that in the transformed cells. Furthermore, the platelet
ggregation and serotonin release activities in these vesicles
vere greatly reduced or absent. (32 refs.)

**78-4399 Selection of Nonidentified Viral Sequences of the Avian Sarcoma and B77-C Virus (Meeting
Abstract).** (Fre) Pluquet, N. (Unite de Virologie, U. 102 INSERM, place de Verdun, 59045 Lille Cedex, France); Stehelin, D. *Bull Cancer (Paris)* 65(1): 40; 1978. (no refs)

78-4400 Resistance to Marek's Disease. Effect of *Corynebacterium parvum* and Marek's Tumor Cell Vaccines on Tumorigenesis in Chickens. (Eng) Donahoe, J. (Dept. Veterinary Preventive Medicine, Ohio State Univ., 1900 Coffey Road, Columbus, OH, 43210); Kleven, S.; Eidson, C. *J Natl Cancer Inst* 60(4): 829-833; 1978.

Tumor formation was studied in chickens after nonspecific cell-mediated immunity (CMI) stimulation with *Corynebacterium parvum* and specific CMI stimulation with fixed Marek's disease (MD) tumor cells. Nonspecifically stimulated outbred White Leghorn-type cockerels had higher incidences of tumors than did controls. A study of tumor cell cytotoxicity of sera from MD virus-exposed birds indicated that humoral factors may have some role in MD tumor cell control, but probably not a major one. The results suggest that cellular immunity is more important in MD tumor cell vaccine protection than humoral immunity. (21 refs)

78-4401 Sites of Integration of Reticuloendotheliosis Virus DNA in Chicken DNA. (Eng) Keshet, E. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI, 53706); Temin, H. M. *Proc Natl Acad Sci USA* 75(7): 3372-3376; 1978.

The pattern of integration of spleen necrosis virus (SNV) DNA in DNA from a large population of SNV-infected chicken cells was studied by nucleic acid hybridization with iodinated viral RNA with the use of a blotting technique. SNV DNA was found to be integrated at multiple sites in acutely infected chicken cells. Concomitantly with the transition from acute to chronic infection, pattern of integration shifted. The majority of integrated SNV DNA found in acutely infected cells was absent from chronically infected cells. This result is consistent with the hypothesis that the cell death that occurs after infection of avian cells with reticuloendotheliosis viruses is a consequence of the multiple integrations of the provirus. Viral DNA was also integrated at multiple sites in chronically infected cells. However, infectious viral DNA molecules in chronically infected cells migrated uniformly during agarose gel electrophoresis after *Eco*RI digestion (which does not cut viral DNA), indicating that not all integrated SNV copies are equally infectious. (20 refs.)

78-4402 Biochemical Evidence of the Nonintegrated Status of Marek's Disease Virus DNA in Virus-Transformed Lymphoblastoid Cells of Chicken. (Eng) Tanaka, A. (Lab. Molecular Virology, Life Sciences, Inc., St.

Petersburg, FL, 33710); Silver, S.; Nonoyama, M. *Virology* 88(1): 19-24; 1978.

The status of latent virus genomes in MKT-1 cells, a lymphoblastoid cell line derived from a kidney tumor in a Marek's disease virus (MDV)-infected chicken, was studied. The cells were virus-nonproductive, and they contained 15 copies of MDV genomes per cell, 80%-90% of which was separable from the high-mol-wt cellular DNA. The fast-sedimenting viral DNA from MKT-1 cells showed values of 90S to 100S in a neutral glycerol gradient and a value of 200S in an alkaline glycerol gradient, indicating that the 100S DNA is probably in the form of closed circular molecules. When 100S molecules were centrifuged to equilibrium in an ethidium bromide-CsCl gradient, at least 60% of the molecules were located in the region where the simian virus 40 DNA component 1 was found. It is possible that the circular plasmid form of viral DNA is the stable and preferred form in herpesvirus-transformed cells. (16 refs.)

78-4403 **Herpesviruses and Cancer (Letter to Editor).** (Eng) Makari, J. G. (Makari Res. Labs., Inc., Englewood, NJ, 07631). *Lancet* 1(8076): 1256-1257; 1978.

An association between Marek's disease herpesvirus (MDHV) and human cancer, including carcinoma of the colon, is reported. An immunological technique capable of detecting 0.1 femtogram, a sensitivity more than a million times that of radioimmunoassay methods, was used. The association was based on the finding of common antigenicity between glycoprotein antigens extracted from human cancers and those extracted from tumors of chickens infected with MDHV. Additional evidence for this association is given. (4 refs)

78-4404 **Transformation of Indian Muntjac Cells by Murine and Avian Sarcoma Viruses.** (Eng) Yuasa, Y. (Inst. Medical Science, Univ. Tokyo, Shiroganedai 4-6-1, Minato-ku, Tokyo 108, Japan); Hirai, R.; Huh, N. H.; Shimojo, H.; Yamamoto, T. *Gann* 69(3): 441-445; 1978.

The transformation of Mm-2K Indian muntjac cells by murine sarcoma virus (MSV) and avian sarcoma virus (ASV) is reported. Of the various MSV and ASV strains tested, only the MSV with the envelope properties of xenotropic murine leukemia virus (X-MuLV) [MSV(X-MuLV)], the B77 strain of ASV subgroup C, and the Schmidt-Ruppin strain of Rous sarcoma virus subgroup D (SR-RSV-D) were capable of transforming the cells. Colony formation in soft agar was examined by infecting monolayers of Mm-2K cells with high multiplicities of infection of the transforming viruses. After 2 wk, many colonies were seen in the SR-RSV-D-infected cells and a few were noted in the B77-infected cells, but no colonies were seen in the MSV(X-MuLV)-infected cells. The ASV-transformed cell clones differed from each other w respect to morphology, presence of inducible ASV genor and karyotype. (8 refs.)

78-4405 **The Effect of Heat-inactivated Muri Cytomegalovirus on Host DNA Synthesis Different Cells.** (Eng) Gonczol, E. (Dept. Microbiolo Medical Sch. Debrecen, 4012 Debrecen, Hungary); Stone, Melero, J. M. *J Gen Virol* 39(3): 415-426; 1978.

Heat-inactivated murine cytomegalovirus (MCMV) stimul ed cellular DNA synthesis in whole mouse embryo cells, n mal murine mammary gland cells, murine fibrobla (BALB/c 3T3 A31), Chinese hamster fibroblasts (WgI primary chicken embryo cells, and Syrian hamster fibrobla (NIL-8), as evidenced by a two- to eightfold increase in incorporation of methyl-^{3}H-thymidine into the total DNA virus-infected cells. Incorporation of label was lower or changed with active or UV-treated MCMV. NIL-8 and ch cells were the least susceptible to virus replication and w thus chosen for further experiments. During the 48-hr stu period, both nuclear and mitochondrial DNA (nDNA a mDNA) synthesis was stimulated in these cells, but there no viral DNA synthesis. The stimulation of nDNA synthe was about 5 times greater in NIL-8 nDNA and about times greater in chick cell nDNA than in mock infected ce as measured by rate of incorporation of ^{3}H-thymidine. T stimulation was about 9.5-fold in NIL-8 and 1.7-fold in ch cells as detected by autoradiography. There was a 3-fold a 2.2-fold increase in the incorporation of label into the mDN of NIL-8 and chick cells, respectively. The amount of mDN in both kinds of infected cells was about twice that in cont cells. The synthesis of mDNA was also stimulated by a fac of 2 in thymidine kinaseless 3T3 cells that incorporate exo nous thymidine exclusively into mDNA. Although MCMV specific antigens were detectable by immunofluor cence at 5 hr after infection, diffuse nuclear antigens w demonstrated in NIL-8 and chick cells 24 hr after infecti Since there was no virus DNA synthesis in the cells, th antigens can be considered early antigens, coded for by parental virus DNA. (29 refs)

78-4406 **Immunohistochemical Detection of Cross-reacting Virus Antigen in Mouse Ma mary Tumors and Human Breast Carcinomas.** (E Mesa-Tejada, R. (Inst. Cancer Res., Coll. Physicians Surgeons Columbia Univ., 701 W. 168th St., New York, N 10032); Keydar, I.; Ramanarayanan, M.; Ohno, T.; Fenog C.; Speigelman, S. *J Histochem Cytochem* 26(7): 532-5 1978.

An indirect immunoperoxidase method was used to local mouse mammary tumor virus (MMTV) antigens in para sections of mammary tumors of Paris RIII and CD8F$_1$ m nd to detect an antigen that cross-reacts with a group-specif- antigen (gp52, a 52,000-dalton glycoprotein) of MMTV in araffin sections of human breast carcinomas. The specificity f the staining reaction with antibody against MMTV was xamined by absorption of IgG with: (1) purified gp52; (2) everal relevant and irrelevant viral preparations; (3) normal uman plasma, WBC breast tissue, milk, actin, collagen, and yaluronic acid; (4) sheep RBC, bovine mucin, and fetal calf erum. Only MMTV and purified gp52 eliminated the im-nunohistochemical reaction in human breast tumors. Posi-ive reactions were seen in 73/191 human breast carcinomas f various histopathologic types, but negative reactions were btained in all 137 normal and benign cases tested. A positive eaction of uncertain specificity was observed in foci of apo-rine metaplasia. With one exception, 99 carcinomas from 13 rgans other than breast and 8 cystosarcomas were negative. 40 refs.)

8-4407 **Polyproteins Related to the Major Core Protein of Mouse Mammary Tumor Virus.** (Eng) Dick-on, C. (Imperial Cancer Res. Fund Labs., Lincoln's Inn Fields, London, WC2A 3FX, England); Atterwill, M. *J Virol* 26(3): 660-672; 1978.

Immunoprecipitation with monospecific antiserum and tryptic peptide analysis were used to characterize the polyproteins related to the major core protein, p27, of murine mammary tumor virus (MuMTV). Immunoprecipitation of cell extracts derived from the mammary tumor cell lines GR and Mm5MT with anti-MuMTV-p27 serum resulted in the isolation of several proteins with mol wts of 34,000 to 160,000. Pulse-chase experiments and tryptic peptide analyses of these proteins demonstrated that p77/75, p61, p48, p38, and p34 were related by amino acid sequence to p27 of the virion. The p34 to p61 proteins were probably derived by proteolytic cleavage of the p77/75 protein. After a 15-min pulse with ^{35}S-methionine, all of the p27-related proteins in the GR and Mm5MT cell lines were labeled and, during a subsequent chase, all progressively disappeared. The p27 was labeled poorly during the pulse, but the amount of label increased during the chase. The majority of the p27-related proteins were rapidly turned over in the cell rather than being processed into p27. The GR and Mm5MT cell lines showed a clear difference in their ability to convert the presumptive precursors into p27. When MuMTV was isolated from the culture medium of mammary tumor cells harvested at 5-min intervals, the major p27-related protein was p34. p27 accounted for only 29% of the anti-p27 serum immunoprecipitable proteins, compared with 95% in virus isolated from an 18-hr harvest. Incubation of the rapid-harvest virus at 37 C for 2 hr resulted in some conversion of p34 to p27. The data suggest that some of the p27 in MuMTV is formed in the virions by proteolytic cleavage of p34. (41 refs.)

78-4408 **Bioactivity of Male (Sperm Transmitted) Mouse Mammary Tumor Virus as Influenced by Donor, Recipient and Age at Treatment.** (Eng) Squartini, F. (Spedali Riuniti di S. Chiara, Istiuto di Anatomia e Istologia Patologica, Via Roma 57, 56100 Pisa, Italy); Di Coscio, G. *Tumori* 64(2): 103-114; 1978.

Sperm collected from mammary tumor virus (MTV)-carrying mice (C3H, BALB/cfC3H, BALB/cfRIII, and RIII) was tested separately for its mammary tumor-inducing activity in (BALB/c x C3Hf)F_1, (BALB/c x RIIIf)F_1, and BALB/c female recipients inoculated ip with 0.1 ml of the sperm at age 1-2 wk or 3 mo. A total of 551 recipients were observed, including control mice. Mammary tumor incidence was not affected by the presence or absence of histocompatibility between the sperm donors and the recipient mice. The incidence was related to the type of sperm-transmitted MTV (C3H or RIII), and it was influenced by age at inoculation. MTV of C3H origin tended to be more effective than that of RIII origin in 1- to 2-wk-old recipients, whereas the latter was equally effective in both age groups. BALB/c recipients were susceptible to C3H infection, but (BALB/c x RIIIf)F_1 hybrids were resistant to sperm-released MTV, especially to C3H infection. These hybrids had a 34% incidence of late spontaneous lymphomas inherited by the RIIIf male parent. (BALB/c x C3Hf)F_1 hybrids were susceptible to both C3H and RIII sperm-released MTV, and they had a 30% incidence of late spontaneous mammary tumors due to genetic transmission of MTV by the C3Hf male parent. (40 refs.)

78-4409 **Typing of Murine Oncornavirus by Thin Layer Gel Electrophoresis (Meeting Abstract).** (Fre) Chuat, J. C. (U. 107 INSERM, Hopital Saint-Louis, 75475 Paris, Cedex 10, France); Laprevotte, A.; Pilon, C. *Bull Cancer (Paris)* 65(1): 36; 1978. (no refs)

78-4410 **The Presence of Type C-like P30 Antigen in Human Prostatic Tissues (Meeting Abstract).** (Eng) Kouttab, N. M. (Dept. Molecular Carcinogenesis, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); von Eschenbach, A.; Bowen, J. M.; Dmochowski, L. *Proc Am Assoc Cancer Res* 19: 42; 1978. (no refs)

78-4411 **Biological and Biochemical Differences Among Ecotropic C-Type RNA Viral Isolates Chemically Induced from C57BL/Ka Mouse Embryo Cells in Vitro.** (Eng) Gelmann, E. P. (Cancer Biology Res. Lab., Dept. Radiology, Stanford Univ. Sch. Medicine, Stanford, CA, 94305); Decleve, A.; Kaplan, H. S. *Virology* 85(1): 198-210; 1978.

The role of selection pressure on the characteristics of endogenous ecotropic viruses isolated from C57BL/Ka mouse embryo fibroblasts was studied, and the isolates were compared with those from radiation-induced lymphomas arising in this strain. Selection pressure has been found to operate during the isolation of viruses after in vitro induction by 5′-iododeoxyuridine (IUdR). During passage in cultures of both Fv-1 genotypes, it became apparent that the IUdR-induced viral isolates differed distinctively with respect to their Fv-1 tropism and XC plaque-forming ability. These differences appeared to depend on the type of overlay cell in which each virus was first recovered after induction, its subsequent intracellular passage history, its extracellular transfer from one culture to another, and the purification of particles by endpoint dilution on permissive cell lines. Although the original induced isolates were N-tropic, B-tropic virus emerged during passage of one of the isolates. Two distinct N-tropic viruses were also recovered in the process, one of which appears to be identical to BL/Ka(N). Polyacrylamide gel electrophoresis was also used to compare the polypeptides of the viral isolates. Differences in the electrophoretic behavior of the low-mol wt polypeptides were observed that seem to correlate with specific types of viruses classified on the basis of biologic criteria. Similar selection pressures applied to murine RNA virus isolates by in vivo and in vitro manipulations probably explain the numerous discrepancies in the literature with respect to their biologic activities. (39 refs)

78-4412 **Differential Activation of Endogenous Murine Retrovirus in Mouse-Monkey Somatic Hybrids (Meeting Abstract).** (Fre) Sergiescu, D. (INSERM U5, Hopital Cochin, Paris, France); Rousset, S.; Gisselbrecht, S.; Sinoussi, F. *Bull Cancer (Paris)* 65(1): 35; 1978. (no refs)

78-4413 **The Occurrence of Murine Leukemia Virus Antigens in Membranes of Serially Passaged B/c Embryo Cells (Meeting Abstract).** (Eng) Meek, R. L. (Univ. California, Santa Cruz, CA, 95064); Bowman, P. D.; Daniel, C. W. *In Vitro* 14(4): 387; 1978. (no refs)

78-4414 **Cellular Immune Response Induced by the Radiation Leukemia Virus (RadLV).** (Eng) Peled, A. (Dept. Chemical Immunology, Weizmann Inst. Science, Rehovot, Israel). *Leuk Res* 1(4): 333-343; 1977.

The dynamics of the appearance of the cell-mediated immunity responsible for leukemic cell destruction were investigated in female C57BL/6 mice inoculated intrathymically with 0.02 ml of radiation leukemia virus (RadLV). This immunization made the mice resistant to the isotransplantation of leukemic cells (5 x 10^5) for 2 mo. Lymphoid cells from the thymus, lymph nodes, spleen, and peritoneal exudate of immune mice retarded tumor growth. RadLV-inoculated mice had transplantation resistance only to leukemic cells induced by RadLV (127 LC), but not to radiation-induced leukemias
(XRL-1, XRL-2, XRL-3), to EL_4, or to any other syngene
tumors tested. The immunological specificity of the lympho
cells from immunized mice was tested in vivo and in vit
against C57BL/6 leukemic cells induced by RadLV, chem
cal carcinogens, and x-rays. The cells were found to be high
specific for the presence of RadLV associated antigens c
their cell surface. A cytostatic effect rather than a cytolyt
effect was noted when effector:leukemic cell interaction w
tested in vitro. (38 refs)

78-4415 **Cytolysis and Transformation Induced**
Leukemogenic Recombinant Radiation Le
kemia Virus Isolated from Thymoma Cell Lines and by Vir
from Leukemic Thymus Epithelial-Reticulum Cells. (En
Haas, M. (Dept. Cell Biology, Weizmann Inst. Scienc
Rehovot, Israel). *In: Advances in Comparative Leukemia R*
search 1977. Proceedings of the VIIIth International Sympo
um on Comparative Research on Leukemia and Related D
ease, held in Amsterdam, The Netherlands on 22-
August, 1977. Bentvelzen, P.; Hilgers, J.; Yohn, D.
eds. (New York Elsevier/North Holland Biomedi
Press): 503 pp.; 115-126; 1978.

Various properties of the thymotropic leukemogenic vir
produced by the 136.5 thymoma cell line and of virus pr
duced by growth on leukemic thymus epithelial reticulu
cell monolayers are presented. Virus produced by bo
sources was found to be cytolytic and replication defecti
Some of the viruses were able to induce foci in, and morph
logically transform, mink lung cells. The thymotrop
leukemogenic virus was also shown to be recombinant in t
major surface glycoprotein gp70, which contains informati
from both ecotropic and xenotropic viruses. As a model f
this recombination, it is suggested that (1) during ecotrop
proviral integration, the ecotropic virus recombines with re
dent xenotropic viral sequences; (2) the infected thymus e
thelial reticulum cells can thus synthesize recombinant vir
in addition to ecotropic virus; and (3) infection of susceptib
thymocytes or thymocyte precursor cells occurs
cell-to-cell contact. It is conceivable that only a minority
the recombinant viruses synthesized would be able to infe
and transform thymocytes. Those that transform thymocyt
will be amplified and found frequently, but the others w
be rare. (26 refs)

78-4416 **Transmission of Autoimmune Haemolytic An**
mia and Murine Leukaemia Virus to Hybr
Progeny of Allophenic NZB <—> CFW and NZB <
BALB/c Male Mice. (Eng) East, J. (Dept. Immu
pathology, Imperial Cancer Res. Fund, Burtonh
Lane, Mill Hill, London NW7 1AD, England); Harv
J. J.; Tuffrey, M.; Tilly, R. J. *Clin Exp Immunol* 31(
260-269; 1978.

illy, R. J. *Clin Exp Immunol* 31(2): 260-269; 1978.

he transmission of autoimmune hemolytic anemia and mu-
ine leukemia virus (MuLV) was studied in 116 progeny of
llophenic (tetraparental) NZB<->CFW and NZB<->
ALB/c male mice. Of two NZB<->CFW males mated to
ormal albino BALB/c partners, one had an NZB (black)
nd the other a CFW (white) genotype, producing agouti-col-
red F_1(BALB/c x NZB) and albino F_1(BALB/c x CFW)
rogeny, respectively. The former developed a protracted
ourse of autoimmune hemolytic disease and diverse lym-
hoid, lung, and liver tumors and carried C-type MuLV.
ith respect to these features, they were identical to F_1
BALB/c x NZB) natural hybrids. On the other hand, no
utoimmune hemolytic anemia, very little virus, and a pre-
onderance of lung tumors developed in the albinos. A third
ZB<->BALB/c male also sired agouti F_1 and varicolored
$_2$(BALB/c x NZB) hybrids showing the same pattern of
utoimmunity, malignancies, and virus infection. When this
nale was additionally mated to NZB females, he produced
wo generations of black (NZB x NZB) descendants that dis-
layed the typical NZB profile of autoimmune hemolytic dis-
ase. Therefore, only the progeny of allophenic fathers having
he NZB genotypes expressed autoimmunity and large num-
ers of MuLV particles. It is concluded that both autoim-
nunity and virus were transmitted by incorporation of genet-
c information. The range and type of malignancies were also
genetically determined. These findings suggest that the sole
virus in NZB mice is xenotropic. (11 refs)

78-4417 **Assignment of the Receptor for Ecotropic Murine Leukemia Virus to Mouse Chromosome 5.** (Eng) Ruddle, N. H. (Dept. Epidemiology, Yale Univ., New Haven, CT, 06520); Conta, B. S.; Leinwand, L.; Kozak, C.; Ruddle, F.; Besmer, P.; Baltimore, D. *J Exp Med* 148(2): 451-465; 1978.

The genetic control of ecotropic (mouse-tropic) murine leukemia virus (MuLV) replication was analyzed at the receptor level. The experimental material consisted of clones derived from crosses between Chinese hamster cells (E36, a line that segregates murine chromosomes) and murine peritoneal exudate cells (A/HeJ and *Mus poschiavinus* strain I) or embryo fibroblasts (BALB/c). Mouse-Chinese hamster hybrids were analyzed for susceptibility to ecotropic MuLV infection by the vesicular stomatitis virus (MuLV)[VSV(MuLV)] pseudotype assay and the XC cell assay. Segregation of mouse chromosomes in the hybrids was analyzed by chromosome banding and isozyme expression. The ability of the hybrid cells to absorb and replicate both VSV(MuLV) pseudotype virus and ecotropic MuLV segregated with chromosome 5 was demonstrated. Three statistical procedures confirmed that chromosome 5 was essential for receptor expression. Segregation of the receptor in two series of subclones demonstrated that, in both, receptor expression was syntenic with phosphoglucomutase-1, an isozyme that has been mapped to mouse chromosome 5. On the basis of these results, the gene for the receptor for ecotropic MuLV (*Rev*) has been assigned to mouse chromosome 5. (35 refs.)

78-4418 **Are Bone Marrow Derived Fibroblastoid Colony-Forming Cells Involved in Viral Leukemogenesis?** (Eng) Zipori, D. (Radiobiological Inst. TNO, Lange Kleiweg 151, Rijswijk, Netherlands). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 314-315; 1978.

Bone-marrow derived fibroblastoid colony-forming cells were studied in mouse strains with a low and high incidence of spontaneous leukemia. A correlation was noted between high leukemia incidence and ecotropic virus-producing fibroblastoid cells in the marrow. If these cells are involved in regulating hemopoiesis, their infection by leukemia viruses could be the cause of preleukemic hemopoietic changes. They could also be the site of malignant transformations of hemopoietic cells by simultaneously providing differentiation signals and oncogenic viruses. (2 refs)

78-4419 **Detection of Vesicular Stomatitis Virus (Murine Leukemia Virus) Pseudotypes by Immunoelectron Microscopy.** (Eng) Chan, J. C. (Dept. Molecular Carcinogenesis and Virology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. Tumor Inst., Houston, TX, 77030); Hixson, D. C.; Bowen, J. M. *Virology* 88(1): 171-176; 1978.

Immunoelectron microscopy (IEM) was used to detect vesicular stomatitis virus (VSV) pseudotypes formed by superinfection of a murine leukemia virus (MuLV)-producing rat bone tumor cell culture with the temperature-labile VSV mutant t1-17. The VSV pseudotype was detected by IEM as bullet-shaped VSV particles reacting with anti-MuLV serum. The IEM observation of VSV(MuLV) pseudotype formation was confirmed by the results of thermostability and neutralization tests. It is expected that IEM will find broader application for the demonstration of other pseudotypes that cannot be assayed readily by conventional infectivity assays. (14 refs.)

78-4420 **Immunocytochemical Detection of a Murine Leukemia Virus-related Nuclear Antigen in Mouse Oocytes and Early Embryos.** (Eng) Piko, L. (Developmental Biology Lab., Veterans Admin. Hosp., Sepulveda, CA 91343). *Cell* 12(3): 697-707; 1977.

The expression of murine leukemia virus (MuLV)-related antigens in oocytes and early embryos of Swiss mice was studied using the unlabeled antibody peroxidase-antiperoxidase method with antiserum to the major core protein (30) of AKR MuLV. At antibody dilutions up to 1:500, intense staining of the germinal vesicles of oocytes and the interphase nuclei of early embryos was noted. Nuclear staining was restricted to a specific period of oocyte growth and development. It developed gradually in oocytes having reached one-third to one-half the full-grown size, and it persisted until meiotic maturation. In early embryos, nuclear staining was present from the one-cell stage to the morula, but it disappeared after transition to the blastocyst. Nuclear staining was abolished by absorption of anti-p30 serum with disrupted virions of Gross(A), AKR, Kirsten and Moloney MuLV, and Kirsten murine sarcoma virus, but not with the Friend and Rauscher strains of MuLV, xenotropic BALB:virus 2, a mouse-tropic and an amphotropic clone of wild mouse MuLV, or with feline leukemia virus and Rous sarcoma virus. Anti-p30 serum reacted with an AKR MuLV preparation to produce a strong band in the p30 region upon electrophoresis; smaller strongly reacting bands with approx mol wts of 65,000 and 38,000 daltons were also detected. These findings demonstrate the development of stage-dependent expression of MuLV p30-related antigen in mouse oocytes and early embryos. (63 refs.)

78-4421 **Analysis of Erythroid Precursor Cells in Friend Virus Infected Mice.** (Eng) Bertoncello, I. (Dept. Clinical Physiology, Univ. Ulm, 7900 Ulm, Germany); Opitz, U.; Seidel, H. J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 285-287; 1977.

In DBA/2 mice infected with polycythemia-inducing strain of Friend virus (FV-P), the normal population of colony-forming units - erythropoietic (CFU-E) was replaced by an erythropoietin (EP)-independent CFU-E population, and the numbers of burst-forming units - erythropoietic (BFU-E) became undetectable. Normal and EP-independent CFU-E did not differ in modal sedimentation velocity. However, following velocity sedimentation of FV-P-infected marrow cell suspensions, BFU-E reappeared at a modal sedimentation velocity not significantly different from that of controls, which precludes the theory that their disappearance is a result of stem cell damage. (5 refs)

78-4422 **Concanavalin A-Enhanced In Vitro Infection of Murine Spleen Cells by Friend Virus (Meeting Abstract).** (Eng) Isaak, D. D. (Harvard Uni
Boston, MA, 02115); Price, J. A.; Cerny, J. *Fed Pr*
37(6): 1560; 1978. (no refs)

78-4423 **A Single Non-H-2 Gene is Required f**
H-2b/b-Associated Recovery from Friend Vi
Leukemia in Mice. (Eng) Chesebro, B. (Rocky Mount
Lab., NIH, Hamilton, MT); Wehrly, K. *In: Advances*
Comparative Leukemia Research 1977. Proceedings of
VIIIth International Symposium on Comparative Research
Leukemia and Related Diseases, held in Amsterdam, T
Netherlands on 22-26 August, 1977. Bentvelzen, P.; Hilge
J.; Yohn, D. S., eds. (New York: Elsevier/North Holla
Biomedical Press): 503 pp.; 69-73; 1978.

The backcross murine population (C57BL/10 x A.BY)F
A.BY, in which genes other than the major histocompatib
ty complex (H-2) were segregating, was studied to determi
the number of C57BL genes involved in the recovery fr
H-2b/b-associated Friend virus (FV) leukemia. When A.
mice (H-2b/b) were inoculated with B-tropic FV (FV-
75% developed marked leukemic splenomegaly within
days and none showed any indication of recovery from l
kemia. In contrast, the (C57BL/10 x A.BY)F_1 mice (a
H-2b/b) showed a high incidence of recovery (79% at
days), although splenomegaly was evident in 72% by day
Within 68 days after inoculation,45%(C57BL/10 x A.BY)
x A.BY backcross mice had recovered from leukemia, s
gesting that a single, autosomal, dominant, non-
C57BL/10 gene is required for expression of
H-2b/b-associated effect on recovery from FV leuken
When recombinant-inbred C x B (BALB/c x C57BL
mouse strains were tested for recovery from FV leukemia,
difference in FV-2 genotypes between the (C x B) E a
(C57BL/10 x BALB/B)F_1 mice did not appear to influe
recovery from FV leukemia. Although the identity and m
location of the non-H-2 C57BL/10 gene necessary for rec
ery in the A.BY backcross population are not known,
latter experiments indicate that the FV-2 gene is not lik
to be required. (10 refs.)

78-4424 **Histone Synthesis and Differentiation in M**
rine Virus-Infected Erythroleukemia Ce
(Eng) Tanaka, M. (Dept. Human Genetics and Developme
Sch. Physicians and Surgeons, Columbia Univ., New Yo
NY). *Tohoku J Exp Med* 124(1): 13-20; 1978.

The effects of dimethyl sulfoxide (DMSO) on the synth
of chromosomal proteins in murine virus-infected e
throleukemia cells (MELC) were studied. DS 19 cells clo
from MELC line 745A were incubated with 280 mM DM
The cells showed a reduced rate of growth during the f
48 hr, but the number of cells approximated that in cont
MELC cultures by 96 hr. The number of benzidine-react
cells increased progressively up to 91.0% at 5 days; cont

ultures contained only 0.2% benzidine-reactive cells at 5 ays. The rate of total protein synthesis was much lower in he DMSO-treated cells than in control cells: at 96 hr, the ates were 8.33 and 13.00 cpm x 10^{-5}, respectively. The synhesis of histone increased twofold in the 48-hr-incubation DMSO-treated cells, but that of nonhistone protein was not timulated. Histone synthesis was max at 48 hr, and by 72 r it had declined. ^{3}H-Leucine-labeled DMSO-treated cells were mixed with ^{14}C-labeled control cells and the ^{3}H/^{14}C atios in the histone fractions were monitored. At 48 hr, there was a 3.6-fold increase in the ratio in fraction 3 and 3.4-fold ncrease in fraction 2b. The data indicate that the stimulation f histone synthesis is an early event following DMSO treatment of MELC. (13 refs.)

8-4425 **Proteins of Transcriptionally Active and Inactive Chromatin from Friend Erythroleukemia Cells.** (Eng) Lau, A. F. (Dept. Microbiology, Univ. Minnesota, Minneapolis, MN, 55455); Ruddon, R. W. *Exp Cell Res* 107(1): 35-46; 1977.

Transcriptionally active and inactive chromatin fractions were prepared from control and dimethyl sulfoxide (DMSO)-treated murine erythroleukemia cells by a DNase II digestion technique. The active and inactive chromatin fractions from control and DMSO-treated cells had essentialy the same total histone content, but the active chromatin fractions from both control and treated cells contained significantly less H1 histone. The amount of nonhistone protein was similar in the corresponding chromatin fractions of control and DMSO-treated cells. For both treated and control cell chromatin, however, the content of nonhistone protein was consistently lower in the inactive fraction than in the active fraction. No significant differences were observed between the polyacrylamide gel banding patterns (determined by Coomassie blue staining) of histone or nonhistone chromatin proteins of the corresponding fractions from treated and control cells. However, dual-radioisotope-labeling studies indicated an increased incorporation of radioactive amino acids into a 50,000-dalton polypeptide of unfractionated chromatin and a 23,000-dalton polypeptide of active chromatin from DMSO-treated cells. The significance of the elevated incorporation of labeled amino acids into the 50,000- and 23,000-dalton polypeptides is not currently evident. (41 refs)

78-4426 **Comparison of RNA Components from Feline and Rodent Sarcoma-Leukemia Virus Complexes.** (Eng) Kimball, P. C. (Ohio State Univ. Comprehensive Cancer Center, Columbus, OH, 43210). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 235-236; 1978.

The S values of RNA subunits from two feline and two murine sarcoma-leukemia virus isolates (FeSV-FeLV, MuSV-MuLV) were compared in an attempt to determine the source(s) of feline oncornavirus genetic information. MuSV-MuLV isolates had a 38S MuLV component, with uniform S values in all MuLV isolates, and a more slowly moving MuSV subunit with varying S values in different isolates. FeSV-FeLV complexes contained two or three distinct RNA subunits with different S values, all lower than those for the MuLV RNA subunits. The most slowly sedimenting (25S) RNA subunit from all feline viruses was observed only in preparations containing FeSV, suggesting that this RNA component is FeSV-specific. It was also found in virus stocks obtained by rescue of FeSV with FeLV from nonproducer cells transformed with FeSV in the absence of helper virus. Electrophoresis of various RNA components suggested that the 30S-40S RNA subunits of all self-replicating C-type viruses examined (MuLV's, FeLV of different serotypes, and RD114 endogenous cat virus) have a mol wt of approx 2.3 x 10^6 daltons, despite sedimentation data suggesting mol wt differences of $> 5\%$. In contrast, the defective Rickard FeLV RNA, which lacks methylated caps on the 5′ end, and two different strains of FeSV-FeLV had mol wts of about 2.1 x 10^6 daltons. This finding suggested that specific degradation of FeLV RNA near the 5′ end is a frequent occurrence. (no refs)

78-4427 **Nucleotide Sequence Homology of Mammalian RNA Tumor Viruses.** (Eng) East, J. L. (Dept. Molecular Carcinogenesis and Virology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); Nell, L. J.; Knesek, J. E.; Chan, J. C.; Dmochowski, L. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 141-143; 1978.

A competition hybridization assay was used to quantitate nucleotide sequence relationships of C-type mammalian RNA tumor viruses. Representive members of three viral groups were examined: Friend leukemia virus, Gross leukemia virus, Moloney sarcoma virus, Kirsten leukemia virus (MuLV-K), and Kirsten sarcoma virus (MSV-K) of the murine group; gibbon ape leukemia virus and simian sarcoma virus of the simian group; and RD-114 virus of the feline group. Reciprocal cross-hybridization data revealed that (1) the murine viruses can be placed into four unrelated subgroups, with MuLV-K and MSV-K belonging to the same subgroup; (2) the two simian viruses belong to different yet related subgroups; and (3) RD-114 virus does not share sequence homology with any other virus examined. (5 refs)

78-4428 **Identification of an FMR Cell Surface Antigen Associated with Murine Leukemia Virus-infected Cells.** (Eng) Nowinski, R. C. (Fred Hutchinson Cancer Res. Center, Seattle, WA, 98104); Emery, S.; Ledbetter, J. *J Virol* 26(3): 805-812; 1978.

FMR-specific viral and cell-surface antigens, which are found on the surface of cells infected with exogenous murine leukemia viruses [MuLV: Friend (F), Moloney (M), and Rauscher (R)] were analyzed immunochemically. A variety of antisera were prepared in mice, and their reactivity against the FMR antigens of Friend, Moloney, and Rauscher MuLV was determined. In absorption tests, the cytotoxic activity of FMR antisera was removed by disrupted density gradient-purified FMR MuLV and by FMR MuLV-infected cells, but not by purified endogenous MuLV or by cells infected with endogenous viruses. Antisera prepared against G antigens (found on the surface of cells infected with endogenous or Gross MuLV) were cytotoxic for unlabeled target cells. The cytotoxic reaction of the G typing antiserum was absorbed by unlabeled target cells, but not by normal mouse spleen cells and only partially by MuLV leukemia cells. In absorption tests with viruses of the FMR and G serotypes, FMR antisera reacted primarily with the gp70, p15(E), and p12(E) proteins of MuLV. Reactions with the p15(E) and p12(E) proteins were based exclusively on the detection of group-specific antigens, whereas the reactions with gp70 were based on the detection of type-specific and group-specific antigens. G antiserum detected antigenic determinants of the major core protein, p30, of endogenous MuLV. The results of cytotoxic absorption tests and immune precipitation assays with extracts of radiolabeled cells indicated that cells infected with endogenous MuLV contained the viral envelope protein gp70 and the glycosylated polyprotein precursors of the internal structural proteins of MuLV on their cell surfaces. FMR antisera reacted with the gp70 proteins of these cells, but not with the glycosylated core polyproteins. In contrast, the G antiserum reacted with both gp70 and the glycosylated proteins. It is concluded that FMR cell-surface antigen is a determinant on the MuLV *env* gene product. (24 refs.)

78-4429 **Surface Expression of Murine Leukemia Virus Structural Polypeptides on Host Cells and the Virion.** (Eng) Schneider, J. (Forschergruppe Tumorimmunologie, D-7800 Freiburg, W. Germany); Hunsmann, G. *Int J Cancer* 22(2): 204-213; 1978.

The expression of murine leukemia virus structural polypeptides on the surface of cells producing exogenous Friend leukemia virus, endogenous ecotropic AKR virus, and xenotropic BALB/c virus was investigated. Antisera to Friend virus gp71, p30, p15E, p12, and p10 were employed in a complement-dependent chromium release assay and in the immunoprecipitation of lactoperoxidase iodinated surface polypeptides prior to their analysis by polyacrylamide gel electrophoresis. With the latter technique, gag gene-encoded proteins and their precursors were not discovered on the virus- and cell-surface membranes. Only the env gene-enco ed polypeptides gp85, gp71 and p15E were detectable. p15 was embedded into the lipid membrane, and gp85 was forme by disulfide linkage of p15E to surface-exposed gp71. Tl ratio of gp71 to gp85 was variable and apparently determine by the host cell. Antibodies of strong cytotoxicity were tho against type- and group-specific epitopes of gp71 as well type-specific epitopes of p12. (40 refs.)

78-4430 **Hormone-Sensitivity Profiles of Virus-induce T-Lymphocyte Malignancies (Meeting A stract).** (Eng) Peterson, R. D. (Dept. Pediatrics and Pha macology, Univ. South Alabama Coll. Medicine, Mobil AL, 36617); Funkhouser, J. D.; Palmer, G. C. *Fed Proc* 37(6 1748; 1978. (no refs)

78-4431 **Isolation of a Cell-Surface Antigen Associate with Gross Virus in Liposomes.** (Fre) Gerlie D. (Laboratoire d'Immunologie, Centre Leon-Berard, 28, ru Laennec, 69373 Lyon Cedex 2, France); Sakai, F.; Dore, F. *C R Acad Sci [D] (Paris)* 286(5): 439-442; 1978.

A Gross virus-associated cell-surface antigen was extracte from (C58NT)D lymphoma cells by detergent solubilizatio of the membranes and then partially purified. Absorption ex periments, in which the cytotoxicity of W/Fu r anti-(C58NT)D serum toward E(male)G_2 lymphoma cel was determined, indicated that the antigen was located on th surface of sensitized liposomes. (13 refs)

78-4432 **Glycoproteins from Murine C-Type Virus a more Acidic in Virus Derived from Transforme Cells than from Nontransformed Cells.** (Eng) Forchhamme J. (Fibiger Lab., 2100 Copenhagen, Denmark); Turnock, C *Virology* 88(1): 177-182; 1978.

Viral proteins from purified Moloney-murine sarcoma viru (murine leukemia virus) [M-MSV(MuLV)] and M-MuL grown in established BALB/3T3 cells in vitro were studie using two-dimensional electrophoresis. Several of the vir proteins were present in multiple forms differing in charg but not in size, and, except for the glycoprotein gp70, th isomers were identical in virus from transformed (TF) an nontransformed (NTF) cells. The gp70 from both types virus showed 10 to 15 spots, indicating great charg heterogeneity. The gp70 from virus from TF cells had isoele tric points that were an av of 1 pH unit more acidic than th gp70 from NTF cells, and their apparent mol wt was in creased slightly. That the TF state of the culture was impo tant for these differences to occur was substantiated using mutant M-MSV(MuLV) that is temperature-sensitive fo maintenance of transformation. It is likely that the viral gp7 is one of the substrates for the increased activity of sialyltransferase found in transformed cells. (23 refs.)

78-4433 **Genome Organization of RNA Tumor Viruses. II. Physical Maps of In Vitro-synthesized Moloney Murine Leukemia Virus Double-stranded DNA by Restriction Endonucleases.** (Eng) Verma, I. M. (Tumor Virology Lab., Salk Inst., San Diego, CA, 92112); McKennett, M. A. *J Virol* 26(3): 630-645; 1978.

In vitro-synthesized double-stranded DNA from Moloney murine leukemia virus (M-MuLV) was cleaved with the restriction endonucleases (RE) *Sal* I, *Hind* III, *Bam* I, *Hpa* I, *Sma* I, and *Bgl* I + II in an effort to construct physical maps of the M-MuLV genome. *Sal* I and *Hind* III cleaved the viral DNA at only one site generating two fragments each. The fragments generated by *Sal* I, designated *Sal* IA and *Sal* IB, had mol wts of 3.5 x 10^6 and 2.4 x 10^6, respectively. The *Hind* III fragments had mol wts of 3.6 x 10^6 (*Hind* IIIA) and 2.3 x 10^6 (*Hind* IIIB). *Bam*I generated four fragments of mol wts 2.1 x 10^6 (*Bam* IA), 2 x 10^6 (*Bam* IB), 1.25 x 10^6 (*Bam* IC), and 0.21 x 10^6 (*Bam* ID). *Hpa* I generated three fragments of mol wts 4.4 x 10^6 (*Hpa* IA), 0.84 x 10^6 (*Hpa* IB), and 0.74 x 10^6 (*Hpa* IC). *Sma* I generated four fragments of mol wts 3.9 x 10^6 (*Sma* IA), 1.3 x 10^6 (*Sma* IB), 0.28 x 10^6 (*Sma* IC), and 0.21 x 10^6 (*Sma* ID). A mixture of *Bgl* I and *Bgl* II generated five fragments of mol wts 2 x 10^6 (*Bgl* I + IIB), 1.25 x 10^6 (*Bgl* I + IIC), 0.40 x 10^6 (*Bgl* I + IID), and 0.31 x 10^6 (*Bgl* I + IIE). The order of the fragments in relation to the 5′ and 3′ ends of the genome was determined for each enzyme separately by using fractional-length M-MuLV double-stranded DNA for digestion by the RE or by redigestion of the *Sal* IA, *Sal* IB, *Hind* IIIA, and *Hind* IIIB fragments with other RE. Other RE that cleaved in vitro-synthesized M-MuLV double-stranded DNA were *Ava* I, *Sst* I, *Xho* I, *Xba* I, *Bgl* I, *Bgl* II, *Hind* II, *Hae* II, *Hpa* II, *Hin* f, and *Hae* III. (22 refs.)

78-4434 **Nonproducer Malignant Tumor Cells with Rescuable Sarcoma Virus Genome Isolated from a Recurrent Moloney Sarcoma.** (Eng) Weiland, E. (Federal Res. Inst. Animal Virus Diseases, Tubingen, W. Germany); Mussgay, M.; Weiland, F. *J Exp Med* 148(2): 408-423; 1978.

The viral, oncogenic, and antigenic properties of a transplantable tumor that reappeared at the site of a primary regressed Moloney sarcoma were studied to obtain information on the escape of tumor cells from immune destruction. Cells from the recurrent tumor were passaged in STU mice by im and ip inoculation. Tumors induced by these cells and a cell line derived from them grew progressively and led to the death of the animal 3-7 wk after transplantation. There was no evidence of virus production or of viral antigens in the tumor cells. Infection with helper virus allowed rescue of the sarcoma virus genome and led to the establishment of a cell line producing focus- and XC plaque-forming virus. The nonproducer cells were not immunogenic, but the producer cells possessed a strong immunogenicity. However, tumors induced by the producer cells frequently grew progressively. Although, they were nonimmunogenic, the nonproducer cells showed weak immunosensitivity when used as challenge cells in the transplantation protection assay or as target cells for the demonstration of antibody-mediated cytotoxicity and cell-mediated cytotoxicity, if the immune response was induced by cells producing the sarcoma-helper virus complex, but not by cells producing only helper virus. It was assumed that the recurrence of the regressed Moloney sarcoma was the result of transformed nonproducer cells escaping immune destruction and not of a depleted immune resistance in the host. (31 refs.)

78-4435 **Biochemical Characterization of the Amphotropic Group of Murine Leukemia Viruses.** (Eng) Chattopadhyay, S. K. (Pediatric Oncology Branch, Div. Cancer Treatment, NCI, Bethesda, MD, 20014); Hartley, J. W.; Lander, M. R.; Kramer, B. S.; Rowe, W. P. *J Virol* 26(1): 29-39; 1978.

Amphotropic murine leukemia virus (MuLV) strain 1504-A, isolated from feral mice (*Mus musculus*) in the La Puente trapping area of California, was studied by nucleic acid hybridization to determine its relationship to other MuLV's and to determine the distribution of the genome sequences in various mouse strains. A $[^3H]$DNA probe synthesized in an endogenous reaction from detergent-lysed 1504-A indicated that the amphotropic MuLV's are distinct biochemically: 20% of the viral genome sequences were not shared by AKR-type ecotropic or any of the three types of xenotropic MuLV's (NZB-IU-1, BALB-IU-1, and Cas E No. 1-x) tested. One-half of these amphotropic unique sequences were present in the genome of wild mouse ecotropic viruses and in Moloney and Rauscher viruses. Sequences homologous to the entire genome of 1504-A amphotropic virus were present in the cellular DNA of all eight inbred mouse strains tested, as well as in wild *Mus* in Asia, in amounts varying from three to six complete viral genomes per haploid cell genome. At least 20% of the DNA sequences in both mouse- and mink-grown MuLV probes were found to be of host cell origin. (34 refs)

78-4436 **Genetic Control of Sensitivity to Moloney-Virus-induced Leukemias in Mice. I. Demonstration of Multigenic Control.** (Eng) Gisselbrecht, S. (Laboratoire d'Immunologie et Virologie des Tumeurs-U.152, Hopital Cochin, Batiment G. Roussy, 27 rue du Faubourg-Saint-Jacques, 75674 Paris Cedex 14, France); Pozo, F.; Debre, P.; Hurot, M. A.; Lacombe, M. J.; Levy, J. P. *Int J Cancer* 21(5): 626-634; 1978.

The appearance of hematopoietic malignancies and viremia levels were studied in Moloney leukemia virus (MLV)-infected mice of nine inbred strains and their F_1 or F_2 hybrids. The mice were inoculated ip at age 3-4 wk with 0.1 ml of MLV at a 1:300 dilution, and they were surveyed for viremia at 3 wk and 2, 4, and 6 mo postinoculation by radioimmunoassay of the major internal virion component p30. The level of circulating virus was controlled by at least two genes, of which a histocompatibility (H-2)-linked gene (tentatively designated *Rmv-1*) displayed a dominant sensitivity. Alleles for resistance existed in the H-2b and H-2r haplotypes and alleles for sensitivity in H-2a, H-2d, and H-2f. Another gene with dominant resistance mapped outside the H-2 complex and probably interacted with *Rmv-1*. Although there was a good correlation between viremia and the appearance of leukemia, genes that were not involved in viremia control could be determinants in leukemia induction. One with a resistant allele in DBA-2 mice inhibited leukemia development despite high viremia levels. Another gene controlled the spleen involvement that results in generalized leukemias in sensitive lines, as opposed to the thymus-localized tumors in resistant animals. (38 refs)

78-4437 **Homology Between Cellular Repeated Nucleotide Sequences and a Murine Leukemia Viral Genome.** (Eng) Ginelli, E. (Istituto di Biologia Generale, Facolta di Medicina, Universita degli Studi, Via Viotti 5, 20133 Milan, Italy); Gianni, A. M.; Corneo, G. *Tumori* 64(4): 383-388; 1978.

Moloney murine leukemia virus complementary DNA was hybridized to an excess of NIH-Swiss mouse spleen DNA that had been fragmented to different lengths, heat-denatured, renatured (Cot = 50), and fractionated on an Ag^+/Cs_2SO_4 gradient according to extent of renaturation. The results confirm that the genome of an RNA oncogenic virus contains nucleotide sequences homologous to cellular repeated sequences. (12 refs)

78-4438 **Alterations in Chromatin of Cells Infected with RNA Tumor Viruses.** (Eng) Schincariol, A. L. (Cancer Res. Unit, Univ. Western Ontario, London, Ontario N6A 5B7, Canada); Maher, P.; Rip, J. *Nucleic Acids Res* 5(7): 2617-2627; 1978.

Differences in the chromatin (Chr) between normal and virus-transformed cell lines and among different virus-infected cell lines were determined. These lines were a cloned line of lymphocyte-like TB cells, derived from a culture of fetal thymus and bone marrow from CFW/D mice; producer clone 124, derived after successive infection of TB cells with the Moloney complex of murine leukemia/sarcoma virus [MuLV(MuSV)]; producer clone 349; nonproducer clone 605; and nontransformed producer clone TB+MuLV (produces MuLV, but not MuSV). Chr's were isolated from infected TB cells and compared immunologically and bic
chemically. Chr from virus-infected cells did not fi
complement as well as Chr from uninfected cells, suggestin
that conformational changes had occurred in the forme
This alteration was detected within 24 hr of infection. Th
structural changes in the Chr appeared to reflect alteratior
in the nonhistone proteins as the quantity and mol wt of th
histone proteins did not vary. The nonhistone proteins wer
synthesized in greater amounts in the infected cells, especiall
four proteins of mol wt 52,000, 60,000, 70,000, and 80,00
The 60,000-dalton protein is of interest, as cells infected wit
RNA tumor viruses synthesize a similarly sized polycistron
protein precursor containing viral structural polypeptides.
is not known whether this protein is related to the viral pr
cursor or whether it is involved in regulating gene transcri
tion. The conformational changes in Chr occurred not onl
in cells transformed and producing virus (clones 124 an
349), but also in clones 605 and TB+MuLV. The chang
are presumed due to the specific transcription of viral RN
and possible alterations in the transcription of cellular gene
(29 refs.)

78-4439 **The Discovery of a Murine Sarcoma Viru
Transforming and Nondefective for Replicatio**
(Meeting Abstract). (Fre) Sinoussi, F. (Unite d'Oncolog
Virale, Institut Pasteur, 25, rue du Dr Roux, 750
Paris, France); Chermann, J. C. *Bull Cancer (Par*
65(1): 35; 1978. (no refs)

78-4440 **Arrangement of Murine Leukemia Provira
Genes Integrated in Chromatin.** (Eng) Panet, A
(Dept. Virology, Hebrew Univ.-Hadassah Medical Sch
Jerusalem, Israel); Cedar, H. *In: Advances in Comparativ
Leukemia Research 1977. Proceedings of the VIIIth Interna
tional Symposium on Comparative Research on Leukemia an
Related Diseases held in Amsterdam, The Netherlands c
22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn D. S
eds. (New York: Elsevier/North Holland Biomedical Press
503 pp.; 446-447; 1978.

The expression of murine leukemia proviral DNA genes wa
studied at the chromatin level by measuring the sensitivit
of these genes to DNase I. Isolated nuclei of Swiss NI
mouse cells infected with Moloney murine leukemia viru
(MuLV) were treated with DNase. The results indicated tha
at least 80% of the proviral sequences in these cells wer
clustered in regions of chromatin sensitive to nuclease dige
tion. The localization of endogenous proviral sequences wa
also studied in the uninfected parental cells. A random distr
bution of the proviral sequences between DNase I-sensitiv
and -resistant portions of the nuclear DNA was noted. Sinc
the Moloney MuLV-infected cells contained as much endog
nous DNA as the uninfected cells, it is concluded that bot
exogenous and endogenous proviral genes are susceptible t

Nase I in these cells. This suggests that the presence of ogenous virus affects the arrangement of chromosome pro-ins along endogenous viral genes. Examination of the corre-tion between proviral gene activity, virus production, and ie disposition of proteins along the integrated proviral se-uences suggested that during the cell cycle, there is a rear-ngement of the chromosomal proteins along the integrated roviral genes and that these proteins are involved in the egulation of proviral gene expression. (no refs)

8-4441 **Genetic Control of Immune Responses to Moloney Leukemia Virus in Rats.** (Eng) Jones, J. 1. (Dept. Pathology, Natl. Center Toxicological Res., Jeffer-on, AR, 72079); Jensen, F.; Feldman, J. D. *J Natl Cancer nst* 60(6): 1467-1472; 1978.

he nature of the genetic differences that control responses o antigens of Moloney murine leukemia virus (M-MuLV) nd their relationship to the major histocompatibility (*AgB*) ocus were investigated in BN and LEW rats. The rats were mmunized with untreated or formalin-inactivated M-MuLV, and their sera were examined by radioimmunoas-ay for antibody to p15, p30, and gp70. BN rats produced igh antibody responses to the viral antigens, whereas LEW ats were low responders. BN rats also exhibited a high re-ponse and LEW rats a low response when the two strains vere immunized with purified p30. Studies of (LEW x BN)F$_1$ and backcross rats suggested that factors as-ociated with *AgB* exerted major influences on res-ponses to antigens of M-MuLV and that other factors vere also important. When other rat strains (F344, DA, ACI, AUG, and BUF) representing five *AgB* alleles vere tested, some were high and some were low res-ponders to M-MuLV, and responses to p15, p30, and gp70 were not always parallel. Since M-MuLV replica-ion was greater in cells of BN rats than in cells of LEW rats, replication of M-MuLV may have influenced the levels of responses to some viral antigens. Control of virus replication appeared to be due to cell mechanisms rather than to the environment of the host. (34 refs)

78-4442 **Effect of Rauscher Leukemia Virus-specific Proteins on Reverse Transcriptase: Binding Between Reverse Transcriptase and p30.** (Eng) Bandyopadhyay, A. K. (Section Immunology and Cell Biology, Lab. Molecular Biology, NIH, NCI, Baltimore Cancer Res. Center, Baltimore, MD 21211). *J Biol Chem* 252(16): 5883-5887; 1977.

The reverse transcriptase (RT) from Rauscher leukemia virus (R-LV) was purified to homogeneity. The RT, a single polypeptide chain of 70,000 mol wt, interacted with the major internal protein p30 of R-LV. The RT p30 complex stimulated the incorporation of ^{3}H-thymidine monophosphate into oligodeoxythimidylic acid·polyriboadenylic acid two to threefold compared to RT alone. Monospecific antisera directed against either p30 or RT precipitated the entire complex. The RT·p30 complex had a mol wt of approx 400,000 by chromatography on Sepharose 6B and a sedimentation rate of 12S in a glycerol gradient. (32 refs)

78-4443 **Detection of Membrane-bound Type-C Viral Antigens on Murine Spleen Cells by an Antibody-dependent Cell-mediated Cytotoxicity Assay.** (Eng) Becker, M. J. (Clinical Immunology and Arthritis Section, Veterans Admin. Hosp., 4150 Clement St., San Francisco, CA, 94121); Blackman, M.; Talal, N. *Ann Immunol (Paris)* 129(2/3): 179-181; 1978.

The utility of antibody-dependent cell-mediated cytotoxicity (ADCC) as a detection system for membrane-bound C-type viral antigens on murine lymphocytes and lymphatic tumor cells was studied. ADCC activity against NZB/NZW mouse lymphoma 141 was mediated by rabbit antisera to Rauscher leukemia virus proteins (gp69/71 or p30) and human lymphoid effector cells. At a 10^{-2} dilution of anti-gp69/71, 66.3% to 11.3% of the 141 cells were killed at effector to target cell ratios of 50:1 and 2:1, respectively. Significant lysis was demonstrated at a 10^{-4} dilution of antiserum and an effector to target cell ratio of 10:1. The specific lysis of lymphoma 141 and NZB/NZW and CBA/J splenic lymphocytes coated with rabbit anti-Rauscher gp69/71 was also assessed. At an effector to target ratio of 10:1, both lymphoma 141 and NZB/NZW lymphocytes were lysed to an equal extent. CBA/J lymphocytes were lysed to a significanty lower degree at all antiserum dilutions tested. Under similar conditions, rabbit anti-Rauscher p30 was also able to induce lysis of lymphoma 141 and NZB/NZW spleen cells, but not CBA/J spleen cells. It is concluded that ADCC is a specific and reproducible method for detecting C-type viral proteins on the surface of live normal and malignant murine lymphocytes. (24 refs)

78-4444 **Retarded Cleavage and Characterization of a Type C Virus Gag-Gene Coded Polyprotein in L929 Cells (Meeting Abstract).** (Eng) Schultz, A. (Frederick Cancer Res. Center, Frederick, MD, 21501); Oroszlan, S. *Fed Proc* 37(6): 1773; 1978. (no refs)

78-4445 **Restriction of Multiplication of Type-C Ecotropic Virus in Nondifferentiated Cells Derived from Mouse Teratocarcinoma (Meeting Abstract).** (Fre) Canivet, M. (INSERM, 75010 Paris, France); Peries, J.; Debons-Guillemin, M. C.; D'Auriol, L.; Emanoil-Ravicovitch, R.; Tavitian, A.; Sherr, C. J. *Bull Cancer (Paris)* 65(1): 33; 1978. (no refs)

78-4446 **Virus-Specific Receptors for Murine Leukemia Viruses on Murine Thymocytes (Meeting Abstract).** (Eng) Bednarz-Prashad, A. J. (Univ. of Texas Medical Sch., Houston, TX, 77030). *Fed Proc* 37(6): 1747; 1978. (no refs)

78-4447 **The Influence of Rauscher Leukemia Virus on the Hemapoietic System of BALB/c and DBA Mice. The Establishment of a New Erythroid Cell Line.** (Eng) de Both, N. J. (Dept. Experimental Pathology, Faculty Medicine, Erasmus Univ., Rotterdam, Netherlands); Vermey, M.; Klootwijk, E.; van't Hull, E.; Montfoort, A.; van Griensven, L. J.; Stoof, T. J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 288-290; 1978.

Five days after BALB/c mice are infected with Rauscher murine leukemia virus (R-MuLV), islands of large immature blast cells arise and proliferate in the red pulp of the spleen with a doubling time of 7.5 days. About 96% of these cells are lost from the spleen, mostly through cell death. Studies indicated that erythropoiesis took place in the spleen and that the disease-associated progressive anemia appeared to be caused by a defect in peripheral RBC function. No permanent transformed lines could be obtained from BALB/c spleens, but one was established from R-MuLV-infected DBA mice. (8 refs)

78-4448 **Cross-Linking and Co-Capping of Leukemia Virus Envelope Glycoprotein and H-2 Antigens on Leukemia Cell Surfaces and Their Recognition as Target Antigens by Syngeneic Virus-Immune Cytotoxic T Lymphocytes (Meeting Abstract).** (Eng) Zarling, D. A. (Univ. Wisconsin, Madison, WI, 53706); Duke, R. E.; Watson, A.; Bach, F. H. *Fed Proc* 37(6): 1570; 1978. (no refs)

78-4449 **Morphological Studies of Oncornaviruslike Particles from Human Haematological Malignancies.** (Eng) Mooren, H. (Lab. Pathology, State Univ. Leiden, Wassenaarseweg 62, 2300 RC Leiden, Netherlands); Warnaar, S.; Prins, F. *In: Advances in Comparative Leukemi Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Relate Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D.S., eds. (Ne York: Elsevier/North Holland Biomedical Press): 503 pp 154-155; 1978.

Viruslike particles were detected by electron microscopy i three tumors from patients with hematological malignanci (non-Hodgkin's lymphoma, myelofibrosis, and hairy ce leukemia). Computer analyses of the rotational symmetri of the particles showed that they had the same levels of tw and threefold symmetries as murine mammary tumor viru type B, and Rauscher murine leukemia virus, type C. The results strengthened the biochemical evidence (reverse tra scriptase and 70S RNA) for the presence of oncornavirus in the three tumors. (4 refs)

78-4450 **The Effect of Pseudotype on Abelso Virus-induced Leukemia.** (Eng) Scher, C. (Sidney Farber Cancer Inst., Harvard Medical Sch., Bosto MA, 02115); Stone, M. *In: Advances in Comparati Leukemia Research 1977. Proceedings of the VIII International Symposium on Comparative Research Leukemia and Related Diseases, held in Amsterdam, T Netherlands on 22-26 August, 1977.* Bentvelzen, Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/Nor Holland Biomedical Press): 503 pp.; 318-319; 197

Nonproducer Abelson murine leukemia virus (A-MuLV) w infected with NB-tropic Moloney MuLV, NB-tropic Rausc er MuLV, NB-tropic Friend MuLV, or N-tropic endogeno helper virus to prepare pseudotypes of A-MuLV. The help virus profoundly influenced the frequency of A-MuLV-i duced leukemia in vivo (Fv-1-nn CD-1 Swiss mice) and t frequency of C57/L mouse bone marrow cell transformati in vitro. This effect is not controlled by the murine Fv locus. The pseudotype of A-MuLV, rather than the presen of an added effective helper, controls transformation freque cy. (3 refs)

78-4451 **Purification and Characterization of DN Polymerases from the Plasmacytoma MOP 104E and Abelson Murine Leukemia Viruses.** (Eng) W mann, B. J. (Basel Institut fur Immunologie, Grenzache strasse 487, Postfach, CH-4005 Basel, Switzerland); Schmi J.; Takacs, B. *Eur J Biochem* 85(2): 571-579; 1978.

RNA-dependent DNA polymerases were isolated from t intracisternal A particles of plasmacytoma MOPC 104E a from Abelson murine leukemia viruses and characterize Both polymerases were similar in their elution patterns fro phospho- and diethylaminoethyl-cellulose, template speci cities, requirements for optimum activity, and inactivation by anti-(reverse transcriptase) antiserum. Both polymerases were associated with ribonuclease H activity. For mol wt determinations, antibody-precipitated enzymes were bound to staphylococcal-protein A-Sepharose, solubilized, and run on dodecyl sulfate/polyacrylamide gels. Their apparent mol wt was estimated to be 80,000. (35 refs)

78-4452 **Use of Trypsin for Purification of Murine Sarcoma and Leukemia Virus (MSV-MuLV) from Supernatants of the Culture of 78 A_1 Cells (Meeting Abstract).** (Fre) Rugheton, M. (Laboratoire de Biochimie, Centre Paul Lamarque, B. P. 5054, 34033 Montpellier Cedex, France); Blaas, D.; Jeanteur, P. *Bull Cancer (Paris)* 65(1): 39; 1978. (no refs)

78-4453 **Amino Acid Analog Induction or Inhibition of Type C Virus Expression (Meeting Abstract).** (Eng) Suk, W. A. (Frederick Cancer Res. Center, Frederick, MD, 21501); Long, C. W. *Fed Proc* 37(6): 1745; 1978. (no refs)

78-4454 **Biological Properties of a Murine Sarcoma Virus Transforming and Nondefective for Replication (Meeting Abstract).** (Fre) Chermann, J. C. (Unite d'Oncologie Virale, Institut Pasteur, 25, rue du Dr Roux, 75015 Paris, France); Sinoussi, F.; Lidereau, R. *Bull Cancer (Paris)* 65(1): 35-36; 1978. (no refs)

78-4455 **Isolation of a Leukaemogenic Type-C Oncovirus from a Leukaemic Child.** (Eng) Nooter, K. (Radiobiological Inst. TNO, Lange Kleiweg 151, Rijswijk, Netherlands); Overdevest, J.; Dubbes, R.; Koch, G.; Bentvelzen, P.; Coolen, J.; Zurcher, C.; Calafat, J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 471-474; 1978.

A C-type viral isolate from a leukemic child was characterized. Bone marrow cells from the child were cocultivated with A7573 canine cells. In early passages, C-type oncoviruses were released. Cocultivation of the infected canine cells with nonproducing K-NRK rat cells transformed by Kirsten murine sarcoma virus (MSV) produced a new MSV phenotype that could transform rabbit SIRC cells. A cell line (SKA21-3) derived from the transformed SIRC cells became a high virus producer. The virus proteins were closely related to those of simian sarcoma virus. Injection of SKA21-3 cells producing human helper virus pseudotypes of MSV plus human helper virus into rats induced sarcomas, erythroleukemias, and lymphosarcomas. When the MSV genome was eliminated from SKA21-3 virus, the resulting human-derived virus induced generalized lymphosarcomas in rats. The leukemogenicity of this virus in animals is suggestive of an etiological role for the virus in human disease. (11 refs)

78-4456 **Origin and Biological Properties of a New BALB/c Mouse Sarcoma Virus.** (Eng) Aaronson, S. A. (Lab. RNA Tumor Viruses, NCI, Bethesda, MD, 20014); Barbacid, M. *J Virol* 27(2): 366-373; 1978.

A focus-forming virus previously isolated from a BALB/c mouse hemangiosarcoma has been shown to be replication defective. Analysis of individual BALB/c mouse sarcoma virus (BALB-MSV) nonproducer transformants for expression of helper virus-coded proteins revealed genetically stable variants that expressed 2, 3, or all 4 *gag* gene products in the absence of detectable helper viral *env* gene expression. The type-specific antigenic determinants of helper viral proteins encoded by the BALB-MSV genome and by the B-tropic virus isolated from the BALB-MSV stock were demonstrated to be indistinguishable from those of BALB:virus-1, a known endogenous virus of BALB/c cells. These findings imply that a BALB/c endogenous virus was involved in the generation of BALB-MSV. By the same immunological approach, the presence of at least a portion of the Moloney murine leukemia virus *gag* gene has been identified in two other transforming viruses--Moloney-MSV and Abelson lymphosarcoma virus---previously isolated from the BALB/c strain. The tissue culture properties of cells transformed by these defective viruses were also shown to be distinguishable. These findings indicate that transforming virus isolates of the same inbred strain differ in their transforming activities as well as in the helper viral sequences stably associated with their genomes. (36 refs.)

78-4457 **Activation of Endogenous Type C Virus by Amino Acid Alcohols.** (Eng) Long, C. W. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD, 21501); Suk, W. A.; Greenawalt, C. *Virology* 88(1): 194-196; 1978.

Amino acid alcohols were examined for their ability to inhibit protein synthesis and induce endogenous C-type virus from Kirsten sarcoma virus (KiSV)-transformed Balb/c (K-Balb) mouse cells. Histidinol and tyrosinol proved to be highly effective short-term virus activators; valinol and methioninol gave low but reproducible activation. Histidinol, tyrosinol, and valinol were the most effective inhibitors of protein synthesis in this cell system, each reducing ^{3}H-leucine incorporation by > 95%. Methioninol reduced incorporation by 80%, and the other alcohols produced marginal effects. Treatment of K-Balb cells with amino acid alcohols did not induce virus capable of forming foci on Balb/c or NIH swiss monolayers. Decay of virus expression following induction with either histidinol or tyrosinol was rapid, and 24 hr after removal of histidinol, ^{3}H-leucine incorporation had returned to normal levels. Histidinol- or tyrosinol-induced virus activation was completely inhibited by actinomycin D or cytosine arabinoside, showing a requirement for RNA and DNA synthesis during induction. The fact that valinol efficiently blocks protein synthesis without appreciable virus activation indicates that a secondary process may be necessary for induction other than inhibition of protein synthesis. (10 refs.)

78-4458 **3T3 Variants Lacking Receptors for Epidermal Growth Factor Are Susceptible to Transformation by Kirsten Sarcoma Virus.** (Eng) Pruss, R. M. (Dept. Biological Chemistry, UCLA Sch. Medicine, Los Angeles, CA, 90024); Herschman, H. R.; Klement, V. *Nature* 274(5668): 272-274; 1978.

The susceptibility of variant Swiss 3T3 cells lacking receptors for epidermal growth factor (EGF) to transformation by Kirsten sarcoma virus was studied. The Swiss 3T3 parental cell line and three variants, NR1, NR4, and NR6, were essentially equivalent targets for transformation by Kirsten sarcoma virus, as determined by the number of foci produced. Assays for nuclear EGF receptors in preparations of membrane fragments from NR6 cells showed no membrane-associated EGF receptors; these receptors were demonstrated on parental 3T3 cells. Another assay for inactive substances that might block surface EGF receptors or bind to and/or degrade externally provided EGF showed no such inhibitory activity in extracts of either parental 3T3 or NR6 cells. The strictest interpretation of the data suggests that stimulation of mitogenesis by the action of a sarcoma gene product on EGF receptors is not the basis for uncontrolled growth as a consequence of transformation by murine sarcoma viruses. (7 refs)

78-4459 **FBJ Osteosarcoma Virus in Tissue Culture. III. Isolation and Characterization of Non-Virus-producing FBJ-transformed Cells.** (Eng) Levy, J. A. (Cancer Res. Inst., Univ. California, San Francisco, CA, 94143); Kazan, P. L.; Reilly, C. A.; Finkel, M. P. *J Virol* 26(1): 11-15; 1978.

The establishment and characterization of hamster and rat lines that have been transformed by FBJ murine sarcoma virus (FBJ-MuSV) but that do not produce virus are reported. The hamster line originated from an osteosarcoma that appeared in a Syrian hamster inoculated at birth with an extract of a CF 1 mouse FBJ osteosarcoma. The rat cell line was obtained by transferring the FBJ-MuSV genome to nor-
mal rat kidney cells in the absence of FBJ C-type virus (FB
murine leukemia virus), which usually accompanies FB
MuSV in high concentration. Both transformed hamster an
rat cell lines contained the FBJ-MuSV genome, and th
genome could be rescued by ecotropic and xenotropic murir
C-type viruses. The rescued genome produced characterist
FBJ-MuSV foci in tissue culture and, in the appropria
hosts, induced osteosarcomas typical of those induced b
FBJ-MuSV. These lines should be helpful in molecular st
dies of the origin of spontaneous sarcoma genomes in mic
(17 refs)

78-4460 **Characterization of Virus-like Particle
Released from the Hamster Cell Line CHO-K
after Treatment with 5-Bromodeoxyuridine.** (Eng) Manly, K
F. (Dept. Medical Viral Oncology, Roswell Park Memoria
Inst., Buffalo, NY, 14263); Givens, J. F.; Taber, R. L.; Zeige
R. F. *J Gen Virol* 39(3): 505-517; 1978.

The release of C-type viruslike particles from Chinese ham
ster ovary (CHO) K1 cells treated with 20 μg/ml of steri
5-bromodeoxyuridine (BUdR) was investigated. Few part
cles that had RNA, an associated DNA polymerase, and th
density typical of RNA tumor viruses were released by th
BUdR-induced cells. However, some immature C-type form
were observed by electron microscopy in partially purifie
preparations of DNA polymerase-containing particles fron
induced CHO cells. The max production of particle-boun
polymerase by BUdR-treated CHO cells was roughly 1% c
the production by a leukemia virus-infected mouse cell line
Similarly, uridine labeling suggested that the release c
RNA-containing particles was less than 1% of that from
similarly induced mouse cell line. It is concluded that simpl
biochemical measurements seriously overestimate the pro
duction of C-type particles by BUdR-induced CHO cells. (1
refs)

78-4461 **Induction of Type C RNA Virus from Culture
Rabbit Lymphosarcoma Cells.** (Eng) Bedigia
H. G. (Jackson Lab., Bar Harbor, ME, 04609); Fox, R. R
Meier, H. *J Virol* 27(2): 313-319; 1978.

C-type RNA virus(es) was readily released from prima
lymphosarcoma cell cultures of WH/J rabbits after inductic
with halogenated pyrimidines. The virus contained a
RNA-directed DNA polymerase and the p30 structural pr
tein. The rabbit virus RNA-directed DNA polymerase an
p30 protein shared antigenic homologies with other man
malian C-type oncornaviruses, but they also appeared to po
sess unique antigenic determinants. Normal rabbit liver co
tained DNA homologous to a ^{3}H-labeled complementa
DNA transcript of the rabbit viral genome, indicating th
C-type viral genetic information is present in at least th
WH/J strain of rabbits. (26 refs.)

78-4462 **The Influence of Virus Subgroup on the Epidemiology of Feline Leukemia Virus.** (Eng) Jarrett, O. (Dept. Veterinary Pathology, Univ. Glasgow, Glasgow, Scotland); Russell, P. H.; Hardy, W. D. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 25-28; 1978.

One hundred and forty-nine isolates of feline leukemia virus (FeLV) were analyzed for subgroup composition. FeLV occurred in three subgroups: FeLV-A present in every isolate, FeLV-B was common but was always associated with FeLV-A (FeLV-AB); and FeLV-C was rare and was found with FeLV-A (FeLV-AC) or with FeLV-AB (FeLV-ACB). FeLV-B was more common in cats with leukemia than in carrier cats, but the reverse was true for FeLV-A. However, there was no association between FeLV subgroup and any particular clinicopathological type of lymphosarcoma or leukemia. Among healthy cats from multicat households (MHC) yielding FeLV-AB, half of the cats yielded only FeLV-A. In MHC yielding FeLV-A, 28% of the cats were viremic, whereas in MHC yielding FeLV-AB, 53% were viremic. All neonatal cats infected by ip inoculation with 10^4 infectious units of FeLV-A developed FeLV-A viremia in about 10 days, and the viremia was persistent. Only 25% of neonatal cats infected with FeLV-B became viremic, and virus was not detectable until 6 to 12 mo after infection. The data suggest that the growth of FeLV-B in cats is restricted and that it is not transmissible by contact. When cats were infected simultaneously with FeLV-A and FeLV-B, the viruses acted independently to some extent, although the results indicated that the viruses may also have interacted. FeLV-A appears to enhance the growth of FeLV-B in cats and to be necessary for the transmission of FeLV-B. The preponderance of FeLV-AB over FeLV-A in MHC and in leukemic cats compared with carrier cats is probably due to infection with a high dose of virus and to a more long-standing infection. (5 refs.)

78-4463 **Evidence for the Possible Functional Involvement of Endogenous Type C Viral Gene Expression in Natural Malignancies of Domestic Cats.** (Eng) Roy-Burman, P. (Univ. Southern California Sch. Medicine, Los Angeles, CA, 90033); Niman, H. L.; Stephenson, J. R.; Gardner, M. B. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 452-455; 1978.

The role of C-type endogenous feline viruses in natural malignancies of the cat was studied by immunofluorescence using 60 histologically normal, 55 lymphoma, 9 sarcoma, and 14 carcinoma tissue specimens. A significantly higher level of RD-114 virus transcription and translation was noted in the lymphoma, sarcoma, and carcinoma tissue, compared with normal tissue. Lymphoma tissue from younger cats had significant levels of feline leukemia virus (FeLV) gene expression, but RD-114 virus expression was high in lymphomas of both young and old cats. Feline embryonal placenta and thymus also expressed viral genes. Although both RNA and p30 viral gene expressions were detected in the placenta, the marked elevation of RD-114 gene transcription in the thymus resembled the expression in most of the adult lymphoma tissues. The widespread occurrence of elevated levels of RD-114 virus-specific gene expression in the neoplastic tissues, independent of FeLV genome expression, suggests that the expression of certain functions of RD-114 virus may be involved in the development of spontaneous neoplasms in the domestic cat. (4 refs)

78-4464 **Transfection with Endogenous RD-114 Provirus of Cat Cells.** (Eng) Nicolson, M. O. (Univ. Southern California Sch. Medicine, Childrens Hosp., Los Angeles, CA); McAllister, R. M.; Okabe, H.; Gilden, R. V. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 139-140; 1978.

The transfection of human rhabdomyosarcoma TE-32 cells and dog D-17 sarcoma cells by endogenous provirus in cat cell DNA was examined. Only the DNA's of cells releasing RD-114 virus were infective for the human and dog cells, with minimum infectious doses ranging from 0.5 to 10 μg/ml. DNA from nonproducer cat fibroblasts and tissues did not transfect either cell line at doses up to 100 times the minimum infectious DNA dose. In virus-producing F100 osteosarcoma cells, proviral infectivity was covalently integrated in cell chromosomal DNA. All the cat cells contained 20 viral gene copies per haploid genome, which suggests that RD-114 provirus in productive cells differs in some qualitative characteristic from that in nonproductive cells. (5 refs)

78-4465 **RNA Tumor Virus Phosphoproteins: Subvirion Location of the Multiple Phosphorylated Species.** (Eng) Pal, B. K. (Dept. Pathology, Univ. Southern California Sch. Medicine, Los Angeles, CA, 90033); Roy-Burman, P. *Virology* 83(2): 423-427; 1977.

The structural location of the multiple phosphorylated

15,000-mol wt protein (pp15) of the endogenous RD-114 feline C-type virus was investigated. RD-114 cores were prepared by removal of the envelope components; the cores banded at a density of 1.23 g/cm^3. Guanidine-agarose chromatography showed that pp15 was present in the core structure. The data also indicated the presence of p30, p12, and p10 and protein kinase activity in the core preparation. A comparison of the content of multiple phosphorylated species of the pp15 derived from the core with that isolated from the whole virion demonstrated that all major phosphorylated species of RD-114 pp15 were located in the core structure. The finding of both protein kinase activity and the multiple phosphorylated protein species in the virus core suggests that biologically significant interactions occur between the enzyme and the core structural proteins. (24 refs)

78-4466 Endogenous Type C RNA Virus of Mink (*Mustela vison*). (Eng) Klement, V. (Dept. Pediatrics and Microbiology, Univ. Southern California Sch. Medicine, 1840 North Soto St., Los Angeles, CA, 90032); Dougherty, M. F.; Roy-Burman, P.; Pal, B. K.; Shimizu, C. S.; Rongey, R. W.; Nelson-Rees, W.; Huebner, R. J. *Virology* 85(1): 296-306; 1978.

The isolation and basic biological and biochemical characteristics of a new C-type RNA virus are described. The virus was isolated from a mink lung cell line (CCL64) that had been cocultivated with 5-bromodeoxyuridine-treated mouse spleen cells. The virus sedimented in sucrose density gradients at 1.13-1.17 g/ml, and the virus nucleocapsids sedimented at 1.19 g/ml. Immunofluorescence tests distinguished the new virus from murine C-type virus, and complement fixation tests distinguished it from the C-type viruses of rat, cat, mouse, baboon, and woolly monkey. The mink virus did not replicate in simian or bovine cells, but it did propagate in mouse, rat, cat, lamb, dog, and human cells. Two major species of RNA were identified: one species banded near the 70S region in sodium dodecyl sulfate-sucrose gradients, and the second species banded at about 4S. The protein profile was similar to that observed for other mammalian C-type viruses. The DNA complementary to the poly(A)-containing virion RNA hybridized about 72% with the RNA from virus-producing mink lung cells but not with the RNA from mouse cell lines or uninfected mink lung cells. The nucleotide sequences homologous to mink viral complementary DNA were found in mink cell DNA from both virus-producing and nonproducing cells, but not in mouse, rat, or feline DNA. The data indicate that the new virus is an endogenous mink C-type virus, although its biological significance in the diploid mink genotype remains unknown. (33 refs)

78-4467 **Detection of C-Type Virus in Immunologically Reactive Ovine Lymph Nodes.** (Eng) Atkins, G. J. (Dept. Microbiology, Trinity Coll., Dublin 2, Ireland); Moynihan, M.; Arbuthnott, E.; Hartigan, P. J. *FEMS M* *crobiol Lett* 2(2): 75-77; 1977.

Electron microscopy of a reactive lymph node from a shee with a scab mite (*Psoroptes communis*) infection revealed th presence of C-type virus particles. To confirm this observation, lymph nodes from this and two other infested sheep an from five normal sheep were studied for reverse transcriptas activity. This activity was consistently observed in the lymp node culture fluids from 2/3 mite-infested sheep; no activit was observed in the culture fluids of lymph nodes from fiv normal sheep. In the infested sheep, reverse transcriptase activity was low compared with the activity in fluids from cell producing murine sarcoma/leukemia virus, but it approached the level observed in cells producing bovine leukemia. This virus could either be exogenous or endogenous and activated by a persistent immune respons (8 refs)

78-4468 **Susceptibility of Cattle to Bovine Leukemi Virus Infection by Various Routes of Exposur** (Eng) Van der Maaten, M. J. (P. O. Box 70, United State Dept. Agriculture, Agricultural Res. Service, Natl. Anima Disease Center, Ames, IA, 50010); Miller, J. M. *In: Advance in Comparative Leukemia Research 1977. Proceedings of th VIIIth International Symposium on Comparative Research o Leukemia and Related Diseases, held in Amsterdam, Th Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilger J.; Yohn, D. S., eds. (New York: Elsevier/North Hollan Biomedical Press): 503 pp.; 29-32; 1978.

The routes by which bovine leukemia virus (BLV) is mos likely to gain access to susceptible tissues and initiate ne infections were studied in BLV-negative calves and cows. A of six newborn calves exposed to BLV po developed persiste infections, compared with only one of two 3-day-old calv and neither of two 3-wk-old calves exposed in the same manner. Both 3-mo-old calves exposed to an aerosol of cell-fre BLV developed infections, but only one of two 2- to 3-mo-ol calves exposed by the intranasal instillation of infected WB developed an infection. Four of six mature cows exposed t BLV by the instillation of infected WBC into their reproductive tract developed antibody responses indicative of infection. All five calves inoculated id with infected periphera blood WBC developed BLV infections, as did all three calve inoculated id with cell-free material. The results suggest tha the skin may play an important role in the horizontal transmission of BLV infections. (6 refs.)

78-4469 **Natural Mode of Transmission of the Bovin Leukemia Virus: Role of Bloodsucking Insect** (Eng) Bech-Nielsen, S. (Dept. Epidemiology and Communit Health, Sch. Veterinary Medicine, Louisiana State Univ., Ba ton Rouge, LA, 70803); Piper, C. E.; Ferrer, J. F. *Am J Vet Res* 39(7): 1089-1092; 1978.

The development of bovine leukemia virus (BLV) infection was studied in 14 noninfected young adult cattle exposed to 25 to 30 BLV-infected cows in a 0.5-hectare area. Of seven cattle exposed between July and August, four showed signs of BLV infection by October, and the remaining three were infected by November. All seven cattle exposed in February remained negative for at least 3 mo, and only one was positive after 6 mo exposure. However, four of the cattle were infected by October. Approx 1.8 x 10^6 BLV-infected lymphocytes (50% viability) were recovered from the midgut of at least 35 engorged tabanids that had been allowed to feed on a BLV-infected cow. Numerous syncytia were observed in indicator bovine embryonic spleen cells inoculated with these lymphocytes or with lymphocytes obtained directly from the infected cow. The data support the contention that bloodsucking insects play a major role in the spread of BLV. (28 refs.)

78-4470 **Etiologic Association of Bovine Leukemia Virus with Lymphosarcoma of Domestic Cattle.** (Eng) Devare, S. G. (NCI, NIH, Bethesda, MD, 20014); Stephenson, J. R. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 231-232; 1978.

Radioimmunoassays were developed for detecting antibody directed against the major 24,000-dalton structural protein (p24) and the 51,000-dalton envelope glycoprotein (gp51) of bovine leukemia virus (BLV). These radioimmunoassays, in which ^{125}I-labeled p24 and gp51 are used, are much more sensitive and reliable for the detection of BLV infection in domestic cattle than previously described serologic techniques. Sera from 100% of adult cattle tested with clinically diagnosed lymphosarcoma exhibited high-titered antibody against both p24 and gp51. In contrast, sera from cattle in a BLV-negative herd lacked detectable antibody to BLV. Approx 6,000 animals from 22 states were tested for antibodies to ^{125}I labeled BLV p24. The frequency of animals exhibiting high titers of p24 antibody varied from a low of 2% to $>40\%$ in certain states. (3 refs)

78-4471 **Direct Syncytial Assay for the Quantitation of Bovine Leukemia Virus.** (Eng) Benton, C. V. (Viral Oncology Program, Natl. Cancer Inst. Frederick Cancer Res. Center, Frederick, MD, 21501); Soria, A. E.; Gilden, R. V. *Infect Immun* 20(1): 307-309; 1978.

A direct quantitative bioassay for bovine leukemia virus (BLV) was developed based on cell fusion following BLV infection of F81 cells, a feline sarcoma-positive leukemia-negative (S+L-) cell line. F81 cells were incubated for 24 hr and BLV was added to the incubation mixture. After 16 days of infection, the plates were fixed and stained with methylene blue and carbol fuchsin. Using this assay system, BLV infection resulted in large, multinucleated syncytia that retained less stain than the surrounding monolayer. These fused cells were distributed evenly throughout the monolayer. Titration of the virus resulted in a reproducible linear decrease in syncytia that was directly proportional to the virus dilution, consistent with a one-hit response. The assay was specific for BLV. Viruses from several subprimate [Rauscher murine leukemia virus (MuLV), mink cell focus-inducing MuLV, and mouse mammary tumor virus] and primate (baboon leukemia virus and Mason-Pfizer monkey virus) species were tested, but they did not produce a morphological change similar to that seen with BLV. (18 refs)

78-4472 **Physico-chemical and Biological Properties of Viruses Isolated from Cultures of Primary Human Tumors.** (Rus) Shevlyagin, V. Ya. (N. F. Gamaleia Inst. Epidemiology and Microbiology, Moscow, USSR); Klenova, A. V.; Borodina, N. P.; Chizhevskaia, V. I.; Voskoboinik, A. D.; Eremina, L. A. *Vestn Akad Nauk SSSR* (5): 73-78; 1978.

Viruses isolated from a human embyro fibroblast culture transformed by a cell-free extract of human malignant paranglioma cells (transformed culture 63) were identified. Incubation of the culture with ^{3}H-uridine or ^{3}H-thymidine and subsequent centrifugation in a sucrose gradient showed the presence of DNA at a density of 1.23 g/ml and RNA at a density of 1.16-1.8 g/ml. The RNA-containing virus showed no reverse transcriptase or hemagglutinating activity. The DNA-containing virus showed hemagglutinating activity with guinea pig RBC and human blood group O RBC. Electron microscopically, the RNA-containing virus was classified as an orthomyxovirus, the DNA-containing virus as a papovavirus. Incubation of a human embryo brain cell culture with a cell-free extract of transformed culture 63 induced cytolytic changes in 19/74 experiments: in 17 experiments, these changes disappeared with culturing (abortive transformation), but in 2 experiments the changes became stable. (15 refs)

78-4473 **Inverted Repetitive Sequences in Human Papilloma Virus 1 (HPV-1) DNA.** (Eng) Gissmann, L. (Zentrum fur Hygiene, Hermann-Herder-Strasse 11, 78 Freiburg im Breisgau, W. Germany); zur Hausen, H. *Virology* 83(2): 271-276; 1977.

The identification by electron microscopy of two pairs of inverted repetitive (palindromic) sequences in the human papilloma virus 1 (HPV-1) genome is reported. The two sequences were designated IR 1a/b and IR 2a/b, respectively. The distance between IR 1a and IR 1b corresponded to 0.8 x 10^6 in mol wt, but IR 2a and IR 2b were separated by a DNA stretch of 0.4 x 10^6. The positions of the sequences in the HPV-1 genome were mapped by examining the DNA cleaved by *Bam*HI or *Eco*RI endonucleases. The mol wts of these sequences were determined to be 0.11 and 0.10 x 10^6, but it was not determined which value corresponded to IR 1a/b and which corresponded to IR 2a/b. (13 refs)

78-4474 **Search for Evidence of Papovavirus Involvement in Human Cancer.** (Eng) Takemoto, K. K. (Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD): Solomon, D.; Israel, M.; Howley, P. M.; Khoury, G.; Aaronson, S. A.; Martin, M. A. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Disease, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York Elsevier/North Holland Biomedical Press): 503 pp.; 260-263; 1978.

The involvement of BKV and JCV papovaviruses in human neoplasia was examined using (1) tests for the presence of intranuclear T antigen in tumor cell lines, (2) tests of the serum from cancer patients for T antibody, and (3) analyses of tumor cell DNA for viral sequences. Fifty-five different tumor lines, including those derived from the brain and kidney, were tested for the presence of T antigens against BKV and JCV. All were negative for T antigen. Sera from patients with kidney, brain, bladder, and esophageal tumors were tested for T-antibody to BKV and JCV, and all were found to be negative. Fifteen tumor cell lines that were negative for T antigens were also examined for BKV DNA sequences; all tumor lines were negative for BKV sequences. Analyses with JCV DNA were not performed because of the lack of suitable cell systems for growing JCV. If there is a relationship between BKV or JCV and human cancer, the mechanism of tumor induction does not conform to known models of papovavirus-induced transformation. (9 refs)

78-4475 **Virus-Specific Early RNA in 3T6 Cells Infected by a tsA Mutant of Polyoma Virus.** (Eng) Cogen, B. (Tumor Virology Lab., Salk Inst., San Diego, CA, 92112). *Virology* 85(1): 222-230; 1978.

The transcription of stable early polyoma-specific cytoplasmic RNA in mouse 3T6 cultures infected by wild-type polyoma virus or by a polyoma tsA mutant (tsA25E) was studied. 3T6 cultures infected by the mutant accumulated approx 20 times more virus-specific early RNA than did cultures infected with wild-type virus at both 32 and 39 C. This abnormal accumulation was observed when the infection was confined to the early stage by treatment of the infected cultures with cytosine arabinoside to block viral DNA replication. The abnormal accumulation was also observed 40 to 60 hr postinfection, and virus-specific early RNA continued to accumulate in the tsA25E mutant-infected cultures that had been shifted from the permissive to the nonpermissive temperature late in infection to block viral DNA replication. Mixed infection of 3T6 cultures by wild-type virus and tsA25E did not result in an enhanced accumulation of virus-specific early cytoplasmic RNA. The results suggest that the A gene product of polyoma regulates the transcription of early RNA and that the wild-type A-gene product overcomes the effect of the temperature-sensitive A-gene product. (34 refs)

78-4476 **Induction of Peripheral Neuroblastomas in Syrian Hamsters after Injection as Neonate with JC Virus, a Human Polyoma Virus.** (Eng) Varakis J. (Dept. Pathology, Univ. Southern Alabama, Mobile AL, 36688); ZuRhein, G. M.; Padgett, B. L.; Walker D. L. *Cancer Res* 38(6): 1718-1722; 1978.

Newborn Syrian hamsters were inoculated intraocularly with partially purified JC virus (0.02 ml of viral material containing 5,120 hemagglutinating units), a human polyoma virus, and tumor induction was studied. Neuroblastomas developed in 10/31 hamsters after a latent period of 6-11 mo. No tumors were noted in controls. Primary tumors were noted in the abdominal cavity (6), pelvis (1), and neck and mediastinum (3). One tumor arose from the adrenal gland. Metastases to the liver, bone marrow, and lymph nodes were observed. Two of the neuroblastomas were transplanted serially in weanling hamsters, and a tissue culture line (HJCN-1) was established from one of the transplanted tumors. T antigen was detected in 3/5 primary tumors tested and in the transplanted tumors. Antibody against T antigen was detected in the sera of 5/6 animals with neuroblastomas. A neuroblastoma was also noted in an animal inoculated sc and ip with JC virus; it was transplanted serially five times in weanling hamsters. Some animals given sc inoculations of this neuroblastoma exhibited visceral and lymph node metastases. (12 refs)

78-4477 **Effects of BK Virus Infection on Primary Cultures of Rodent and Primate Cells.** (Eng) Greenlee, J. E. (Dept. Neurology, Univ. Virginia Medical Center Charlottesville, VA, 22901); Narayan, O.; Johnson, R. T *Proc Soc Exp Biol Med* 158(3): 437-441; 1978.

The effect of BK virus (BKV) at high multiplicity was studied on cultures of hamster, rat, monkey, and human cells. BKV produced multiple transforming events in rodent cells without evidence of replication of infectious virus. Rhesus monkey choroid plexus cells were not susceptible to the virus. Of the three human cell types studied, colloid cyst cells and human embryo kidney, both epithelial cell types, developed lytic infection without evidence of cell transformation. Fibroblastic human fetal skin and muscle cells also developed lytic infection. Although 15%-20% of surviving cells showed T antigen, other criteria for transformation were not met. Approx 90%-100% of transformed rodent cells exhibited T antigen. BKV could be recovered only from rat cells and not from hamster cells. Interestingly, rat embryo cells are among the few cell types from which recovery of infectious virus has been reported after transformation by polyoma virus. BKV-transformed rat cells could not be transplanted, and transformed hamster cells were poorly oncogenic for hamsters. Immunosuppression was required for tumor production in newborns, and inoculation into the cheek pouch, an immunologically privileged site, was required in adults. These data support the hypothesis that the poor oncogenicity of BKV in vivo may represent at least in part, an immunological recognition and rejection of transformed cells by the host. (21 refs.)

78-4478 **Metastatic Properties, Immunogenicity and Cell Surface Sialylation of Tumor Cell Lines Derived from a Polyoma-induced Rat Renal Sarcoma (Meeting Abstract).** (Eng) Salk, P. (Salk Inst. Biological Studies, San Diego, CA, 92112); Yogeeswaran, G. *Fed Proc* 37(6): 1760; 1978. (no refs)

78-4479 **Polyoma Virus Has Three Late mRNA's: One for Each Virion Protein.** (Eng) Siddell, S. G. (Institut fur Virologie und Immunobiologie der Universitat Wurzburg, D8700 Wurzburg, W. Germany); Smith, A. E. *J Virol* 27(2): 427-431; 1978.

Polyoma virus messenger RNA (mRNA), isolated from the cytoplasm of 3T6 cells late after infection and purified by hybridization to *Hpa*II fragment 3 of polyoma virus DNA, was separated on 50% formamide-containing sucrose density gradients, and the fractionated RNA was recovered and translated in vitro. Analysis of the cell-free products showed that the minor virion protein VP3 was synthesized from an mRNA sedimenting at approx 18S between the 19S VP2 mRNA and the 16S VP1 mRNA. Other experiments showed that the VP2 and VP3 can be labeled with formyl methionine from initiator transfer RNA. It is concluded that there are three late polyoma virus mRNA's, each directing the synthesis of only one viral capsid protein. (25 refs.)

78-4480 **The Effect of Neonatal Thymectomy on the Growth Kinetics of a Virus-induced Renal Sarcoma of the Rat.** (Eng) Lang, W. (Dept. Pathology, Medical Sch., Hannover, W. Germany); Zobl, H.; Georgii, A. *Eur J Cancer* 14(4): 431-437; 1978.

The growth of polyoma virus-induced renal sarcomas in normal and neonatally thymectomized Wistar rats was compared. The animals were inoculated sc with 0.3 ml of a virus suspension ($TCID_{50}$ 10^6 or 10^7) within 24 hr after birth. A total of 41 unilateral and 65 bilateral renal tumors developed in 149 nonthymectomized (NT) rats; 17 unilateral and 44 bilateral sarcomas were found in 77 thymectomized rats. In both groups, there was an increase in tumor wt up to 50 days of age; thereafter, a slowdown of growth or even an incipient regression was observed. Tumor doubling time was 110 hr in the NT group and 76 hr in the thymectomized group. The quotient of absolute cell loss rates (loss in the proliferative compartment/loss in the nonproliferative compartment) was 70 in 30-day-old NT animals, 1.7 in 30-day-old thymectomized animals, 1.3 in 50-day-old TN animals, and 0.7 in 50-day-old thymectomized animals. The reduction of doubling time in tumors of thymectomized rats was due to an increase of the proliferative fraction despite increased cell loss rates. (39 refs)

78-4481 **Neoplastic Transformation of Hamster Brain Cells In Vitro by Polyoma Virus.** (Eng) de Micco, C. (Laboratoire de Neuropathologie, Faculte de Medecine, 27 Bd. Jean Moulin, 13005 Marseilles, France); Tripier, M. F.; Hassoun, J.; Lipcey, C.; Meyer, G.; Toga, M. *Int J Cancer* 21(4): 516-522; 1978.

The in vitro transformation of a line of hamster brain cells (HCxPy) by polyoma virus is reported. The primary cultures were initiated from newborn Syrian hamster cerebral hemispheres. Foci of transformed cells first appeared 120 days after viral infection, during the fourth passage in vitro. Three transformed cell types grew simultaneously: (1) small, stellate, triangular or multipolar cells with long, thin branched processes; (2) large, flat, often multinucleated cells; and (3) cells intermediate between these two types. About 10% of the cells were positive for polyoma-specific nuclear antigen and 60% to 90% were positive for polyoma-specific surface antigen. HCxPy cells were as effective as polyoma virus in protecting hamsters against challenge by a polyoma-induced transplantable sarcoma. Sc and intracranial injection of these cells into hamsters produced tumors that were serially transplanted in adult animals. All HCxPy cells were positive for brain-specific S 100 protein. Microscopic examination revealed H-type virus particles in some cells. The cells had glial features both in vivo and in vitro. The morphologic and immunohistochemical evidence indicates that the HCxPy cell line is neuroglial in nature. (30 refs)

78-4482 **Primase Initiates Okazaki Pieces During Polyoma DNA Synthesis.** (Eng) Eliasson, R. (Dept. Biochemistry I, Medical Nobel Inst., Karolinska Inst., S-104 01 Stockholm, Sweden); Reichard, P. *Nature* 272(5649): 184-185; 1978.

Study of an RNA that initiates Okazaki pieces (iRNA) during polyoma DNA synthesis revealed that iRNA is not synthesized by any of the known RNA polymerases. Instead, a mammalian enzyme, primase, is involved in catalyzing the initiation of Okazaki pieces of polyoma DNA. The primase can substitute deoxynucleotides for ribonucleotides, and, thus, it resembles the dnaG product of *Escherichia coli.* The mammalian enzyme appears to prefer ribonucleotides for primer formation, but in the presence of equimolar concentrations of ribo- and deoxynucleotides, a certain amount of misincorporation of deoxynucleotides can also take place. (12 refs)

78-4483 **Transformation of African Green Monkey Kidney Cells with the RF Strain of Human Papovavirus BKV.** (Eng) Bradley, M. K. (Dept. Microbiology, State Univ. New York, Upstate Medical Center, Syracuse, NY, 13210); Dougherty, R. M. *Virology* 85(1): 231-240; 1978.

The establishment of a stable transformed primate cell line by infection of semipermissive African green monkey kidney (AGMK) cells with the RF strain of human papovavirus BKV is described. The infected cultures showed slow cytopathic effects, and the surviving cells were cultured in the presence of virus-neutralizing antibody. This resulted in the emergence of a stable cell line (MRF-325) that is presently at the 100th tissue culture generation. The transformed line showed no tendency to form multiple layers. The monolayers were composed of small epithelioid cells interspersed with nests of larger cells and occasional multinucleate giant cells. Compared with normal AGMK cells, the MRF-325 cells attained a higher saturation density and displayed a decreased serum requirement for growth. They formed colonies in soft agar and multiplied in suspension culture. No infectious virus particles were isolated by cocultivation or fusion of MRF-325 with permissive human embryo kidney cells, and no virion antigens were detected. Papovavirus T antigen and U antigen were detected in 100% of the MRF-325 nuclei by immunoperoxidase staining. Normal and MRF-325, but not AGMK, cells were relatively resistant to challenge with simian virus 40 (SV40). MRF-325 cells supported high levels of human adenovirus type 5 replication compared with primary AGMK cells. Thus, RFV appears to provide a helper effect for adenovirus replication in AGMK cells similar to that observed with SV40. The data confirm that the oncogenic potential of human papovaviruses extends to primate cells. (48 refs)

78-4484 **Transcription and Coupled Transcription-Translation of Cloned DNAs in *Xenopus* Oocytes (Meeting Abstract).** (Eng) Mertz, J. E. (McArdle Lab. Cancer Res., Madison, WI); Gurdon, J. B.; De Robertis, E. M. *Miami Winter Symp* 14: 269; 1977. (no refs)

78-4485 **Binding of Dextran Sulfate by Virus-transformed Cells, with Reference to Altered Growth Behavior by Its Treatment.** (Eng) Goto, M. (Dept. Pharmacology, Pharmaceutical Inst., Aramaki aza Aoba, Sendai 980, Japan); Hori, K.; Ozawa, H.; Goto, K.; Sato, H. *Gann* 69(1): 31-37; 1978.

The binding of ^{3}H-dextran sulfate to untransformed (3T3) and virus-transformed (SV3T3) fibroblasts was studied in relation to the altered growth of transformed cells by this treatment. SV3T3-R5 cells, SV3T3 cells that were insensitive to dextran sulfate treatment, were also used. Liquid scintillation counting indicated that the rate of dextran sulfate binding to SV3T3 cells was dose-dependent. Autoradiography indicated that there was no difference in the binding capacity of dextran sulfate among 3T3, SV3T3, and SV3T3-R5 cells. The rate of binding proceeded linearly during the first 10 hr and then declined. Autoradiography of 0.3- to 0.5-μm sections of SV3T3 cells indicated that after 1 hr, all the dextran sulfate was located on the cell surface. After 8 hr, radioactivity was detected in the cytoplasm, and after 24 hr, it was detected in the nucleus. The localization of dextran sulfate was similar in 3T3 cells. The binding of dextran sulfate on SV3T3 cells was independent of the presence of calf serum in the medium. These findings suggest that the amount of dextran sulfate bound to transformed cells is not the main cause of the altered growth of these cells. (11 refs)

78-4486 **Involvement of DNA Polymerase α in Simian Virus 40 DNA Replication.** (Eng) Edenberg, H. J. (Dept. Biochemistry, Indiana Univ. Sch. Medicine, Indianapolis, IN, 46202); Anderson, S.; DePamphilis, M. L. *J Biol Chem* 253(9): 3273-3280; 1978.

Replicating simian virus 40 (SV40) chromosomes that were capable of continuing DNA synthesis in vitro were partially separated from mature SV40 chromosomes on sucrose gradients and analyzed for DNA polymerase activities. The DNA polymerases were extracted with salt, fractionated by 2-(diethylamino)ethylcellulose chromatography, and assayed under conditions selective for each polymerase. Both DNA polymerase α and DNA polymerase γ were found, although the former accounted for about 95% of the total DNA polymerase activity. The amounts of both enzymes were severalfold higher in replicating chromosomes than in mature chromosomes. DNA polymerase β was not found associated with SV40 chromosomes. These experiments were complemented by a study of the inhibition of DNA synthesis by $2'$:$3'$-dideoxythymidine $5'$-triphosphate (d_2TTP). SV40 DNA synthesis in nuclei isolated from infected CV-1 cells, in a soluble nuclear extract, or in replicating SV40 chromosomes was resistant to d_2TTP inhibition. DNA polymerase α was nearly as resistant to d_2TTP inhibition as were the in vitro DNA replication systems. DNA polymerases β and γ, however, were extremely sensitive to inhibition by d_2TTP. In the presence of d_2TTP, Okazaki fragments continued to be synthesized and joined to long chains of nascent DNA, and mature viral DNA forms were produced. Thus, virtually all the DNA synthesis observed in SV40 chromosomes replicating in vitro is performed by DNA polymerase α. (55 refs)

78-4487 **Simian Virus 40 as a Cloning Vehicle in Mammalian Cells.** (Eng) Hamer, D. H. (Lab. Molecular Genetics, Natl. Inst. Child Health and Human Development, NIH, Bethesda, MD, 20014). *Miami Winter Symp* 14: 37-47; 1977.

The in vitro generation of a simian virus 40 (SV40) transducing virus is described. An 870 base pair (bp) fragment of *Escherichia coli* DNA was ligated to a 3,700 bp fragment of SV40 DNA produced by cleavage with *Hpa*II and *Eco*RI. The recombinant molecules, which retained the early viral gene *A* but lacked sequences necessary for late gene expression, were propagated as virus by coinfection with a conditional lethal SV40 early gene mutant as helper. Preliminary experiments indicate that this system can be used to propagate foreign DNA fragments in productively infected, permissive monkey cells and in transformed, nonpermissive cells. (20 refs)

78-4488 **Association of DNA Polymerase α with Highly Purified Replicating Simian Virus 40 (SV40) Chromosomes (Meeting Abstract).** (Eng) Tsubota, Y. (Roswell Park Memorial Inst., Buffalo, NY, 14263); Waqar, M. A.; Spotila, L.; Kowalski, D.; Eddy, M.; Evans, M. *Fed Proc* 37(6): 1603; 1978. (20 refs)

78-4489 **Presence of Free Viral DNA in Simian Virus 40-Transformed Nonproducer Cells.** (Eng) Daya-Grosjean, L. (Institut de Recherches Scientifiques sur le Cancer, 94800 Villejuif, France); Monier, R. *J Virol* 27(2): 307-312; 1978.

Extracts from several simian virus 40 (SV40)-transformed nonproducer cells were prepared by the hot-phenol procedure normally used to extract cellular RNA. The cells were TSV-5 clone 2 Syrian hamster cells, CHK-SVLP clone 5.1 Chinese hamster cells, MKSV clone 11D mouse cells, V5 F4 clones 1 and 4 rat kidney cells, and V11 F1 clones 1 and 3 rat lung cells. All the extracts contained SV40 infectious units. Some of the infectious units were identified as SV40 form I DNA molecules. The results of reconstruction experiments suggest that SV40 form I DNA is extractable by the hot-phenol procedure because of its fast renaturation rate. Two hypotheses are proposed with respect to the origin of the free viral DNA: (1) spontaneous excision could occur at very low frequency in the transformed cells; (2) viral DNA replication might be initiated repeatedly at the level of an integrated copy, resulting in free linear molecules produced by branch migration. The significance of the presence of free viral DNA in nonproducer transformed cells is discussed. (16 refs.)

78-4490 **DNA Polymerase α Is Associated with Replicating SV40 Nucleoprotein Complexes.** (Eng) Otto, B. (Fachbereich Biologie, Univ. Konstanz, D-7750 Konstanz, W. Germany); Fanning, E. *Nucleic Acids Res* 5(5): 1715-1728; 1978.

Replicating simian virus 40 (SV40) nucleoprotein complexes were isolated from CV-1 cells infected with the Rh911 strain of SV40 and examined for the presence of DNA polymerase α to determine if this polymerase is the one responsible for cellular DNA replication. The complexes were extracted from the nuclei of infected cells and fractionated on neutral sucrose density gradients. Complexes containing replicating SV40 DNA (95S) were separated from those containing closed circular supercoiled viral DNA (75S). DNA polymerase activity was associated with the replicating nucleoprotein complexes but not with the slower sedimenting complexes. This polymerase activity coprecipitated with the nucleoprotein complexes in the presence of $MgCl_2$ and remained associated with the 95S complexes. It was identified as the α form on the basis of its sedimentation behavior, optimum salt concentration, and sensitivity to N-ethylmaleimide. DNA polymerase γ activity was also detected in the complexes, but not that of DNA polymerase β. These findings provide additional support for the role of the α polymerase in SV40 replication. It is not known, however, whether both the α and γ forms are required for replication. (28 refs)

78-4491 **Canine Kidney Cells: I. Neutral Lipids, Phospholipids and Fatty Acids of SV40 Transformed Canine Kidney Cells in Suspension and Monolayer Culture.** (Eng) Hougland, A. E. (Dept. Health Sciences, East Tennessee State Univ., Johnson City, TN, 37601); Gaush, C. R.; Hougland, M. W. *Growth* 42(1): 59-69; 1978.

Studies were undertaken to determine differences in lipid composition between simian virus 40 (SV40)-transformed canine kidney whole cells and their plasma membranes and to determine the effects of monolayer and suspension culturing on the lipid content of whole cells and their plasma membranes. The plasma membranes from monolayer and suspension cultures of transformed cells were isolated by the Warren fluorescein-mercuric acetate technique. Free cholesterol was the major neutral lipid extracted from the whole cells and plasma membranes of all these cultures. Triglyceride increased about 6- to 50-fold in suspension-grown whole cells and their plasma membranes, compared with those from monolayer cultures. The micromoles of total neutral lipids and phospholipids were elevated in the plasma membranes from monolayer cultures compared with whole cells from monolayer cultures, but they were similar in both suspension cultures. The distribution of fatty acids in the major lipid classes of plasma membranes and whole cells generally followed the composition of calf serum used in the culture media. (23 refs)

78-4492 **Okazaki Pieces Grow Opposite to the Replication Fork Direction During Simian Virus 40 DNA Replication.** (Eng) Kaufmann, G. (Biochemistry Dept., Weizmann Inst. Science, Rehovot, Israel); Bar-Shavit, R.; DePamphilis, M. L. *Nucleic Acids Res* 5(7): 2535-2545; 1978.

In vitro-synthesized simian virus 40 (SV40) nascent DNA chains of $<$ 200 nucleotides (Okazaki pieces) were hybridized to an excess of single-stranded DNA fragments of unidirectional fork polarities in order to determine the fork polarity of the Okazaki pieces. SV40 replicating DNA was pulse-labeled with α-^{32}P-deoxy ATP by an acellular DNA replication system. The Okazaki pieces were then isolated from the denatured replicating DNA by electrophoresis using a polyacrylamide gel column. The purified Okazaki pieces were hybridized to separated strands of Bgl$_1$ + Hpa$_1$ SV40 DNA restriction fragments immobilized on nitrocellulose filters. Only strands with the polarity of the DNA replication fork direction hybridized with the Okazaki pieces. The results are consistent with semidiscontinuous DNA synthesis in SV40, in which Okazaki pieces are synthesized only against the DNA replication fork direction. (18 refs.)

78-4493 **Stimulation of RNA Synthesis in Isolated Nucleoli by Preparations of Simian Virus 40 T Antigen.** (Eng) Whelly, S. (Univ. Nebraska Medical Center, Obstetrics Gynecology Res. Lab., Conkling Hall, 42nd and Dewey Ave., Omaha, Nebraska, 68105); Ide, T.; Baserga, R. *Virology* 88(1): 82-91; 1978.

The ability of simian virus 40 (SV40) tumor (T) antigen to directly stimulate RNA synthesis in isolated rat liver nucleoli was studied. Both partially and highly purified SV40 T antigen preparations stimulated the incorporation of ^{3}H-uridine triphosphate into the RNA of isolated nucleoli. The stimulation was largely α-amanitin-resistant, and it appeared that the product of nucleolar RNA synthesis included at least some preribosomal RNA sequences. T antigen was also partially purified from Chinese hamster cells transformed by the temperature-sensitive *ts*A mutant of SV40. The complement-fixing and RNA-stimulating activities of the *ts*T antigen, but not those of the T antigen from wild-type SV40-transformed cells, were inactivated by preincubation at 50 C. The stimulation of nucleolar RNA synthesis by three different preparations of T antigen was proportional to the complement-fixing activity of the preparation. Cellular RNA synthesis was also stimulated when quiescent 3T3 cells were infected with wild-type SV40. The results strongly suggest that one of the sites of action of SV40 T antigen may be at the level of transcription of nucleolar DNA. (49 refs.)

78-4494 **Inhibition of Postreplication Repair and the Enhancement of Induction of SV40 Virus from Transformed Hamster Kidney Cells.** (Eng) Zamansky, G. B. (Infectious Disease Unit, Massachusetts General Hosp., Boston, MA, 02114); Little, J. B.; Black, P. H.; Kaplan, J. C. *Mutat Res* 51(1): 109-119; 1978.

The effect of caffeine on the postreplication repair of UV-damaged DNA in untreated and mitomycin C-treated simian virus 40 (SV40)-transformed hamster kidney cells was studied. Exposure to 0.5, 1.0, or 2.0 mM caffeine after UV irradiation dramatically inhibited postreplication repair, preventing the cellular DNA molecules from reaching parental size. Caffeine decreased the survival of cells previously exposed to various concentrations of mitomycin for 8 hr, although caffeine in the absence of mitomycin C had no effect on cell survival. The level of virus induction was increased two- to threefold in cells grown for 8 hr in medium containing 0.025 or 0.05 μg/ml mitomycin C. At higher mitomycin C concentrations, virus yields decreased in the presence of caffeine. It is postulated that the persistence of gaps in UV-irradiated DNA, caused by the presence of caffeine, results in the enhancement of SV40 induction. (33 refs.)

78-4495 **Interference with Simian Virus 40 DNA Replication by Adenovirus Type 2 During Mixed Infection of Monkey Cells.** (Eng) Van Roy, F. (Lab. Molecular Biology, State Univ. Ghent, Ledeganckstraat, 35, B-9000 Ghent, Belgium); Fiers, W. *J Virol* 27(2): 275-287; 1978.

The inhibiting effect that human adenovirus (Ad) superinfection exerts on simian virus 40 (SV40) DNA replication was examined in detail, using established African green monkey kidney (AGMK) cell lines and SV40 strain 776. In AGMK cells coinfected with SV40 and human Ad2, the Ad2 interfered strongly with SV40 DNA biosynthesis. This interference was found to be a reproducible, delicately controlled phenomenon that was proportional to the multiplicity of infection of Ad and dependent on the active expression of the Ad genome. A pulse-chase experiment showed that Ad interference was not due to degradation of newly synthesized SV40 DNA molecules. Several early events of SV40 infection (adsorption, penetration, undercoating, induction of cellular DNA synthesis, and enhancement of Ad infection) were not markedly influenced by Ad-mediated interference. It is unlikely that interference is simply due to competition between SV40 and Ad for metabolites, enzymes, or replication sites, as interference by a constant amount of Ad is gradually suppressed as more SV40 is used in coinfections. The interference effect could also be partially neutralized by an increase in the time interval between SV40 infection and Ad coinfection. It was concluded that the interference was due to the activity of an Ad early gene product. The detailed mechanism of this Ad interference remains unclear. (28 refs.)

78-4496 **Decrease of Collagen Biosynthesis Ability of Rat Kidney Fibroblasts Transformed with SV-40 Virus.** (Eng) Bankowski, E. (Dept. Biochemistry, Inst. Biochemistry and Medical Analytics, Bialystok, Poland); Rzeczycki, W.; Nowak, H. K.; Jodczyk, K. J. *Mol Cell Biochem* 20(2): 77-84; 1978.

The effects of simian virus 40 (SV40) transformation on the ability of newborn rat kidney fibroblast cultures to produce collagen were determined. Both normal and transformed fibroblasts incorporated radioactive proline into their proteins. In normal cells, about 17% of the prolyl residues that were incorporated into the medium, 15% of the proteins extracted with 1 M NaCl, and about 5% of those bound with the cell sediment were hydroxylated. However, only about 1% of the proline-containing proteins synthesized by the transformed cells were hydroxylated. Study of the proline-containing proteins synthesized by the cells indicated that the transformed cells produced a significantly lower amount of collagenase-sensitive hydroxyproline-containing material than normal cells. Attempts to stimulate hydroxylation of the propyl residues indicated that insufficient activation of propylhyuroxylase in the transformed cells was not a cause of this phenomenon. Further study indicated that there were no collagenous proteins in the culture medium or cell extract. It is concluded that transformation of rat kidney fibroblasts by SV40 causes a loss or decrease in their ability to synthesize collagen. (17 refs.)

78-4497 **Localization of the 5′ Terminus of Late SV40 mRNA.** (Eng) Haegeman, G. (Lab. Molecular Biology, Univ. Ghent, Ledeganckstraat 35, 9000 Ghent, Belgium); Fiers, W. *Nucleic Acids Res* 5(7): 2359-2371; 1978.

A cap-containing T_1 oligonucleotide of simian virus 40-specific, cytoplasmic late messenger RNA (mRNA) was isolated by chromatography on acetylated dihydroxyboryl cellulose followed by fingerprinting, and it was identified by subsequent analysis with several nucleases. Its structure was determined to be 7m-G(5′)ppp(5′)-m-AmpU(m)pUp(Up,Cp)ApGp. This oligonucleotide constitutes the major 5′ terminus of both the 16S and 19S late mRNA species. Assuming that viral mRNA caps are derived from a 5′-terminal triphosphate or diphosphate, these results mean that late transcription is initiated mainly at nucleotide L 308. The cap is followed by a contiguous leader sequence that is at least 194 nucleotides long. The sequence of the leader of the viral late RNA's in this region corresponds exactly to a continuous sequence of the DNA genome. (33 refs.)

78-4498 **Immunity to Antigens Associated with Primate C-Type Oncoviruses in Pregnant Women.** (Eng) Hirsch, M. S. (Massachusetts General Hosp., Harvard Medical Sch., Boston, MA, 02114); Kelly, A. P.; Chapin, D. S.; Fuller, T. C.; Black, P. H.; Kurth, R. *Science* 199(4335): 1337-1340; 1978.

Cell-mediated and humoral immune responses against antigens associated with primate C-type oncoviruses were evaluated in humans by microcytotoxicity and radioimmunoprecipitation assays. Five of six women tested sequentially during pregnancy developed selective cell-mediated reactivity against baboon endogenous virus (BEV)-infected human fibroblasts. Responsiveness peaked during the second and third trimesters and corresponded temporarily with elevated antibody levels to BEV antigens. Similar cell-mediated reactivity was not observed in nonpregnant individuals (5 women and 9 men). Selective cell-mediated reactivity directed against cells infected with the simian sarcoma virus-simian sarcoma associated virus complex (SSV-SSAV) was observed in 4/20 healthy adults (3/14 nonpregnant and 1/6 pregnant). These observations suggest that cell-mediated reactivity against primate C-type oncoviruses is occasionally detected in healthy nonpregnant adults, but that during pregnancy both cell-mediated and humoral reactivity against BEV may become selectively expressed. (21 refs)

78-4499 **Template-specific Requirements for DNA Synthesis by the Mason-Pfizer Monkey Virus DNA Polymerase: Unique Aspects.** (Eng) Marcus, S. L. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Sarkar, N. H.; Modak, M. J. *Biochim Biophys Acta* 519(2): 317-330; 1978.

The biochemical properties of DNA polymerase from Mason-Pfizer monkey virus (MP-MV) were investigated with respect to synthetic and natural template-primer utilization. The polymerase preferred template:primer ratios of 2.5-5:1 for optimal rates of synthesis with polycytidylic acid [poly (C)]-deoxyguanosine $_{12018}$ as template-primer. The addition of low concentrations of inorganic phosphate to the reaction mixture stimulated polyadenylic acid-directed synthesis. The chemically modified template poly(rCm) was less efficiently utilized than poly(C). Poly(2′-O-methylcytidylate), however, was the only template for which MP-MV DNA polymerase expressed a preference for $Mn^{2}+$ as divalent cation. In addition, heteropolymeric regions of rabbit globin messenger RNA and avian myeloblastosis virus (AMV) 70S RNA could be copied by the MP-MV polymerase with oligodeoxythymidylic acid, oligouridylic acid, or with endogenous primers, as in the case of AMV RNA. Although the MP-MV enzyme shared some properties with B- and C-type mammalian viral enzymes, other properties rendered it unique. The results show that, under the proper conditions, MP-MV DNA polymerase can copy natural RNA templates as efficiently as B- and C-type viral enzymes. (24 refs.)

78-4500 **Distribution and Transmission of Primate Type-C Virus.** (Eng) Kawakami, T. G. (Comparative Oncology Lab., Univ. California, Davis, CA, 95616); Sun, L.; McDowell, T. S. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 33-36; 1978.

Blood samples from 416 captive animals representing several different primate species (orangutan, chimpanzee, gorilla, macaque, woolly monkey, and marmoset) were examined for serum humoral antibody and infectious virus in an effort to determine the distribution of infectious primate C-type virus. The gibbon was the only primate that was found to be infected with a C-type virus. The virus was isolated from 9% of viremic gibbons, and humoral antibody was detected in 16% of gibbons exposed to the virus. Evidence of virus infection was found in 2/4 gibbon species studied. Contact transmission of virus was studied in pairs of gibbons in which both members were noninfected, one member was noninfected and the other was antibody-positive, one member was antibody-positive and the other was viremic, or one member was viremic and the other was noninfected. Transmission of the virus occurred only when viremic animals were involved, and virus infection occurred only when a viremic juvenile was housed with a noninfected animal of the same age. In parent-progeny transmission, virus infection occurred in offspring from viremic parent but not from noninfected or antibody-positive parents. Nucleic acid hybridization experiments using DNA from the muscle tissue of viremic animals indicated that infection in parent-progeny-infected animals occurred during the prenatal period of development or by vertical transmission. (8 refs.)

78-4501 **Identification of a Lymphotropic Simian Herpesvirus Receptor on Lymphoid Cells (Meeting Abstract).** (Eng) Nigida, S. M. (Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL, 60612); Falk, L. A. *Fed Proc* 37(6): 1747; 1978. (no refs)

78-4502 **Biochemical Transformation of Mouse Cells by Herpes Simplex Virus Type 2: Enhancement by Means of Low-Level Photodynamic Treatment.** (Eng) Verwoerd, D. W. (Veterinary Res. Inst., Onderstepoort 0110, S. Africa); Rapp, F. *J Virol* 26(1): 200-202; 1978.

The effect of low-level photodynamic treatment on the transformation of thymidine kinase-deficient (TK-) 3T3-derived mouse cells (N clA cl10) by UV-irradiated herpes simplex virus type 2 (HSV-2) was determined. The inactivation of HSV-2 was first studied at high (5 μg/ml) and low (0.08 μg/ml) proflavine concentrations. Exposure of the virus to the lower concentration and 30-watt fluorescent light for 2 min had no effect on the infectivity of the virus. Studies with primary hamster embryo fibroblasts under standard conditions (1 hr incubation with 0.08 μg/ml proflavine/ml followed by exposure to light for 2 min) revealed only a 2%-5% decrease in DNA synthesis. Low-level photodynamic treatment of N clA cl10 cells at different intervals after infection also had little effect on DNA synthesis. However, treatment of TK- N clA cl10 cells under standard conditions for 2 or 5 min had an enhancing effect on the biochemical transformation of these cells to TK-positive cells. There was a two- to threefold increase in the number of transformed foci over untreated controls. With 0.08 μg/ml proflavine, the virus was not inactivated and virus and cellular DNA syntheses were inhibited only marginally. These findings suggest that photodynamically activated dyes could have a cocarcinogenic effect at very low concentrations. (4 refs)

78-4503 **Neutralizing Antibodies Against Herpes Simplex Virus Type 2 in Women with Cervicouterine Cancer.** (Spa) Ruiz-Gomez, J. (Laboratorio Nacional de Virologia, Mexico City, Mexico); Gallegos Vargas, G.; Carranza Garcia, J.; Alvarez y Munoz, T. *Ginecol Obstet Mex* 42(254): 369-375; 1977.

The presence of neutralizing antibodies against herpes simplex virus type 2 in the cervix or women with gynecological neoplasms was determined and compared to findings in women with nonneoplastic gynecologic disorders and 121 healthy controls. Neutralizing antibodies were found in 44/121 controls, in 56/93 women with cancer of the uterus and cervix ($p < 0.001$), in 7/20 patients with mild cervical dysplasia, in 8/15 patients with moderate dysplasia, in 8/15 patients with severe dysplasia, in 11/19 patients with cervicitis, in 20/63 patients with colpocervicitis, and in 25/69 patients with nongenital neoplasias. The findings indicate that there is a correlation between the incidence of virus-neutralizing antibodies and gynecological cancer and the degree of cervical dysplasia. (16 refs)

78-4504 **Induction of Cervical Carcinoma in the Mouse by Prolonged Genital Exposure to Formalin**

Inactivated Herpes Simplex Viruses Types 1 and 2 (HSV-1 and HSV-2) (Meeting Abstract). (Eng) Wentz, W. B. (Dept. Reproductive Biology, Case Western Reserve Univ. Sch. Medicine, Cleveland, OH); Reagan, J. W.; Heggie, A. D. *Pediatr Res* 12(4, part 2): 476; 1978. (no refs)

8-4505 **Binding to Chromosomes of Herpes Simplex-related Antigens in Biochemically Transformed Cells.** (Eng) Kit, S. (Div. Biochemical Virology, Baylor Coll. Medicine, Houston, TX, 77030); Kurchak, M.; Wray, W.; Dubbs, D. R. *Proc Natl Acad Sci USA* 75(7): 3288-3291; 1978.

The association of herpes simplex virus (HSV)-related antigens with chromosomes was demonstrated in human [HeLa(-BU25)] and mouse [LM(TK-)] cells biochemically transformed by UV-irradiated HSV-1. This was accomplished by using the sensitive peroxidase-antiperoxidase immunological staining technique with rabbit antisera that had high neutralizing titers against both HSV-specific thymidine kinase activity and virus infectivity. Antisera against HSV did not react with chromosomes of uninfected cells, and normal sera did not react with any of the constituents of biochemically transformed cells. Methanol/acetic acid treatment of biochemically transformed cells eliminated their nuclear staining for HSV-related antigens. In vitro binding of HSV-related antigens to chromosomes was demonstrated by incubating soluble antigens from high salt extracts of HSV-infected cells with methanol/acetic acid-fixed chromosomes of biochemically transformed or uninfected cells, followed by exposure to antiserum against HSV and peroxidase-anti peroxidase staining. There was no staining when soluble extracts from uninfected cells were substituted for those from HSV-infected cells. The results show that cells biochemically transformed and lytically infected by HSV contain antigens that, like the Epstein-Barr virus-associated nuclear antigen, bind to chromosomes in vivo and in vitro. (25 refs.)

78-4506 **The Effect of Herpesvirus Infection and 2-Deoxy-D-Glucose on Glycosphingolipids in BHK-21 Cells.** (Eng) Ray, E. K. (Dept. Physiology and Biochemistry, Medical Coll. Pennsylvania, Philadelphia, PA, 19129); Blough, H. A. *Virology* 88(1): 118-127; 1978.

The effect of herpes simplex virus (HSV) infection and 2-deoxy-D-glucose (dGlc) on the amount of radiolabeled glycosphingolipids in BHK-21 cells was studied after a 16-hr pulse with D-[3-^{3}H]serine and [1-^{14}C]palmitate. HSV infection resulted in a two- to threefold increase in the amount of labeled ceramides, ceramide monohexosides, and ceramide dihexosides and in a 50% reduction in labeled hematosides. Treatment with 10 mM dGlc increased the proportion of ceramide relative to its hexosides and reduced the total content of labeled glycolipids significantly. Glycosylation of endogenous ceramide was studied in vitro using microsomal fractions and uridine diphosphate (UDP)-[^{3}H]glucose as the sugar donor. The activity of UDP-glucose: ceramide-glucosyltransferase in cellular microsomal fractions was stimulated 30% to 80% by HSV infection and 30% to 60% by dGlc treatment. Both HSV infection and dGlc treatment increased the number or accessibility of glucose-acceptor sites. Thus, dGlc inhibits glycolipid synthesis in uninfected and HSV-infected cells by blocking the glycosylation of ceramide. The inhibition of glycolipid synthesis may play a role in virion assembly. (35 refs.)

78-4507 **Antibodies to Herpesvirus Type 1 and Type 2 among Japanese Cervical Cancer Patients.** (Eng) Ozaki, Y. (Dept. Microbiology, Shiga Univ. Medical Science, Seta, Ohtsu, Shiga-ken 520-21, Japan); Ishiguro, T.; Ohashi, M.; Sawaragi, I.; Ito, Y. *Gann* 69(1): 119-122; 1978.

The presence of antibodies to herpesvirus simplex virus types 1 and 2 (HSV-1,-2) was determined in 69 Japanese women (aged 31-72 yr) with cancer of the uterine cervix and 112 matched controls (aged 14-73 yr). The cancer patients included 7 with cervical dysplasia, 9 with carcinoma in situ, and 53 with invasive carcinoma. Antibodies to HSV-2 were found in 3 patients with dysplasia, 1 with carcinoma in situ, and 15 with invasive cancer; 17 controls also had antibodies to HSV-2. The mean antibody titer (log$_{10}$) to HSV-1 and HSV-2 was 1.89 and 1.70, respectively, in the dysplasia group, 1.95 and 1.35 in the dysplasia group, 2.12 and 1.65 in the invasive cancer group, and 2.14 and 1.67 in the control group. These findings do not support the hypothesis that there is an association between genital herpes (HSV-2) and cervical cancer. (7 refs)

78-4508 **Antibody Patterns to Herpesviruses in Kaposi's Sarcoma: II. Serological Association of American Kaposi's Sarcoma with Cytomegalovirus.** (Eng) Giraldo, G. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Beth, E.; Henle, W.; Henle, G.; Mike, V.; Safai, B.; Huraux, J. M.; McHardy, J.; de-The, G. *Int J Cancer* 22(2): 126-131; 1978.

The major finding of an extended serologic analysis of American and African Kaposi's sarcoma (KS) patients and appropriately matched control groups was the detection of a specific serologic association of cytomegalovirus (CMV) with American KS patients. All American KS sera contained CMV antibodies, and their geometric mean titers (GMT) were significantly higher than those in sera of melanoma patients [GMT ratio $k = 5.3$ to 7.7 by complement fixation (CF), $k = 8.9$ by indirect hemagglutination (IHA)] or in sera of age- and sex-matched healthy controls ($k = 12.6$ to 16.0

by CF, k = 12.6 by IHA). These results are similar to the data obtained previously for European KS. Although the GMT to CMV of African KS patients were similar to the GMT of the American KS groups, their significance cannot be demonstrated because of the high background of CMV infections in the control groups. Complex mechanisms are hypothesized, by analogy with the Epstein-Barr virus involvement in Burkitt's lymphoma, for a CMV involvement in the development of KS. (48 refs.)

78-4509 **Association of Cytomegalovirus (CMV) Infection with Cervical Cancer: Isolation of CMV from Cell Cultures Derived from Cervical Biopsy.** (Eng) Melnick, J. L. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX, 77030); Lewis, R.; Wimberly, I.; Kaufman, R. H.; Adam, E. *Intervirology* 10(2): 115-119; 1978.

Cytomegalovirus (CMV) was isolated from cell cultures derived from 2/10 cervical cancer biopsy specimens of patients with invasive carcinoma. Five serial passages were necessary before extensive cytopathic changes characteristic of CMV infection appeared. All patients tested had higher titers of complement-fixing antibodies to both isolates than antibodies to the prototype AD169 strain of CMV. Six of eight patients tested also had herpesvirus type 2 antibodies. (18 refs.)

78-4510 **Epstein-Barr Virus-positive Japanese Burkitt Lymphoma.** (Eng) Miyoshi, I. (Dept. Medicine, Okayama Univ. Medical Sch., Shikatacho 2-5-1, Okayama 700, Japan); Hiraki, S.; Tsubota, T.; Uno, J.; Nakamura, K.; Ota, T.; Hikita, T.; Hayashi, K.; Kataoka, M.; Tanaka, T.; Kimura, I.; Sairenji, T.; Hinuma, Y. *Gann* 69(3): 449-450; 1978.

The first reported case of a Japanese patient with Burkitt's lymphoma whose lymphoma cells contained Epstein-Barr virus (EBV) nuclear antigen (EBNA) is described. The 29-yr-old man presented with massive ascites and large abdominal masses; cytology was suggestive of abdominal Burkitt's lymphoma. Laboratory studies indicated that 60%-70% of the ascitic lymphoma cells were EBNA-positive and that 7%-16% were EBV membrane antigen-postive. Chemotherapy resulted in regression of the ascites and abdominal masses, but 2 mo later the abdominal masses recurred with the development of meningeal leukemia. Treatment was ineffective. Autopsy revealed massive tumors involving the gastrointestinal tract, mesentery, and retroperitoneum. Histology revealed Burkitt's type undifferentiated malignant lymphoma. This case suggests that EBV may cause certain lymphomas in Japan. (10 refs.)

78-4511 **Epstein-Barr Virus-associated Antigens in Nasopharyngeal Carcinoma. Relation to Clinical Course of American Patients.** (Eng) Coates, H. L. (Mayo Clinic, 200 First St. SW, Rochester, MN, 55901); Pearson, G. R.; Neel, H. B.; Weiland, L. H. *Arch Otolaryngol* 104(8): 427-430; 1978.

The relationship between antibody titers to Epstein-Barr virus (EBV)-associated antigens and nasopharyngeal carcinoma (NPC) in American patients was quantitated with the use of 35 serum specimens. These sera were compared with 85 specimens from patients with other head and neck cancers, 80 specimens from patients with lymphoma, and 47 specimens from healthy controls. There was a definite correlation between the presence of NPC and the level of antibody titers to EBV. In particular, tests that detected antibody to early antigen and antibody to viral capsid antigen in the serum IgA fraction were highly specific for the presence of this carcinoma. Treatment that resulted in clinical remission of the disease brought about a significant decrease in these antibody titers. These findings indicate that there was a consistent and direct association between the presence of and antibody levels to EBV antigens and the presence of NPC in American patients. (20 refs.)

78-4512 **Epstein-Barr Virus Infection Following Bone-Marrow Transplantation.** (Eng) Sullivan, J. L. (Dept. Pediatrics, Univ. Massachusetts Medical Center, 55 Lake Ave. North, Worcester, MA, 01505); Wallen, W. C.; Johnson, F. L. *Int J Cancer* 22(2): 132-135; 1978.

A 12-yr-old boy with acute lymphoblastic leukemia received a bone-marrow transplant (BMT) from a matched sibling donor. Nine weeks prior to transplant, the donor experienced Epstein-Barr virus (EBV)-induced infectious mononucleosis. The bone-marrow recipient was EBV-negative at the time of transplant; however, 4 wk posttransplant, the recipient developed clinical symptoms of graft-vs-host disease (GVHD) coincident with serological evidence of acute EBV infection. In addition, a lymphoblastoid cell line positive for Epstein-Barr nuclear antigen was established for a bone-marrow sample obtained at the onset of symptoms compatible with GVHD. Sera obtained from the recipient over the ensuing 2 mo showed the appearance of antibodies to specific EBV antigens, a phenomenon consistent with a primary immune response to EBV infection. This association of acute EBV infection with symptoms of GVHD in a BMT recipient suggests a need for further investigation of the epidemiology of EBV infections in human bone-marrow transplantation and the relationship between EBV infection and GVHD. (16 refs.)

78-4513 **Heterogeneity of Epstein-Barr Virus. IV. Induction of a Specific Antigen by EBV from Two**

Transformed Marmoset Cell Lines in Ramos Cells. (Eng) Fresen, K. O. (Institut fur Virologie, Zentrum fur Hygiene, Universitat Freiburg, Hermann-Herder-Strasse 11, 78 Freiburg, W. Germany); Cho, M. S.; zur Hausen, H. *Int J Cancer* 22(2): 160-165; 1978.

nfection of cells of the Epstein-Barr virus (EBV) genome-negative human B-lymphoma Ramos line with viral solates obtained from two EBV-transformed marmoset cell ines (B95/8; Nyevu) resulted in the induction of a nuclear antigen (RAM-ag) that apparently was different from other EBV-associated antigen complexes. This antigen was revealed by indirect immunofluorescence, and it showed no deectable cross-antigenicity with Epstein-Barr nuclear antigen (EBNA) or any other known EBV-associated antigen. EBV solates from P3HR-1 cells failed to induce a similar antigen n Ramos cells, although they induced EBNA. In addition, no RAM-ag was expressed after infection of cells of another EBV genome-negative human B-lymphoma line (BJAB) with B95/8 EBV or in a series of EBV-harboring cell lines. Thus, he antigen appears to be specific for Ramos cells. It was also nduced upon infection of either B95/8 or P3HR-1-converted Ramos sublines with EBV from B95/8 cells. All human sera with RAM-ag reactivity revealed antibodies against virus capsid antigen (VCA) However, sera from patients with acute nfectious mononucleosis containing high anti-VCA antibodies did not react with RAM-ag. Apparently, seroconversion for this antigen coincides more closely with the appearance of EBNA-directed antibodies. (23 refs.)

78-4514 **Polyploidization of Epstein-Barr Virus (EBV)-carrying Lymphoma Lines Decreases the nducibility of EBV-determined Early Antigen Following P3HR-1 Virus Superinfection or Iododeoxyuridine Treatment.** (Eng) Shapiro, I. M. (Dept. Tumor Biology, Karolinska Institutet, S-104 01 Stockholm 60, Sweden); Andersson-Anvret, M.; Klein, G. *Intervirology* 10(2): 94-101; 1978.

Four tetraploid cell lines were derived from the Epstein-Barr virus (EBV)-carrying, nonproducer Burkitt's lymphoma line Raji after colcemide treatment. EBV early antigen was induced by superinfection with the P3HR-1 strain of EBV and by iododeoxyuridine treatment. Inducibility was reduced in all tetraploid lines compared with the near-diploid counterpart. The av number of EBV genome equivalents per tetraploid cell was 60-70, compared with 50 in diploid cells. This contrasts with the EBV genome amplification previously observed in somatic cell hybrids. (16 refs.)

78-4515 **Effect of Cordycepin Triphosphate on In Vitro RNA Synthesis by Picornavirus Polymerase Complex.** (Eng) Panicali, D. L. (Dept Cell and Molecular Biology, Medical College of Georgia, Augusta, GA, 30902); Nair, C. N. *J Virol* 25(1): 124-128; 1978.

The effect of cordycepin triphosphate (3′-deoxyATP; 3-dATP) on in vitro RNA synthesis by crude viral polymerase complexes isolated from HeLa cells infected with human rhinovirus type 14 or poliovirus type 2 was investigated. Polymerase complexes were also prepared from uninfected cells. In the presence of all four nucleotide triphosphates, 0.5 mM 3-dATP stimulated ^{3}H-AMP incorporation; when ATP was the sole precursor, 3-dATP caused marked stimulation of AMP incorporation not only by uninfected cell enzyme but also by the viral polymerase complexes. 3-dATP inhibited ^{3}H-guanosine monophosphate incorporation by both viral polymerase complexes. This inhibition could be reversed by adding ATP to the reaction mixture along with the inhibitor, but not by adding excess guanosine triphosphate or by adding ATP 10 min after the inhibitor. Viral products synthesized in the presence of 3-dATP lacked full-length single-stranded viral RNA. It is suggested that 3-dATP inhibits picornavirus-specific RNA synthesis by ATP competition with RNA chain termination. (17 refs.)

78-4516 **The Importance of Transcription Unit Definition.** (Eng) Darnell, J. E. (Molecular Cell Biology Dept., Rockefeller Univ., 1230 York Ave., New York, NY, 10021); Weber, J.; Goldberg, S.; Jelinek, W.; Fraser, N.; Sehgal, P. *Miami Winter Symp* 14: 197-215; 1977.

In attempts to define late adenovirus 2 (Ad2) transcripts, RNA molecules in the process of formation, "nascent" RNA, were examined. Both inside the cell and in isolated nuclei, the labeling time for RNA synthesis can be restricted to such a short period that only the termini of nascent molecules are labeled. Pulse-labeled RNA from labeled cells or from isolated nuclei labeled in vitro were separated into size classes. The RNA was then hybridized to a series of DNA fragments. The longest labeled nascent molecules were complementary to the rightmost Ad2 DNA fragment, *Eco* C, the next longest molecules hybridized to *Eco* E, and so on, back to *Sma* b. As much as 90% of the transcriptional output of any region of the Ad genome could be detected as part of a single sedimenting species. A single promoter may exist in the region around 0.2; therefore, the RNA molecules that arise from the righthand 80% of the genome will all start in the same region. RNA synthesis in a given transcription unit was decreased within the unit as an exponential function of distance from the promoter. These findings were confirmed in assays of the effect of transcription on specific regions of the Ad2 genome using HeLa cells late in Ad2 infection that were exposed to UV radiation. More than 90% of the transcription that occurs from Ad2 DNA late in infection probably begins near 0.2 and reaches to the righthand end of the genome. Since at least 20% of the RNA formed in the nucleus exits to the cytoplasm, it is concluded that the cell is able to synthesize a long RNA molecule and then modify this molecule stepwise through to messenger RNA formation. (36 refs)

78-4517 Inhibition of Exogenous RNA-dependent Protein Synthesis by a Low-Molecular-Weight RNA from Nuclear Ribonucleoprotein Particles of Adenovirus-infected HeLa Cells. (Eng) Sarma, M. H. (Div. Labs. and Res., New York State Dept. Health, Albany, NY, 12201); Beach, T. A.; Chatterjee, N. K. *Biochem Biophys Res Commun* 82(1): 384-391; 1978.

The effect of low-mol-wt RNA (I-RNA: 4S to > 5.5S) isolated from the nuclear ribonucleoprotein particles of adenovirus-infected HeLa cells on the cell-free protein synthesis directed by polyribosomal RNA from rabbit reticulocytes was studied. Increasing concentrations of I-RNA inhibited the incorporation of leucine by > 80% when protein synthesis was directed by either total or poly(A)-containing rabbit reticulocyte polyribosomal RNA. The kinetics of inhibition were similar with both preparations. Comparison of the kinetics of this inhibition with those produced by aurintricarboxylic acid indicated that I-RNA-dependent inhibition occurs predominantly at the level of chain initiation. I-RNA-dependent inhibition was relieved approx 30% by adding an excess of initiation factors, suggesting that I-RNA interacts directly with some of these factors. In a reconstituted system, I-RNA did not prevent the binding of Met-tRNAf-GTP-IF ternary complex to 40S subunits, although it did repress the formation of 80S from 40S-mRNA complex and 60S subunits. In binding assays in which authentic IF-M2A and IF-M2B were present, the inhibitor competed with messenger molecules for binding site(s) in IF-M2B. The inhibitory RNA appears to be a 5.5S RNA. (14 refs)

78-4518 Transformation of a Rat Cell Line by an Adenovirus 7 DNA Fragment. (Eng) Sekikawa, K. (Cancer Res. Inst., Sapporo Medical Coll., Sapporo 060, Japan); Shiroki, K.; Shimojo, H.; Ojima, S.; Fujinaga, K. *Virology* 88(1): 1-7; 1978.

The transforming fragment of the weakly oncogenic adenovirus 7 (Ad7, subgroup B) was studied via transfection experiments using a clonal rat embryo cell line, 3Y1. Partial digestion of Ad7 DNA by *Hin*dIII yielded fragments *Hin*dIII-B and "a"; further digestion of the latter yielded fragments *Hin*dIII-I and *Hin*dIII-J. Transfection of 3Y1 cells with Ad7 DNA and the *Hin*dIII-I-J fragment, but not with the *Hin*dIII-B fragment, yielded transformed foci and cell lines positive for adenovirus tumor antigen. Most, if not all, of the *Hin*dIII-I-J fragment persisted in the cells transformed by this fragment at a rate of 7.8 to 9.5 copies per haploid cell DNA quantity. Approx two-thirds of the viral DNA genome was present in cells transformed by Ad7 whole DNA at a rate of 3.7 to 5.7 copies per haploid quantity of cell DNA. The data indicate that the transforming gene of Ad7 is located in the *Hin*dIII-I-J fragment and is mapped at the left 8% of the Ad7 genome. (20 refs.)

78-4519 Adenovirus Type 2 Early Proteins Synthesized In Vitro and In Vivo: Identification in Infected Cells of the 38,000- to 50,000-Molecular-Weight Protein Encoded by the Left End of the Adenovirus Type 2 Genome. (Eng) Harter, M. L. (Cold Spring Harbor Lab., Cold Spring Harbor, NY, 11524); Lewis, J. B. *J Virol* 26(3): 736-749; 1978.

Sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) was used to identify the proteins synthesized early after the infection of human (HeLa) cells with adenovirus type 2 (Ad2). The major products of cell-free protein synthesis with messenger RNA (mRNA) complementary to the *Eco*RI C fragment had mol wts of 21,000 daltons (21K), 19K, 17K, and 11K; small amounts of 25K, 14K, and 13K polypeptides were also identified. With mRNA complementary to the *Eco*RI D fragment, 14K, 13K, and 15.5K polypeptides were synthesized. Polypeptides 15K and 44K to 50K were synthesized with mRNA complementary to the *Eco*RI A fragment. Six prominent proteins appeared in nuclei from Ad2-infected but not mock-infected cells: 72K, 24K, 21K, 19K, 15K, and 11K. 12K also appeared as a minor species. Cytoplasmic extracts of infected but not mock-infected cells contained 72K, 21K, 14K, and 12K polypeptides. Proteins 40K to 50K, 17K, 15.5K, and 13K were not detected in infected cells. Comparison of extracts from infected and mock-infected cells using two-dimensional isoelectric focusing/SDS-PAGE revealed six additional polypeptides with mol wts of 38K to 50K in the infected cells. A cluster of polypeptides of 38K to 50K was also synthesized in vitro using early mRNA complementary to *Eco*RI A. Partial protease digestion showed the 38K to 50K proteins synthesized in infected cells to be related in sequence to each other and to the 44K to 50K polypeptides synthesized in vitro. The results show that several closely related proteins very similar to the 38K to 50K species encoded by the left 4.5% of the Ad2 genome are synthesized in infected cells and that they accumulate in the nucleus. (34 refs.)

78-4520 Adaptation of Murine MOPC-315 Myeloma Cells to Growth In Vitro and Further Characterization of Their C-Type Viruses. (Eng) Yaniv, A. (Dept. Human Microbiology, Sackler Sch. Medicine, Tel-Aviv Univ., Tel-Aviv, Israel); Gazit, A.; Dvir, M.; Guthmann, D.; Eylan, E. *Eur J Cancer* 14(7): 771-779; 1978.

MOPC-315 myeloma cells were successfully adapted to growth in tissue culture, and the established cell line has been propagated in suspension for 2 yr (210th generation). Its tumorigenic potential was tested in BALB/c mice. Plasma cell neoplasms developed within 10 days following ip or sc injection of 10^6 cells from various generations. The viral particles released by the tumor cells during their replication in vitro were indistinguishable by nucleic acid hybridization from the C-type particles secreted into the ascitic fluid of mice bearing MOPC-

315 myelomas. The in vitro-secreted viruses, unlike those released in vivo, were unstable in sucrose and glycerol-TNE [TNE: 0.01 M Tris-HCL (pH 7.4), 0.1 M NaCl, 0.001 M EDIA] gradients. This instability was partially overcome by the addition of a reducing agent, such as dithiothreitol. The viral particles banded at densities of 1.16 and 1.19 g/ml in sucrose and glycerol gradients, respectively. Detergent treatment of MOPC-315 particles released the viral core, which had a buoyant density of 1.26 g/ml in a sucrose gradient. Two major species of RNA molecules were detected: a heavy 70S RNA and a 15S RNA; occasionally, 35S RNA molecules were also detected. The myeloma particles were infectious and capable of inducing syncytia in XC cells. (11 refs)

78-4521 **Expression of Murine C-Type Endogenous Virus by Cell Lines Derived from the 129 Mouse Teratocarcinoma.** (Fre) Emanoil-Ravicovitch, R. (Groupe de Recherches sur la Virologie des Leucemies, Unite 107 de l'I.N.S.E.R.M., Hopital Saint-Louis, 75010 Paris, France); Robert, J.; Canivet, M.; d'Auriol, L.; Peries, J. *C R Acad Sci [D] (Paris)* 28(17): 1277-1280; 1978.

Hybridization with complementary DNA (cDNA) viral probes prepared from ecotropic virus (V1) and xenotropic virus (V2) from ML/8155 infected BALB mice was performed in order to detect murine C-type viral information in differentiated and undifferentiated cell lines from the 129 mouse teratocarcinoma. Unlike the differentiated cell lines PCD1 and PCD3, the two undifferentiated cell lines, PCC4 and PCC3, were multipotential; however, only PCC3 was capable of differentiating in vitro. Other cell lines tested for hybridization with cDNA were 129 mouse embryo fibroblasts, BALB/c mouse fibroblasts, dog thymus cells, and single rhesus monkey cells. The DNA probe prepared from V1 demonstrated only slight homology with nuclear DNA in the cell lines tested. The V2 DNA probe demonstrated hybridization kinetics with the PCD1 and PCC4 lines comparable to that with the BALB/c mouse fibroblasts. Viral RNA, as demonstrated by V1, was negligible in both differentiated and undifferentiated teratocarcinoma cell lines. RNA expression using the V2 DNA probe was significantly higher in the PCC4 and PCC3 cell lines than in PCD3. When the PCC4 and PCC3 cell lines underwent differentiation in vivo, the C-type viral RNA information decreased. (6 refs)

78-4522 **Electronmicroscopic Identification of Type C Particles in Cultured Murine Neuroblastoma.** (Eng) Kataoka, S. (Dept. Anatomy, Coll. Medicine, Univ. Iowa, Iowa City, IA, 52242); Williams, T. H.; Sandquist, D.; Sahu, S. K. *Experientia* 34(7): 879-881; 1978.

Examination of monolayers of murine neuroblastoma cells treated with dexamethasone (dex) by electron microscopy revealed C-type virus particles associated with the plasma membrane or in the extracellular space. Stages ranged from budding forms to free particles. Dex increased particle incidence over that in untreated cells, but it is not known whether virus proliferation was induced by dex or if it was due to the presence of a greater number of differentiated cells. (18 refs)

78-4523 **Spontaneous Adrenal Cortical Carcinoma in the NIH Swiss Mouse in the Absence of Overt Retravirus Expression (Meeting Abstract).** (Eng) Strickland, J. E. (Viral Oncology Program, NCI and Litton Bionetics Inc., Frederick Cancer Res. Center, Frederick, MD, 21501); Saviolakis, G. A.; Fowler, A. K.; Weislow, O. S.; Allen, P. T.; Hellman, A. *Fed Proc* 37(6): 1560; 1978. (no refs)

78-4524 **Expression of C-Type Endogenous Virus in Cell Lines Derived from Mouse Teratocarcinomas (Meeting Abstract).** (Fre) Emanoil-Ravicovitch, R. (INSERM 107, Hopital Saint-Louis, 75475 Paris Cedex 10, France); Robert, J.; Canivet, M.; Peries, J. *Bull Cancer (Paris)* 65(1): 34; 1978. (no refs)

78-4525 **SWAN Mice: Autoimmune Mice Nonproductive of Xenotropic C-Type Virus (Meeting Abstract).** (Fre) Blaineau, C. (Laboratoire d'Immunologie et de Virologie des Tumeurs, INSERM U.152, Hopital Cochin, 75 Paris, France); Gisselbrecht, S.; Piater, D.; Hurot, M. A.; Levy, J. P. *Bull Cancer (Paris)* 65(1): 34-35; 1978. (no refs)

78-4526 **Particles Resembling C-Type Oncornaviruses in Normal Rat Prostate.** (Eng) Zeller, N. K. (Dept. Microbiology, Univ. Maryland Dental Sch., Baltimore, MD, 21201); Bashirelahi, N.; Sydiskis, R. J. *Cancer Res* 38(9): 3104-3106; 1978.

The occurrence of viruslike particles in the ventral lobe of the prostate of a retired CD breeder rat is reported. The particles appeared to be budding from the epithelial cells into the intercellular spaces. They exhibited features morphologically similar to immature C-type particles, with an electron-lucent center and a diameter of approx 100 nanometers. Neither intracellular A-type particles nor mature C-type particles were observed. The particles appeared to be associated only with the epithelial cells lining the prostatic lumens. None of the other organs or tissues from this rat contained any virus-like particles. This is the first report of particles resembling oncornaviruses in normal rat prostatic tissue. (27 refs.)

78-4527 **Ecotropic Type C RNA Virus Expression and Its Consequences in Congenic Resistant C57BL Mice.** (Eng) Melief, C. J. (Central Lab. Netherlands Red Cross Blood Transfusion Service, P.O. Box 9190, Amsterdam, Netherlands); Vlug, A.; Barendsen, W.; De Bruyne, C.; Molenaar, J. L. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 78-83; 1978.

The expression of ecotropic C-type RNA viruses was studied in C57BL B10 and B10.A mice, which are genetically identical except for the major histocompatibility (H-2) complex. B10.A (H-2a) and B10 (H-2b) animals that received B-tropic virus through the milk of virus-positive B10.A mothers developed persistent virus infection. Genetic transmission, evidenced in part by the virus-negative status of nursing B10.A mothers, led to the heterogeneous expression of N- and B-tropic viruses in B10 and B10.A animals. This indicated that the mottled pattern for expression of ecotropic viruses in C57BL mice is controlled genetically. The H-2 complex influenced both splenic virus titers and antiviral antibody responses after milk transmission of B-tropic virus. The virus titers were higher in the B10.A animals, but the antibody titers were higher in the B10 animals. The milk-transmitted virus included lymphomas in 36% of 400 B10.A animals. (9 refs)

78-4528 **Large-Scale Isolation and Partial Purification of Type C RNA Viruses on Hydroxyapatite. Biochemical Characterization.** (Eng) Smith, R. G. (Lab. Tumor Cell Biology, NCI, Bethesda, MD, 20014); Lee, S. A. *Anal Biochem* 86(1): 252-263; 1978.

A chromatographic procedure based on the batchwise adsorption of C-type RNA viruses onto hydroxyapatite was developed for the concentration and partial purification of viruses from large volumes of tissue culture fluid (50 ml to 15 liters). The viruses obtained by this procedure have a buoyant density of 1.16 g/cm^3 and contain 70S RNA, an RNA-directed DNA polymerase (reverse transcriptase), surface glycoprotein (gp69/71), and the internal virus-specific polypeptides p10 to p15 and p27 or p30. The method was optimized for the isolation of biochemically pure virus and not for the preservation of biological activity. Th recovery of biological activity was generally below tha obtained by other small-scale methods, but it was con parable to the activity recovered by large-scale ultracei trifugation. The procedure is fast (20 to 50 liters of tissu culture fluid can be processed in 24 hr), and it uses biochemi cal reagents and equipment that are easily and economicall available. The procedure allows complete containment dur ing all stages of production, assuring sterility of the produc and safety for the worker. (33 refs)

78-4529 **C-Type Viral Immune Complexes in Leukemi and Systemic Lupus Erythematosus.** (Eng Panem, S. (Dept. Pathology, Univ. Chicago, 950 E. 59th St., Chicago, IL, 60637); Prochownik, E.; Reynolds, J.; Ordonez, N.; Slamon, D.; Kirsten, W. H. *In: Advances in Con parative Leukemia Research 1977. Proceedings of th VIIIth International Symposium on Comparative R search on Leukemia and Related Diseases, held i Amsterdam, The Netherlands on 22-26 August, 197* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New Yor Elsevier/North Holland Biomedical Press): 503 pp 50-52; 1977.

Immune response to C-type virus was investigated in patient with systemic lupus erythematosus (SLE) or hematologi neoplasms (acute leukemia, Hodgkin's disease, histiocyti lymphoma) using HEL-12 virus (isolated from human embryonic lung fibroblasts) as a probe. Virus-specific immun complexes (IC's) were found routinely in SLE kidneys an in 3/5 kidneys from the cancer patients. Circulating IgG which inhibits viral reverse transcriptases, were found frequently in the cancer patients, infrequently in normal subjects. (5 refs)

78-4530 **Activation of Endogenous Type-C Viral p30 An tigen in Chemically-induced Rat Hepatocellula Carcinomas.** (Eng) Becker, F. F. (Dept. Anatomic and Re Pathology, M. D. Anderson Hosp. and Tumor Inst., Hous ton, TX, 77030); Sherr, C. J. *Int J Cancer* 21(6): 756-76 1978.

A series of chemically induced rat primary hepatocellula carcinomas (PHC's) and several previously established trans plantable hepatocellular carcinoma (THC) lines were exam ined for expression of the major viral structural protein (p3 antigen) of endogenous rat C-type viruses. None of the si PHC's induced by exposure of male ACI rats t N-2-acetylaminofluorine expressed significantly elevated lev els of p30 antigen compared with levels in normal rat live Four PHC's gave rise to THC lines that were characterize by relatively rapid initial growth rates. For all THC lines, lo but significant levels of p30 antigen were detected after onl one transplant generation; subsequent transplants showed inreased levels. THC's derived from two PHC's were coculivated with indicator cell lines in an attempt to isolate relicating C-type virus. All indicator cell lines remained egative for reverse transcriptase activity, indicating the abence of detectable infectious C-type virus. Among six addiional chemically induced THC's, the slowly growing tumors, which tended to be more differentiated throughout their pasage in animals, failed to express elevated levels of viral antien, whereas the more rapidly growing tumors, which apeared more transformed, expressed high levels of ndogenous viral protein. The data suggest that p30 antigen xpression can be viewed as a phenotypic marker in chemically induced, rapidly growing hepatocellular malignancies of ats. (41 refs)

78-4531 **A Human Osteosarcoma Virus (Meeting Abstract).** (Fre) Prevost, J. M. (Lab. Microbiologie, nstitut J. Bordet, Universite de Bruxelles, Brussels, Belgim); Kettman, R.; Norman, C.; DeKegel, D.; Burny, A. *Bull Cancer (Paris)* 65(1): 38; 1978. (no refs)

78-4532 **Protein Metabolism in Human Skin-Muscle Embryo Cells Infected with Oncornavirus D rom J-96 Cells.** (Rus) Kishchak, V. Ya. (Central Inst. Adanced Training for Physicians, Moscow, USSR); Moisiadi, S. A.; Bektemirov, T. A. *Vopr Virusol* (4): 464-469; 1977.

To evaluate the effect of oncornavirus infection on the metabolism of cellular proteins, human skin-muscle embryo cells (HSMEC) were infected with oncornavirus D from J-96 cells. Spectra of the proteins of infected and control HSMEC cells were assessed by disk electrophoresis in polyacrylamide gel. Viral infection resulted in increased synthesis of high-mol-wt proteins and decreased synthesis of low-mol-wt proteins. Approx 90% of the proteins synthesized in the infected HSMEC were detected in the control cells and, thus, were identified as host proteins; 10% of the proteins were similar to those characteristic for J-96 cells. (16 refs.)

78-4533 **Small Virus-like Particles in Leukosis-like Syndrome Induced by Certain Antigens and Immunostimulators.** (Eng) Denner, J. (Dept. Virology, Central Inst. Cancer Res., Acad. Sciences GDR, 1115 Berlin-Buch, E. Germany); Dorfman, N. A. *Acta Biol Med Ger* 36(10): 1451-1458; 1977.

A leukosislike syndrome was induced in BALB/c mice by repeated injections of homologous or heterologous antigens plus an immunostimulator, dextran sulfate (DS) or complete Freund's adjuvant (CFA). The antigens consisted of plasma from mice with Rauscher leukemia or from healthy mice, proteins from these plasmas that were separated and bound to bovine γ-globulin, and crystalline ovalbumin. Five injections were given at 2-wk intervals. After the fifth injection, all mice that received CFA with and without antigens and DS with various antigens, but not mice that received DS alone or normal plasma alone, showed marked splenomegaly. The splenic architecture was distributed by myeloid cells of all developmental stages. The number of peroxidase-positive cells was elevated to 20%-50% of all WBC, compared with 11%-13% in normal spleen, and megakaryocytes and erythroblasts were also increased. These pathological changes were classified as leukosislike syndrome. The syndrome could be transmitted to adult BALB/c mice, mice of other strains (AKR, CC57Br, C57Bl/6), and randombred mice. Ultracentrifugation of the plasma of affected mice followed by electron microscopy of the high-speed pellets revealed the presence of viruslike particles with a diameter of 30-50 nanometers. The particles had two types of electron-dense nucleoids: ringlike or disklike, and they were surrounded by a unit membrane. Antigenic stimulation may result in activation of the virus, or an increase in certain cells may permit a virus that is present in minimal amounts to grow and multiply. (25 refs)

78-4534 **Histology of Benign Virus Induced Tumors of the Skin.** (Eng) Steigleder, G. K. (Dept. Dermatology, Univ. Cologne, Joseph-Stelzmann-Strasse 9, D-5000 Colonge 41, W. Germany). *J Cutan Pathol* 5(2): 45-52; 1978.

Histological sections of various benign, virus-induced skin tumors were studied and the findings reported. Particular attention was focused on inclusion warts, common warts, plane warts, filiform warts, focal epithelial hyperplasia, epidermodysplasia verruciformis, condylomata acuminata, and molluscum contagiosum. Electron microscopy of inclusion and other warts indicated that the viruses were present predominantly or exclusively in the nuclei of the wart cells. Transitions between common warts and condylomata acuminata have been observed, clinically and histologically, but it is not known whether these conditions are caused by the same virus. Because of its high mitosis rate, condyloma may be misdiagnosed as a carcinoma. Similarly, histological sections along the margin of a molluscum contagiosum may reveal a tumorlike proliferation similar to that of an epithelial tumor or a basal cell epithelioma. Epidermodysplasia verruciformis and condylomas, especially giant condylomas, may become carcinomatous in predisposed persons. (19 refs.)

78-4535 **Search for Oncogenic Viruses in Human Prostatic Cancer. Morphologic Studies.** (Eng)

Dmochowski, L. (Dept. Molecular Carcinogenesis and Virology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX); Ohtsuki, Y.; Seman, G.; Bowen, J. M.; Johnson, D. E. *Cancer Bull* 30(4): 112-115; 1978.

Studies attempting to establish a relationship between viruses and human prostatic carcinoma are reviewed. The observation in healthy mouse prostates of viruslike particles resembling virus found in solid tumors and leukemia of humans and of virus particles resembling those present in human breast cancer may indicate that viruses are involved in the origin of human prostatic carcinoma. (21 refs.)

78-4536 **New Virus Expressed in Cultured Human Leukaemic Myeloblasts (Letter to Editor).** (Eng) Karpas, A. (Dept. Haematological Medicine, Univ. Clinical Sch., Cambridge CB2 2QL, England). *Lancet* 2(8080): 110; 1978.

Ultrastructural examination of cultured myeloblasts from an acute myelomonocytic leukemia patient revealed numerous cytoplasmic inclusions containing 40- to 100-nanometer viruslike particles, often with an electron-lucent core and tail formation. Serological tests indicate that the virus differs from animal oncornaviruses, and the fact that antibodies could be found in some human sera supports the ultrastructural studies, which indicate that it might be a human virus. (7 refs)

78-4537 **Reduced Tumorigenicity by Addition In Vitro of Sendai Virus.** (Eng) Matsuya, Y. (Div. Microbiology, Res. Inst. Tuberculosis and Cancer, Tohoku Univ., Sendai 980, Japan); Kusano, T.; Endo, S.; Takahashi, N.; Yamane, I. *Eur J Cancer* 14(8): 837-850; 1978.

Treatment of Ehrlich ascites tumor (EAT) with Sendai virus, which has high cell-fusing activity on the viral envelope, produced polyploid variants that could grow on selective media, whereas no cell growth occurred in untreated control cultures. All of the variants isolated had more chromosomes and showed less transplantability than EAT. The banding patterns of each chromosome were analyzed. The karyotypes of the variants appeared to be more heterogeneous than EAT. Other biological properties, ie, agglutinability by concanavalin A and anchorage dependency of cell growth, proved to be unrelated to tumorigenicity. Similar variants were isolated by treating EAT with lysolecithin or polyethylene glycol, both of which are also cell-free fusion inducers. Like the variants induced by Sendai virus, they had more chromosomes and were less tumorigenic than EAT. However, the frequency of occurrence of these variants ($< 10^{-7}$) was far less than that (10^{-5}) of the variants induced by Sendai virus. It is suggested that variants preexisting in EAT begin to propagate in vitro after treatment of the tumor with fusion-inducing agents. (29 refs.)

78-4538 **Endogenous Cellular Fusion Induced In Vitro by the Visna Virus (Meeting Abstract).** (Fre) Pautrat, G. (Laboratoire de Virologie, C.H.U. Timone, 13385 Marseille Cedex 4, France); deMicco, P.; Benkoel, L.; Tamalet, J. *Bull Cancer (Paris)* 65(1): 32; 1978. (no refs)

78-4539 **Validation of Radioimmunoassays for Retrovirus.** (Eng) Charman, H. P. (NCI, Frederick, MD, 21501); Gilden, R. V. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp. 46-49; 1977.

Ongoing radioimmunoassay (RIA) studies of purified retrovirus proteins are summarized briefly, and some of the limitations of RIA are indicated. The specificity of competition RIA's is difficult to validate in crude extracts or partially purified systems. Extremes of pH, excessive protein concentrations, extremes of osmolarity, and ionic environments can interfere with antigen-antibody reactions and produce false-positive reactions. Proteolysis of labeled antigen or precipitating antibody during incubation may diminish precipitate radioactivity and apparent competition. (11 refs)

78-4540 **Site-directed Mutagenesis as a Tool in Genetics.** (Eng) Weissmann, G. (Institut fur Molekularbiologie I, Universitat Zurich, 8093 Zurich, Switzerland); Taniguchi, T.; Domingo, E.; Sabo, D.; Flavell, R. A. *Miami Winter Symp* 14: 11-36; 1977.

The infectivity of bacteriophage (Qβ) RNA was increased by substrate-controlled RNA synthesis and the introduction of a mutagenic nucleotide at a predetermined position of the viral RNA. The methodology of site-directed mutagenesis is described in detail, and its application to DNA plasmid is outlined. (27 refs)

See also:
*(Rev.): 78-4201, 78-4202, 78-4228.
*(Chem.): 78-4340, 78-4344.
*(Immun.): 78-4542, 78-4549, 78-4554, 78-4557
78-4560, 78-4567, 78-4571, 78-4575
78-4583, 78-4585, 78-4590, 78-4595
78-4596, 78-4600, 78-4605, 78-4607
78-4611, 78-4613, 78-4618, 78-4619
78-4620, 78-4621, 78-4623
*(Path.): 78-4627, 78-4646, 78-4647, 78-4660
78-4732.
*(Epid.-Biom.): 78-4751, 78-4752.

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78-4541 **Similarities Among Factors that Render Macrophages Tumoricidal in Lymphokine and Interferon Preparations.** (Eng) Schultz, R. M. (Lab. RNA Tumor Viruses, NCI, NIH, Bethesda, MD, 20014); Chirigos, M. A. *Cancer Res* 38(4): 1003-1007; 1978.

Lymphokine preparations, including supernatants derived from antigen-stimulated BCG-immune spleen cell cultures and normal (BALB/c x DBA/2)F_1 spleen cells incubated with insoluble concanavalin A (Con A), were compared with partially purified L-cell interferon for the ability to render resting macrophages nonspecifically tumoricidal in vitro. Significant activation of macrophages by lymphokine preparations occurred at concentrations as low as 0.5% and 0.25% of the assay mixture for antigen-stimulated and Con A-induced lymphokine, respectively. These end point concentrations were each determined to contain 0.3 unit of interferon/ml. Supernatants obtained from unstimulated normal spleen cells, Con A-treated nu/nu spleen cells, or BCG-immune spleen cells in the absence of sensitizing antigen did not enhance macrophage tumoricidal function and lacked interferon. Activation by L-cell interferon required at least 1 unit/ml. The macrophage activating factors contained in lymphokine and interferon preparations were stable at pH 2 and at 56 C, but they were destroyed when heated at 80 C for 30 min and they were inactivated by trypsin. (20 refs)

78-4542 **Alterations in Bone Marrow and Thymus Lymphocytes in Virus-inoculated, Inbred, Preleukemic AKR/J Mice: Brief Communication.** (Eng) Hays, E. F. (Lab. Nuclear Medicine and Radiation Biology, Univ. California at Los Angeles, 900 Veteran Ave., Los Angeles, CA, 90024); Haskett, D. R.; Kaspersky, C. J.; Craddock, C. G. *J Natl Cancer Inst* 60(4): 905-909; 1978.

The thymuses and bone marrows of 5- to 6-wk-old AKR/J mice superinfected with Gross murine leukemia virus (AKR-V) were compared with those of normal aged-matched controls (AKR-N). The AKR-V mice had marrow lymphopenia, a reduction of thymus wt and cellularity, and an increase in monocyte-granulocyte progenitor cells. The number of totipotent hematopoietic stem cells did not differ in the two groups of mice. The age-related changes in the marrow and thymus lymphocytes of AKR-V and AKR-N mice were studied by comparing 5- and 8-wk-old animals. In AKR-V mice, the normal age-related fall in marrow lymphocytes was blunted. This was due to a lesser decrease of T cells and null lymphocytes and an increase in immunoglobulin-bearing lymphocytes. The changes in thymus wt and cellularity between 5 and 8 wk in the AKR-V mice reflected the actual malignant transformation in the thymus. Alterations in marrow and thymus cellularity occurring before the appearance of overt malignant lymphoma were considered to be preleukemic changes. (12 refs)

78-4543 **Lymphopoietic Action of Vitamin A in Tumor-Bearing Mice (Meeting Abstract).** (Eng) Seifter, E. (Albert Einstein Coll. Medicine, Bronx, New York, NY, 10461); Barbul, A.; Levenson, S. M.; Rettura, G. *Fed Proc* 37(3): 335; 1978. (no refs)

78-4544 **Glutathione S-Transferase in Human Lymphoid Cell Lines and Fractionated Peripheral Leucocytes.** (Eng) Kaplowitz, N. (Medical and Res. Services, Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA, 90073); Spina, C.; Graham, M.; Kuhlenkamp, J. *Biochem J* 169(3): 465-470; 1978.

Glutathione S-transferase activity was identified in the cytosol from human lymphoid cell lines and peripheral WBC (polymorphonuclear-WBC/monocyte and small-lymphocyte fractions) and compared with that of human liver enzyme. The closely similar elution volumes in gel filtration and substrate (1-chloro-2,4-dinitrobenzene) and inhibitory (probenecid) kinetics indicate that the liver, WBC, and lymphoid cell transferases are closely related. The interaction of reduced glutathione and 1-chloro-2,4-dinitrobenzene occurred in intact lymphoid cell culture, was linear with time and quantity of cells, and had kinetics similar to those of the enzyme reaction catalyzed by cytosol. Peripheral WBC and B- and T-lymphoid cell lines can be added, therefore, to the list of cell types in which glutathione S-transferases and aryl hydrocarbon hydroxylase coexist. To evaluate cancer risk, consideration must be given not only to the activation of carcinogens by aryl hydrocarbon hydroxylase but also to the inactivation of these carcinogenic products by glutathione S-transferase. (27 refs)

78-4545 **Immunological Abnormalities in Children with Acute Leukemia.** (Jpn) Goya, N. (Dept. Pediatrics, Faculty Medicine, Kyushu Univ., Higashi-ku, Fukuoka 812, Japan); Miyazaki, S. *Acta Haematol Jpn* 40(6): 1042-1048; 1977.

Humoral and cellular immunity and neutrophil bactericidal activity were evaluated in 30 children with acute lymphoblastic leukemia (ALL) and 12 with acute myeloblastic leukemia (AML). Compared with controls, IgG was lower in ALL and higher in AML patients. No significant variation in IgA or IgM was found. The patients differed significantly from each other and from the controls in their reactions to all antigens tested, with the reactions of the AML patients being more depressed than those of the ALL patients. The DNCB test was also suppressed. Study of the lymphoblast surface markers revealed that 1/17 ALL patients was T-ALL and the others were null cell ALL. Blasts with C3b receptors were found in 1/10 AML patients. Serial studies of circulating T and B lymphocytes in ALL children showed a marked fall in the number of T cells in patients in remission. After cranial irradiation, there was a significant decline in B cells. Impaired lymphocyte transformation by stimulation with PHA was detected in both patient groups. In some cases, inhibition of transformation was observed when lymphocyte cultures contained sera from children with acute leukemia. No significant relationship between histocompatibility antigens and leukemia was found. Neutrophil function was almost normal in acute leukemia in remission. The values of CH50, C3, and C4 were also within the normal range. Of the 352 cases of primary immunodeficiencies registered in Japan over a 10-yr period, 8 patients developed malignancies, only one of which was acute leukemia. (25 refs)

78-4546 **Lymphocyte Responses to Phytohaemagglutinin in Patients with Asbestosis and Pleural Mesothelioma.** (Eng) Haslam, P. L. (Dept. Medicine, Cardiothoracic Inst., Brompton Hosp., London SW3, England); Lukoszek, A.; Merchant, J. A.; Turner-Warwick, M. *Clin Exp Immunol* 31(2): 178-188; 1978.

The association between depressed cellular immunity and asbestos-associated malignancy was investigated by comparing the lymphocyte responses of 28 asbestosis patients with associated pulmonary fibrosis, 1 patient with asbestosis and lung cancer, 10 patients with pleural mesothelioma (8 with a history of asbestos exposure), and 36 nonasbestos-exposed controls to phytohemagglutinin (PHA). Quantitative impairment of lymphocyte responses was noted in 6/28 asbestosis patients. The impairment tended to occur in patients with fairly severe fibrosis, comparatively short duration of exposure to asbestos dust, and increased serum immunoglobulin (Ig) levels. The one patient with asbestosis and bronchial carcinoma also had depressed lymphocyte responses to PHA. However, 8/10 patients with asbestos-associated pleural mesothelioma without lung fibrosis showed no evidence of impaired cellular immunity, either by in vitro testing with PHA or by in vivo delayed hypersensitivity skin testing. In this group also, patients with impaired PHA responses had increased serum Ig levels. These findings suggest that there is a relationship between defective T-cell function and the fibrotic response in asbestosis, but that this finding is not common to all types of asbestos-associated malignancy. (40 refs)

78-4547 **Macrophage Activation for Tumor Cytotoxi
ty: Increased Lymphokine Responsiveness
Peritoneal Macrophages During Acute Inflammation.** (E
Ruco, L. P. (Immunopathology Section, Lab. Immunobio
gy, US Dept. Health, Education, and Welfare, NCI, N
Bethesda, MD, 20014); Meltzer, M. S. *J Immunol* 120(
1054-1062; 1978.

The responses of peritoneal macrophages from resident (
and inflammatory (I) populations to lymphokine (LK) a
vation were compared and analyzed. The macrophages w
collected from untreated C3H/HeN mice and from m
treated ip 1 day previously with fetal calf serum or 5 d
previously with latex beads, 3% thioglycollate, or a 2
starch suspension. Equal macrophage concentrations fr
each group of mice were cultured for 2-3 hr. Adherent I
were incubated in dilutions of LK supernatant (from purif
protein derivative of tuberculin-stimulated BCG-immu
spleen cell cultures) for 4 hr, the cultures were washed, a
^{3}H-thymidine-labeled cells from tumor 1023
methylcholanthrene-induced) were added to the monolaye
Tumor cytotoxicity by LK-activated I macrophages v
about 10 times greater than that by equal numbers of L
treated R cells. Comparisons of the time course of activati
and of the LK dose response showed that the increased
sponsiveness of the I cells was due to quantitative chang
The numbers of responsive I cells were increased 5-10 tin
with inflammation. These changes coincided with simi
changes in the numbers of peroxidase-positive (PP) mac
phages. Thus, the increased LK responsiveness of I cells
pended on the influx of young PP mononuclear phagocy
rather than the stimulation of R macrophages. This conc
was supported by the finding that whole-body x-irradiati
reduced the numbers of PP and of LK-responsive mac
phages in I- and R-cell populations. The LK-responsive p
cursor for the activated tumoricidal macrophage during b
steady-state and acute inflammatory conditions was t
newly formed blood-derived mononuclear phagocyte. T
tenfold increase in the number of LK responsive mac
phages in the peritoneal exudate over R-cell populations
flected a similar increase in macrophage turnover induced
inflammation. (38 refs)

78-4548 **Effect of Inoculation with Neuraminidase-tre
ed Tumor Cells on Macrophage Cytotoxicity
Vitro.** (Eng) Alley, C. D. (Dept. Anatomy, Medical C
Virginia, Virginia Commonwealth Univ., Richmond, V
23298); Snodgrass, M. J. *Cancer Res* 38(8): 2332-2338; 19

Adherent peritoneal macrophages (PM) and macrophag
isolated from tumor tissue of C57BL/6J x DBA/2J F_1 mi
inoculated with tumor cells treated with *Vibrio choler*
neuraminidase (VCN) and mitomycin C were studied f
their cytotoxic effect on established tumor cell monolay
by time-lapse cinematography. The two murine pulmona
tumors studied were the spontaneous Lewis Lung tumor a

3-methylcholanthrene-induced squamous cell carcinoma. Tumor cells incubated alone, with PM from untreated mice, or with macrophages isolated from tumor tissue of untreated or treated mice grew to confluence by 24 hr of coincubation and displayed no detrimental change by 96 hr. Cytotoxic PM were isolated from tumor-bearing mice that had received an sc inoculation of tumor cells treated with both VCN and mitomycin C and from tumor-free animals (1) inoculated ip with tumor cells treated simultaneously with VCN and mitomycin C or (2) given tumor cells treated with mitomycin C alone. PM from mice inoculated within 3 days of isolation with treated tumor cells killed cultures of the identical tumor by 36 hr, but they did not kill cultures of another tumor until 48 hr of coincubation. If PM were isolated 18 days following the final inoculation of treated tumor cells, they were cytotoxic (at 36 hr) to cells of that tumor only. PM from mice treated with BCG or BCG cell walls killed cultured tumor cells by 48 hr. It is concluded that specifically cytotoxic macrophages kill tumor cells by 36 hr, but nonspecifically cytotoxic macrophages do not kill until 48 hr of coincubation. (42 refs)

78-4549 **Defective Macrophage Immunity in Newborn Rabbits with Fibroma Virus-induced Tumors.** (Eng) Tompkins, W. A. (Center Zoonoses and Comparative Medicine, Coll. Veterinary Medicine, Univ. Illinois, Urbana, IL, 61801); RamaRao, G. V. *J Reticuloendothel Soc* 23(2): 161-166; 1978.

Macrophage immunity was investigated in young adult (3- to 4-mo-old) and newborn (2-day-old) rabbits inoculated with 2.5 x 10^5 plaque-forming units (PFU) of the Patuxent strain of fibroma virus id and sc, respectively. Cultured peritoneal macrophages collected from the rabbits 10 days after fibroma virus inoculation failed to show infectious virus. Vaccinia virus replicated in cultured macrophages from both normal adult and newborn rabbits, resulting in an increase in titer of 2.1 log 10 and 2.4 log 10 PFU/ml, respectively. However, vaccinia virus failed to increase in titer in macrophages from adult tumor-bearing rabbits. It did increase by 2.0 log 10 PFU/ml in macrophages from tumor-bearing newborns. Adult rabbits expressed macrophage immunity to vaccinia between 9 and 21 days after infection. Before and after this time, the macrophages supported vaccinia replication and behaved like those from normal rabbits. In contrast, macrophages from newborn tumor-bearing rabbits had no immunity by day 13 and only partial immunity before 21 days. Max macrophage immunity occurred between days 21 and 41, after which they returned to normal. (8 refs)

78-4550 **Teratocarcinoma Cell Variants Rejected by Syngeneic Mice: Protection of Mice Immunized with These Variants Against Other Variants and Against the Original Malignant Cell Line.** (Eng) Boon, T. (Cellular Genetics Unit, International Inst. Cellular and Molecular Pathology, Avenue Hippocrate, 74, B-1200 Brussels, Belgium); Van Pel, A. *Proc Natl Acad Sci USA* 75(3): 1519-1523; 1978.

The antigenic specificities of nontumorigenic (tum-) teratocarcinoma (TC) cell variants were investigated. The variants, which were obtained by treatment of a malignant TC cell line with N-methyl-N′-nitro-N-nitrosoguanidine, induce a radioresistant immune memory in syngeneic 129/Sv mice. Three weeks after immunization with 2 x 10^6 living tum- cells, the mice were given 600 rads of gamma radiation and then challenged with cells from the same clone or one of three other clones. For a given challenging clone (1 x 10^6 or 2 x 10^6 cells), immunization with a different clone conferred some protection (cross-protection), but this protection was invariably weaker than that conferred by immunization with the same variant (self-protection). Mice inoculated with 3 x 10^6 tum- cells were partially protected against challenge with 5 x 10^5 living tum+ cells, and this protection was not abolished by irradiation. In contrast, tum+ cells killed by irradiation did not elicit a rejection response. These results indicate that each tum- variant carries at least one specific transplantation antigen that is not present on the original TC line or most other variants. However, one or more common antigenic specificities are probably shared by all the tum- variants and the tum+ cells. (16 refs)

78-4551 **Genetic Dissection of the Control of Normal Differentiation in Myeloid Leukemic Cells.** (Eng) Lotem, J. (Dept. Genetics, Weizmann Inst. Science, Rehovot, Israel); Sachs, L. *Proc Natl Acad Sci USA* 74(12): 5554-5558; 1977.

Controls that regulate the induction of normal differentiation in myeloid leukemic cells were investigated using mutants that differ in the timing and degree of inducibility of differentiation-associated properties by two different inducers, the normal regulatory protein MGI and a steroid inducer (SI), dexamethasone. The sequence of differentiation was the induction of C3 and Fc rosettes, C3 and Fc immune phagocytosis (IP), synthesis and secretion of lysozyme, and formation of mature macrophages and granulocytes. Mutant clones of myeloid leukemic cells were isolated with differences in the time of induction of C3 and Fc rosettes and C3 and Fc IP (in which lysozyme was induced without going through Fc or C3 IP) and with differences in inducibility by MGI to mature macrophages or granulocytes. Only 1/5 MGI-D- clones (could not be induced by MGI for any of the properties being studied) gave rise to MGI^+D^+ mutants (clones that could be induced by MGI for differentiation-associated markers, including mature cells). The ability to obtain mutants from this clone was associated with its special chromosome constitution. Dexamethasone could induce in MGI^+D^+ clones differentiation to macrophages but not to granulocytes. Differentiation by dexamethasone in different clones occurred either with or without induction of Fc rosettes and Fc IP, and induction of C3 rosettes was not always associated with induction of C3 IP. The results indicate that there are separate controls for the induction of C3 rosettes, Fc rosettes, C3 IP, Fc IP, lysozyme, mature macrophages, and mature granulocytes. Differences in inducibility of these properties by MGI and SI support the suggestion that MGI and SI have different cellular induction sites. (35 refs.)

78-4552 **Immunologic Characterization of Hairy Cell Leukemias in Continuous Culture.** (Eng) Saxon, A. (Dept. Medicine, Div. Clinical Immunology and Allergy, Univ. California Los Angeles Sch. Medicine, Los Angeles, CA, 90024); Stevens, R. H.; Quan, S. G.; Golde, D. W. *J Immunol* 120(3): 777-782; 1978.

The immunobiologic characteristics of three continuous cell lines established from patients with hairy cell leukemia were investigated. Two of these lines were of B-cell origin, and they were established from peripheral blood samples obtained during the leukemic phase of the disease. The third was of T-cell origin and was established from spleen cells obtained at splenectomy. All lines continued to produce tartrate-resistant acid phosphatase. The B-cell lines carried Fc and C receptors, had surface and internal immunoglobulin, and did not form spontaneous sheep RBC rosettes. One of these lines has remained relatively constant in the amount of IgM produced total protein, while the other has switched immunoglobulin production from IgG to IgM. The T-cell line formed spontaneous sheep RBC rosettes and did not carry Fc or C receptors; the rosette-forming cells contained tartrate-resistant acid phosphatase. They did not possess surface or internal immunoglobulin and did not synthesize immunoglobulin in vitro. Since these hairy cell leukemia cells retain their immunobiologic properties in culture, they can be used to study this disease. (25 refs)

78-4553 **Isolation and Partial Characterization of an Antibody Enhancing Factor from Leukaemic Owl Monkey Cell Cultures.** (Eng) MacDonald, A. B. (Dept. Microbiology, Harvard Univ. Sch. Public Health, Boston, MA, 02115); Fraser, C. E.; Rubin, A. S. *Immunology* 34(1): 137-147; 1978.

The propagation of lymphoid cells from leukemic owl monkeys (*Aotus trivirgatus*) in culture, the release of a soluble factor that amplifies antibody synthesis from these cells, and the partial characterization of this mediator are described. Peripheral lymphocytes from monkeys inoculated with *Herpesvirus saimiri* and grown in culture were predominantly T cells. The supernatants from these cultures contained a factor that enhanced the antibody response of murine B cells to sheep RBC. The greatest degree of augmentation was obtained when active supernatants were added to assay cultures at 1:100 final dilution. The nonspecific stimulatory capacity at this dilution was higher than any previously reported for
similar supernatant activities recovered from appropriatel
stimulated murine or human lymphoid lines. The factor wa
found to be a protein with a mol wt of approx 40,000, an
it was not significantly affected by DNase or RNase. Trea
ment of human peripheral WBC with the factor did not pro
duce any mitogenic activity. (20 refs)

78-4554 **Lysis of Oncornaviruses by Human Serum. Is
lation of the Viral Complement (C1) Recept
and Identification as p15E.** (Eng) Bartholomew, R. M
(Dept. Molecular Immunology, Res. Inst. Scripps Clinic, L
Jolla, CA, 92037); Esser, A. F.; Muller-Eberhard, H. J. *J Ex
Med* 147(3): 844-853; 1978.

The capacity of Moloney leukemia virus to activate huma
complement was investigated. The virus activated both th
classical and alternative pathways of human complemen
about 500,000 virions were required to detect activation
the classical pathway, but 5,000 times as many virions we
needed to initiate the alternative pathway. Thus in this sy
tem, only the former pathway is of biological significanc
Disruption of the virions with Triton X-100 destroyed the
ability to initiate the alternative pathway without affectin
their ability to activate the classical pathway. Following u
tracentrifugation of the disrupted virus, the active compone
was recovered and isolated. The activity was found to resid
in a methionine-containing protein with an isoelectric poi
of 7.5 and a mol wt of 15,000 daltons. The purified prote
interacted strongly with C1q and efficiently activated C
RNase and lipolytic enzymes had no effect on the isolat
protein, but incubation with trypsin abrogated its activit
Enzymatic digestion studies indicated that the active prote
is a viral membrane protein. It is concluded that the compl
ment receptor of Moloney leukemia virus is the surface pr
tein p15E. (22 refs)

78-4555 **Immunogenetic Determinants of Familial Acut
Lymphocytic Leukemia.** (Eng) Blattner, W. A
(3C-07 Landow Building, NCI, NIH, Bethesda, MD, 20014)
Naiman, J. L.; Mann, D. L.; Wimer, R. S.; Dean, J. H.
Fraumeni, J. F. *Ann Intern Med* 89(2): 173-176; 1978.

Acute lymphocytic leukemia developed almost simultaneous
ly (within 3 wk) in two adolescent brothers, and anothe
brother and both parents had rheumatoid arthritis. Laborato
ry studies uncovered no evidence for an underlying im
munodeficiency state in the family. Immunogenetic evalua
tion showed the leukemic siblings to be histocompatibilit
antigen- and mixed leukocyte culture-identical and homozy
gous for a recessively inherited locus dictating the presenc
of antigens on the surface of B cells. These Ia antigens, a
detected by sera from mothers of leukemic children, appeare
to be mapped within the major histocompatibility region
They may be a human analog to murine immune-respons
antigens associated with susceptibility to leukemia. (23 refs.

8-4556 **HLA Antigens and Haplotypes in Acute Leukemia.** (Eng) Albert, E. D. (Tissue Typing Lab.,
inderpoliklinik der Universitat, Pettenkoferstrasse 8a, 8
unich 2, W. Germany); Nisperos, B.; Thomas, E. D. *Leuk*
es 1(4): 261-269; 1977.

he histocompatibility (HLA) antigen and haplotype fre-
uencies were determined in 198 patients with acute lympho-
lastic leukemia (ALL) and 179 patients with acute myeloge-
ous leukemia (AML) to see if there is an association between
ILA and leukemia. The findings were compared with those
f a control population of 1,070 American Caucasians. Only
ILA-A2 and A9 in patients with ALL showed an increase
ver that expected. Genotype testing indicated an increase
the gene frequencies of HLA-A2 and A9 corresponding
an increase in phenotypic expression. The gene and hap-
otype frequencies in AML were essentially normal. A study
f the patients for HLA homozygosity indicated that the fre-
uency of HLA-A homozygotes in ALL was lower than that
xpected; the frequency of HLA-A homozygotes in AML and
ILA-B homozygotes in both diseases did not differ from that
xpected. Furthermore, there was no significant excess of
amilies sharing an HLA-A,B haplotype in either disease.
egregation analysis revealed a significant predominance of
ILA-A2 in ALL patients but normal results in healthy sib-
ings. Studies of the frequency of HLA antigens in relation
o the elapsed time between diagnosis and typing indicated
hat patients with the highest frequency of HLA-A2 and A9
lso had the longest time span between diagnosis and typing.
22 refs)

8-4557 **Resistance to Syngeneic Lymphoma Cells as a Result of Immunization with Chemically Modi-**
ied Allogeneic Lymphoma Cells in Mice. (Eng) Gillette, R.
V. (Basic Science Unit, Cancer Center Hawaii, Univ. Hawaii
t Manoa, 1997 East-West Road, Honolulu, HI, 96822); Ber-
inger, D. C.; Wunderlich, D. A. *J Natl Cancer Inst* 60(6):
427-1432; 1978.

The effect of immunization of mice with trinitrophenyl
TNP)-derivatized allogeneic lymphoma cells on the develop-
nent of a syngeneic lymphoma was determined. This immu-
ization resulted in the development of significant immunity
o poorly immunogenic syngeneic lymphoma cells. Neither
TNP-treated nor x-irradiated syngeneic lymphoma cells were
mmunogenic under similar conditions. Immunization with
ntreated allogeneic lymphoma cells produced only minimal
evels of resistance to challenge with syngeneic lymphoma
ells. Immunization with allogeneic TNP-derivatized lymph
ode and thymus cells also did not confer any resistance,
ndicating that the immunity was carried exclusively on the
ransformed lymphocytes. The immunity was transferred to
onimmune inbred BALB/c and A/J mice by spleen cells
rom immune donors. When immune spleen cell-lymphoma
nixtures were injected sc at a ratio of 1,000:1, nonimmune
nice were completely protected. Immunization with
TNP-derivatized allogeneic lymphoma cells resulted in resistance to inocula of viable syngeneic tumor cells 100 times greater than the tumorigenic dose. Induction of immunity to syngeneic lymphoma cells required that the immunizing cells be histoincompatible at the major *H-2* locus and possess tumor-specific antigen(s). Max immune sensitivity was observed only after chemical modification of the immunizing allogeneic lymphoma cells. (40 refs)

78-4558 **Malignant Lymphoma Arising in Angioimmunoblastic Lymphadenopathy.** (Eng) Nathwani, B. N. (Dept. Anatomic Pathology, City Hope Natl. Medical Center, 1500 E. Duarte Road, Duarte, CA, 91010); Rappaport, H.; Moran, E. M.; Pangalis, G. A.; Kim, H. *Cancer* 41(2): 578-606; 1978.

Histopathologic and clinical data are presented for 48 patients with angioimmunoblastic lymphadenopathy (AILD) and 36 with AILD + immunoblastic lymphoma (IL) in the diagnostic biopsy. In the first group, progression to IL was observed in 8/23 patients with AILD in whom follow-up biopsies or autopsy was performed. Multiple clusters or islands of compactly arranged large lymphoid cells constituted the initial histologic evidence of IL. Subsequent tissue examination revealed progression of the disease in the form of diffuse replacement of lymph nodes by the neoplastic cellular proliferation. There were no significant differences in the past history, clinical, or laboratory findings between the AILD patients and those with AILD + IL. These two groups differed greatly, however, with respect to complete remission following prednisone and/or chemotherapy (63% for AILD vs 26% for AILD + IL); median survival (35 mo vs 6 mo); incidence of malignant lymphoma at autopsy (20% vs 82%); and the finding of extranodal malignant lymphoma at autopsy (10% vs 64%). In the AILD group, the median survival of patients who had complete remission after prednisone was significantly longer than that of patients who had partial or no remissions; the same was true for patients given chemotherapy. In the AILD + IL group, a similar difference in the median survival was observed in patients treated with chemotherapy, but not in those treated with prednisone. (61 refs)

78-4559 **Prolonged Lifespan and High Incidence of Neoplasms in NZB/NZW Mice Treated with Hydrocortisone Sodium Succinate.** (Eng) Walker, S. E. (R4633 Kresge I, 1405 East Ann St., Ann Arbor, MI, 48109); Anver, M. R.; Schechter, S. L.; Bole, G. G. *Kidney Int* 14(2): 151-157; 1978.

The effectiveness of prolonged hydrocortisone sodium succinate (HCSS) therapy (3.3 or 10 mg/kg/day, sc) in preventing early death from renal disease was studied in young female hybrid New Zealand Black/New Zealand White mice. Control mice were given sc saline. Long-term therapy with either

HCSS dose effectively prevented renal disease and prolonged the lives of the mice. Fifty-six percent of the animals given the lower dose and 76% of those given the higher dose developed neoplasms. The most common neoplasms were sarcomas, which appeared in aged animals and tended to involve the reproductive tract. Lymphoreticular malignancies developed in 6/22 animals with tumors. The data support the conclusion that neoplasia is an important consequence of long-term treatment of autoimmune disease in hybrid New Zealand mice. (25 refs.)

78-4560 **Ultrastructural Studies on Hydantoin Induced Lymphomas in Mice.** (Eng) Bedoya, V. (Univ. Antioquia Medical Sch., Medellin, Colombia, South America); Krueger, G. R. *Z Krebsforsch* 91(2): 195-204; 1978.

Ultrastructural studies were done on cells from original tumors and from short term cultures of mouse thymic lymphomas experimentally induced by chronic exposure to diphenyl-hydantoin. The tumors appeared in mouse strains with low (C57Bl) and high (SJL/J) susceptibility to spontaneous lymphoma development and were not observed in a resistant strain (C3Hf). Thymic lymphoma development was usually preceded by increasing lymphoreticular atrophy followed by progressive reticulohistiocytic hyperplasia, and subsequently spread to other tissues. Morphologically the tumor was characterized as a lymphoblastic lymphoma. Abnormal cell changes in the original tumors and cultured cells, and the presence of murine C-type particles in the cultured cells but not in the original tumors, are discussed in relation to the disturbance of the immune system and the lymphoma enhancement produced by diphenyl-hydantoin. (25 refs)

78-4561 **Effects of Light In Vivo on Mouse Lymphoma Cells Pretreated In Vitro with Hematoporphyrin (Meeting Abstract).** (Eng) Coppola, A. (Downstate Medical Center, Brooklyn, NY); Viggiani, E.; Salzarulo, L.; Rasile, G. *Fed Proc* 37(3): 413; 1978. (no refs)

78-4562 **Growth Inhibition of Normal or Drug-treated Lymphoma Cells in Lethally Irradiated Mice.** (Eng) Riccardi, C. (Sch. Medicine, Inst. Pharmacology, Univ. Perugia, Perugia 06100, Italy); Fioretti, M. C.; Giampietri, A.; Puccetti, P.; Goldin, A.; Bonmassar, E. *J Natl Cancer Inst* 60(5): 1083-1090; 1978.

The growth inhibition of LSTRA and L5178Y lymphomas, which carry the *H-2d*, or 5-(3,3-dimethyl-1-triazenyl)-1H-imidazole-4-carboxamide (-DTIC)-modified lymphoma sublines was studied in lethally irradiated (850-900 rads) mice. Graded doses of lymphoma ce were injected iv into histocompatible or allogeneic mi that were either susceptible or resistant to *H-2d* bo marrow grafts according to their hematopoietic-hist compatibility (*Hh*) system. Radioresistant inhibiti of tumor (RIT) responses were determined by measu ing the extent of ^{125}I-2′-deoxyuridine uptake in t spleens and livers of recipient mice. RIT respon were detected against both parental and DTIC-treat sublines in *Hh*-resistant or *Hh*-susceptible allogen hosts. Marginal or no RIT responses against *H-* LSTRA or L5178Y lymphomas were found in inbred or h brid hosts carrying the *H-2d* haplotype. Nevertheless, we but significant responses were detected in recipients of t same genotype challenged with DTIC sublines. The resu of the experiments suggested that: (1) parental DTIC-treated lymphomas could share *Hh* antigens wi normal bone marrow cells of the strain of orig although no direct evidence was obtained in the studies; (2) leukemia cells might express tumor-associat antigens, not detectable in normal bone marrow ce that are capable of eliciting RIT responses in *Hh*-susce tible allogeneic mice; and (3) additional transplantati antigens could be expressed by DTIC sublines th elicited presumably weak RIT responses in letha irradiated histocompatible recipient hosts. (38 ref

78-4563 **Enhancement of Natural and Antibody-Depen ent Lymphocyte Cytotoxicity by Drugs Whi Inhibit Prostaglandin Production by Tumor Target Cel** (Eng) Droller, M. J. (Dept. Urology, Johns Hopkins Hos Baltimore, MD); Perlmann, P.; Schneider, M. U. *Cell I munol* 39(1): 154-164; 1978.

A ^{51}Cr release assay was used to examine the effects of pros glandin inhibitors on natural and antibody-depende cell-mediated cytotoxicity against T_{24} cells, (derived fro bladder transitional tumor, and HCV_{29} cells, derived fro normal bladder transitional epithelium but probably tra formed in tissue culture. Cells labeled with ^{51}Cr were incub ed with lymphocytes from venous blood of normal dono prostaglandin inhibitors [indomethacin (Indo: 10^{-9}-10^{6}M acetylsalicyclic acid (10^{-7}-10^{-5}M), the sodium salt of fenclo acid (10^{-7}-10^{-5}M), and 2,6-xylenol (10^{-7}-10^{-5}M), a T_{24} antiserum for 16-20 hr at 37 C. All four dru enhanced natural and antibody-dependent cell-mediat cytotoxicity against T_{24} and HCV_{29}, generally in dose-dependent manner. The presence of antiseru with or without the inhibitory agents, did not enhan ^{51}Cr-release, nor was enhancement seen when t agents were incubated with target cells in the absen of lymphocytes. Time course studies indicated that t longer the inhibitors were present during incubation, t greater the enhancement of lymphocyte cytotoxicity. The a dition of exogenous prostaglandins (10^{-8}-10^{-7}M) to the inc bation tubes prevented the Indo-induced enhancement cytotoxicity. These results support the suggestion that tum

:lls protect themselves against cytotoxic lymphocytes by ·oducing inhibitory levels of prostaglandins. (14 refs.)

8-4564 **Enhancement of Heterotransplanted Human Tumor Graft Survival in Nude Mice Treated ith Antilymphocyte Serum and in Congenitally Athym- -Asplenic (lasat) Mice.** (Eng) Gershwin, M. E. (Dept. ledicine, Sch. Medicine, Univ. California, Davis, CA, 5616); Ikeda, R. M.; Erickson, K.; Owens, R. *J Natl Cancer nst* 61(1): 245-248; 1978.

leterotransplanted human tumor graft survival was studied 1 unmanipulated and antilymphocyte serum (ALS)-treated ongenitally athymic (nude) mice and in congenitally athym- :-asplenic (lasat) mice to investigate the relationship between he immune status of nude mice and the transplantation of uman tumor lines. The 11 tumor lines used were taken from breast adenocarcinoma, bladder transitional cell carcinoma, steosarcoma, fibrosarcoma, Hodgkin's disease, malignant nelanoma, and rhabdomyosarcoma. Only 3/11 tumor lines vere successfully transplanted in unmanipulated nude mice, ompared with 5/10 tumor lines in ALS-treated nude mice nd 9/11 lines (except for the transitional cell carcinoma and habdomyosarcoma lines) in lasat mice. Moreover, the laten- y period was shorter and the minimum cell inoculum size vas lower for lasat mice than for nude or ALS-treated nude nice. In no experimental situation was there evidence of tu- nor metastasis. It is concluded that tumor grafting occurs nore readily in immunologically deprived nude mice, and it s suggested that both major immunologic and nonim- nunologic events are critical in the balance between success- ul xenografting and rejection and between local tumor pro- luction and metastases. (31 refs)

78-4565 **Growth Response in Athymic Nude Mice to Transplanted Growth Hormone-secreting Rat Pituitary Tumor Cells.** (Eng) Shin, S. (Dept. Genetics, Albert Einstein Coll. Medicine, 1300 Morris Park Ave., Bronx, NY, 10461); Brown, A. L.; Bancroft, F. C. *Endocrinology* 103(1): 223-228; 1978.

Clonal cell lines derived from a somatotropic rat pituitary umor were grafted sc into athymic nude mice, and the growth response induced in the graft carriers was examined. When at least 10^6 rat tumor cells were injected per mouse, virtually all mice developed slow-growing, nonmetastasizing umors at the injection site. In immature nude mice, the somatotropic effect of the rat growth hormone (GH) secreted by the growing tumor was clearly evident, as body wt con- inued to increase well beyond that of control litter- mates (760 g, compared with 32 g). The action of he rat GH was generalized, since even the largest mice etained apparently normal physical proportions. The slets of Langerhans in the host pancreas was especially hypertrophic, presumably due to the anti-insulin activity of GH. The rat pituitary tumor cells synthesized and secreted a protein that was precipitated by rat GH antiserum and that comigrated with authentic rat GH when analyzed by sodium dodecyl sulfate-acrylamide electrophoresis. Through serial transplantation in nude mice, the tumor cells retained their functional and tumorigenic properties. These results demonstrate that rat GH is active in the mouse and they suggest that the athymic nude mouse should be useful for the development of other xenogenic tumor lines that secrete GH. (23 refs)

78-4566 **Association Between Tumorigenesis and the State of the Immunocompetent System in Mice.** (Pol) Neiman, I. M. (Inst. Nutrition, Moscow, USSR); Finogenova, M. A. *Patol Fiziol Eksp Ter* (2): 26-29; 1978.

The effect of stimulation of connective tissue with antilymphocytic serum (ALS) on methylcholanthrene (MC)-induced carcinogenesis was studied in CBA x C57BL mice. ALS administration (0.05 ml, sc) 48 hr before MC injection (0.04 ml of 0.5% soln, sc) inhibited the development of MC-induced skin tumors: 22 wk after MC administration, 60% of the ALS-treated mice developed papillomas, compared with 85% of the controls. Malignant transformation of the papillomas occurred in only 16% of the ALS-treated mice, vs 36% of the controls. (17 refs)

78-4567 **A Quantitative Micro-complement Fixation Method in Studies of Human Wart Viruses.** (Eng) Hefton, J. M. (Lab. Human Tumor Virology, Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Eisinger, M. *J Gen Virol* 38(1): 179-182; 1977.

Microcomplement fixation was used to detect the reactions between human hand wart virus antigens and monospecific rabbit antiserum directed against human wart virions. The antigen titers obtained from the count analysis of the microtest were constantly four to eight times higher than those obtained from a visual analysis of microtest RBC pellets. The monospecific antisera did not react with other papovaviruses such as simian virus 40, BK, or JC virus, and all reactivity could be removed by absorption with human wart virus but not with normal skin. It is concluded that the reactions were specifically between human hand wart viruses and antibodies directed against them. This test can be used to study the roles of these viruses in human neoplasia. (9 refs.)

78-4568 **Tumor Rejection Antigens in Subcellular Fractions Isolated from an Ultraviolet Light (UV) Induced Murine Skin Tumor (Meeting Abstract).** (Eng) Otterstrom, J. R. (Hershey Medical Center, Hershey, PA, 17033); Schengrund, C. L.; Bartlett, G. L. *Fed Proc* 37(6): 1562; 1978. (no refs)

78-4569 **Enhancement of Experimental Lung Metastases by Cultured B16 Melanoma Cells Treated with (±)-1,2-Bis(3,5-dioxopiperazin -1-yl)propane(ICRF-159).** (Eng) Lazo, J. S. (Dept. Pharmacology, Comprehensive Cancer Center, Yale Univ. Sch. Medicine, New Haven, CT, 06510); Ingber, D. E.; Sartorelli, A. C. *Cancer Res* 38(8): 2263-2270; 1978.

The in vitro colony-forming (CF) efficiency of exponentially growing B16 melanoma cells was significantly decreased after they were exposed to ≥ 20 μM (±)-1,2-bis(3,5-dioxopiperazin-1-yl)propane (ICRF-159). Under identical conditions, ICRF-159 significantly increased the in vivo lung CF efficiency of these cells. Thus, 24 hr after 20 μM ICRF-159, the ability of melanoma cells to form colonies in vitro was reduced by 89%, although the lung CF efficiency of these same cells was enhanced 5.5-fold after tail vein injection into syngeneic mice. The ratio of in vivo to in vitro CF efficiency of cells treated with 20 μM ICRF-159 for 24 or 48 hr was 50- to 110-fold higher than that seen with untreated cells. Mice inoculated sc with ICRF-159-treated cells developed tumors later and survived longer than mice that received untreated control cells. The increase in tumor latent period and mean survival time was consistent with a 90% decrease in cell viability observed with the in vitro CF assay. The enhanced ability of B16 cells to form lung colonies after ICRF-159 treatment appears to be due to a drug-induced change in the cells, rather than to the presence in the cell population of a mixture of dead and viable cells. Cell volume and the incorporation of precursors of glycosaminoglycans were increased after ICRF-159 treatment, and this may be responsible for the enhanced lung CF efficiency. The findings suggest that ICRF-159 may increase the ability of circulating melanoma cells to implant and grow in susceptible distant organs. Since circulating tumor cells are often found in patients with solid tumors, the metastatic potential of these cells may be enhanced by only partially effective chemotherapy. (26 refs)

78-4570 **Squamous-Cell Carcinoma of the Floor of the Mouth.** (Eng) Ballard, B. R. (Dept. Pathology, Meharry Medical Coll., 1005 18th Ave., N., Nashville, TN, 37208); Suess, G. R.; Pickren, J. W.; Greene, G. W.; Shedd, D. P. *Oral Surg* 45(4): 568-579; 1978.

The clinical and biologic behavior of squamous cell carcinoma of the floor of the mouth was studied in 100 patients (81 men and 19 women). Ninety-four were Whites and six were Blacks; the age range was 33-85 yr, with an av of 63 yr. The peak incidence in women was in the 50 to 59 yr age group; in men, there was equal frequency in each age group > 50 yr. A total of 43/89 patients diagnosed within 1 yr of the onset of symptoms had regional lymph node metastases. The 5-yr survival rate decreased from 86% to 0% as the stage of disease progressed from I to IV. Thirty-three patients developed a total of 83 other primary cancers, and 72 of these arose in the upper alimentary and respiratory tracts. The oral carcinomas were classified as follows: well-differentiated (49), well- to moderately differentiated (7), moderately differentiated (20), moderately to poorly differentiated (10), poorly differentiated (2), and unspecified (12). Fifty-eight patients were heavy smokers, 33 were heavy drinkers, 28 were both heavy smokers and heavy drinkers, and 21 were nonsmokers and nondrinkers. (48 refs)

78-4571 **Nasopharyngeal and Adjacent Neoplasms: A Clinico-pathologic and Immunologic Study.** (Eng) Uhlmann, C. (ENT Clinic and Immunopathology Labs., Pathology Inst., Univ. Cologne, Joseph-Stelzmann-Strasse 9, D-5000 Cologne 41, W. Germany); Krueger, G. R.; Sesterhenn, K.; Rose, K. G.; Ablashi, D. V.; Wustrow, F. *Arch Otorhinolaryngol (NY)* 218(3/4): 163-177; 1978.

Thirteen cases of nasopharyngeal carcinoma (NPC) and 16 cases of non-NPC tumors in the nasopharynx or in adjacent locations were investigated clinically immunologically, and pathologically. All tumors were classified according to the TNM classification, and the stage and course of the disease were correlated with tumor histology, the T- and B-cell distribution in tumor tissue and in the peripheral blood, as well as with antibody titers against Epstein-Barr virus (EBV). There was a positive correlation between decreased T and B cells in tumor tissue and decreased T cells in the peripheral blood with the extent of the tumor in both NPC and non-NPC cases, except for some lymphoepithelial carcinomas and malignant lymphomas. Elevated titers of anti EBV antibodies were observed in anaplastic carcinomas and in lymphoepithelial carcinomas type Regaud, but not in classical squamous cell carcinomas, giant follicular lymphomas, or non-NPC tumors. Although only a limited number of patients with NPC were studied, repeated serological testing allowed the conclusion that there was some correlation between antibodies to EBV early antigen and viral capsid antigen and the stage of the tumor. (25 refs)

78-4572 **Characterization of Xenoantisera to Human Neuroblastoma (NBL) Cells (Meeting Abstract).** (Eng) Raney, B. (Children's Hosp., Philadelphia, PA, 19104); Schlesinger, H.; Hummeler, K. *Proc Am Assoc Cancer Res* 19: 182; 1978. (no refs)

78-4573 **Modification of the Immunologic Properties of the Cell Surface.** (Eng) Hakim, A. A. (Dept.

Surgery, Physiology Biophysics, Univ. Illinois Medical Center, Chicago, IL, 60680). *Immunol Commun* 7(1): 25-39; 1978.

The comparative immunologic effects of glutaraldehyde-, formalin-, 2,4,6-trinitrophenylate-, *Vibrio cholerae* neuraminidase-, iodoacetate-, heparin-, histamine-, and cyclic AMP-treated living CF_1 mouse-transplantable spontaneous mammary adenocarcinoma cells were determined in syngeneic CF_1 mice. Animals immunized with the fixed (formalinized or glutaraldehyde-fixed) neuraminidase-treated cells or the membrane of these cells rejected two challenging doses of 10^8 viable unmodified adenocarcinoma cells. Animals immunized with adenocarcinoma cells treated with neuraminidase (170 IU/5 x 10^5 cells) or with the spontaneous adenocarcinoma cell-surface-associated glycoprotein, rejected the first challenging dose but developed tumors and died on the second challenge with the viable untreated adenocarcinoma cells. Animals immunized with the adenocarcinoma cells pretreated with trinitrophenylate, glutaraldehyde, or formalin developed temporary resistance to the spontaneous mammary adenocarcinoma. Adenocarcinoma cells pretreated with NaF, iodoacetate, heparin, EDTA, colchicine, or histamine showed reduced oncogenicity and stronger resistance in mice to the development of a mammary tumor than to a smaller number (10^3) of untreated viable adenocarcinoma cells. Cells treated with cyclic AMP accelerated tumor development. (31 refs)

78-4574 **Further Studies on the Relationship Between Large Glycoprotein Molecules and Allotransplantability in the TA3 Tumor of the Mouse: Studies on Segregating TA3-Ha Hybrids.** (Eng) Codington, J. F. (Lab. Carbohydrate Res., Dept. Biological Chemistry, Massachusetts General Hosp., Boston, MA, 02114); Klein, G.; Cooper, A. G.; Lee, N.; Brown, M. C.; Jeanloz, R. W. *J Natl Cancer Inst* 60(4): 811-818; 1978.

The chemical and biological characteristics of epiglycanin were studied in six TA3-Ha/A.CA hybrid cell lines in order to obtain information on its role in antigen masking in the TA3 tumor system. The cell lines were formed by fusion of the TA3-Ha mammary carcinoma of a strain A mouse with a normal embryonic fibroblast of an A.CA mouse and then converted to the ascites form in parental strain A mice. The ability of these lines to grow in foreign mouse strains was inversely related to their ability to absorb anti-H-2a antibody. The absorptive capacities of the hybrid cell lines were intermediate between the low absorptive capacity of the non-strain-specific parent TA3-Ha ascites cell line and the much higher absorptive capacity of the strain-specific ascites line TA3-St of the same tumor. Each hybrid cell line possessed an abundance of large endogenous cell-surface glycoprotein molecules similar to epiglycanin, a glycoprotein detected at the surface of the parent TA3-Ha cell. The results suggested that the amount of epiglycaninlike material at the hybrid cell surfaces may have been directly related to the capacities of the cells to grow in foreign mouse strains and inversely related to their capacities to absorb anti-H-2a antibody. (28 refs)

78-4575 **In Vitro Studies on Splenocyte Cytotoxicity Against Syngeneic Mammary Carcinomas of Mice. 1. Reactivity of Splenocytes from Mice Bearing Mammary Tumors Against the Corresponding and Against Other Neoplasms.** (Eng) More, R. (Lautenberg Center General and Tumor Immunology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Yron, I.; Weiss, D. W. *Isr J Med Sci* 14(1): 146-161; 1978.

As demonstrated by a terminal ^{51}Cr-labeling assay, splenocytes of BALB/c mice bearing large, progressively growing implants of the D7T4S syngeneic mammary carcinoma, which does not express the known mature mammary tumor viruses (MTV), markedly reduced the numbers of viable corresponding tumor target cells after culture in vitro. Similar cytotoxic reactivity was shown by splenocytes from BALB/c genotype animals bearing MTV-positive D14 mammary tumors. Spleen cells from normal BALB/c genotype donors exhibited only occasional cytotoxic activity against these neoplasms. Removal of glass-adherent cells from splenocyte populations from normal syngeneic mice decreased their in vitro cytotoxicity for D7T4S and D14 target cells was manifested. In contrast, removal of glass-adherent cells did not lower the cytotoxic potency of splenocytes from tumor-bearing donors. Preliminary studies were conducted on the specificity of these cytotoxic reactions. Splenocytes from mice with isografts of a plasmacytoma and of two methylcholanthrene(MC)-induced fibrosarcomas, with demonstrated cytotoxic reactivity for the corresponding tumor cells, exerted no cytotoxic effects on D7T4S targets. However, there was cross-reactivity between mammary carcinomas and a third MC-induced sarcoma. Cross-reactivity was also evident between spontaneous and transplanted MTV-positive mammary carcinomas and D7T4S. Thus, the specificity of cellular immunity against murine mammary tumors and other experimental neoplasms is highly problematic. (39 refs.)

78-4576 **Time Distributions of Recurrences of Immunogenic and Nonimmunogenic Tumors Following Local Irradiation.** (Eng) Suit, H. D. (Edwin L. Steele Lab. Radiation Biology, Dept. Radiation Medicine, Massachusetts General Hosp., Harvard Medical Sch., Boston, MA, 02114); Sedlacek, R.; Fagundes, L.; Goitein, M.; Rothman, K. J. *Radiat Res* 73(2): 251-266; 1978.

Tumor recurrences and the appearance of radiation-induced sarcoma were investigated in 314 10- to 12-wk-old C3Hf/Sed mice that received single-dose irradiation (4,000-7,100 rads at 950-975 rads/min) of the leg and thigh as treatment of an 8-mm mammary carcinoma isotransplant. The times for the transplant tumor inoculum to grow to an 8-mm tumor and the times of death due to local regrowth, distant metastasis, or induced tumor were all approx log-normally distributed. The 128 recurrences occurred 30-436 days after irradiation, mainly during the early part of this period; only 3/128 recurrences occurred >300 days postirradiation. These findings were interpreted to mean that in some cases, mice were cured of the leg tumor by radiation alone at doses of 6,500-7,100 rad. Radiation-induced sarcomas began to appear after 300 days. The time of appearance of the radiation-induced tumors was inversely related to radiation dose. Similar data for an immunogenic fibrosarcoma (induced by methylcholanthrene) indicated that recurrences appeared earlier and were more closely bunched with respect to time than the recurrences of mammary carcinoma. The time distribution of the development of radiation-induced tumors in non-tumor-bearing animals was not different from that for radiation-induced tumors in mice that had been treated for tumor. It does not appear, therefore, that the frequency of tumor induction by radiation is altered by the prior growth of a tumor in the irradiated site. (17 refs)

78-4577 **Identification of Lipids Associated with the Ability of Tumor Cells to Resist Humoral Immune Attack (Meeting Abstract).** (Eng) Schlager, S. I. (NIH, Bethesda, MD, 20014); Ohanian, S. H.; Borsos, T. *Proc Am Assoc Cancer Res* 19: 94; 1978. (no refs)

78-4578 **Suppression of Growth of Guinea Pig Line-10 Hepatocarcinoma. I. Effect of Simultaneous Administration of Tumor Cells and Antibodies from Normal Rabbits.** (Eng) Brunda, M. J. (Natl. Jewish Hosp. and Res. Center, Denver, CO, 80206); Minden, P.; Heusser, C. H.; Ferguson, H. R. *J Immunol* 120(3): 831-836; 1978.

Serum or immunoglobulins (Ig) from normal New Zealand white rabbits were mixed with the diethylnitrosamine (DEN)-induced line-10 hepatocarcinoma from strain-2 guinea pigs and injected id into normal guinea pigs to study the role of antibodies in tumor rejection. Suppression of growth of the line-10 hepatocarcinoma occurred when 10^5-10^6 cells were injected with sera or Ig. A significant number of animals were also resistant to subsequent rechallenge with tumor cells. The immunity was specific and it depended on contact of the Ig with the tumor cells and on the concentration of Ig. Repeated injections resulted in the development of immunity in 11/13 recipients. Fc receptors were not detected on line-10 cells. Antibodies reacting with line-10-unique antigens and with antigens common to line 10, DEN-induced line-1, and normal guinea pig spleen cells were found in normal rabbit sera. Injection of line-10 cells together with rabbit Ig from which antibodies reacting with line-10 antigens had been removed did not result in tumor suppression. The specific antigen(s) recognized by antibodies that suppressed the growth of the line-10 tumor in vivo was not identified. (35 refs)

78-4579 **In Vitro Measurement of Carcinogen-resistant Liver Cells During Hepatocarcinogenesis.** (Eng) Laishes, B. A. (Dept. Pathology, Univ. Toronto, Medical Sciences Bldg., Toronto, Ontario M5S 1A8, Canada); Roberts, E.; Farber, E. *Int J Cancer* 21(2): 186-193; 1978.

Hyperplastic nodules from the liver of male Fischer 344 albino rats fed a diet containing 0.02% 2-acetylaminofluorene (AAF) for 14-16 wk were transferred to primary monolayer cultures, and the proportion of nodule cells resistant to aflatoxin B_1 (AFB) was determined. Nodule and normal cell cultures were exposed to 10^{-7}-10^{-5} mole/liter AFB for 24 or 48 hr. The nodule cell cultures contained a larger number of resistant cells than cultures from normal rat liver. At all AFB concentrations, there was approx a threefold drop in the percentage of surviving nodule-derived cells; survival of normal cells decreased twentyfold. A comparatively large population of hepatocytes from nodule liver did not even respond morphologically to the AFB, and they continued to spread out on the substratum in the same way as cells in control cultures without AFB. Furthermore, some nodule cells survived exposure to AFB for up to 2 mo. Dexamethasone supplementation of the culture media increased the longevity of normal hepatocytes and increased the proportion of resistant normal cells exposed to 10^{-6} and 10^{-5} mole/liter AFB, but did not significantly increase the size of the resistant nodule cell population. It is not known whether the resistance to cytotoxicity in vitro is the result of reduced levels of certain components of the chemical-carcinogen activation system, reduced uptake of the carcinogen, or increased amounts of intracellular molecules that bind carcinogens. (35 refs)

78-4580 **Immune Response of the Draining and Distal Lymph Nodes During the Progressive Growth of a Chemically-induced Transplantable Rat Hepatoma.** (Eng) Jones, J. A. (Cancer Res. Campaign Labs., Univ. Nottingham, Univ. Park, Nottingham, NG7 2RD, England); Robinson, G.; Rees, R. C.; Baldwin, R. W. *Int J Cancer* 21(2): 171-178; 1978.

The immune response in lymph nodes draining the site of a 4-dimethylaminoazobenzene-induced hepatoma (D192A) implanted im in male W/Not rats was investigated. Cell-mediated cytotoxicity against hepatoma D192A and 15-day rat embryo cell targets was detected in cells from the draining lumbar node 4 days after tumor implantation; this persisted up to 2 wk of tumor growth and decreased during week 3. The reactivity of tumor bearer cervical lymph node cells increased with progressive tumor growth. The lymph node anergy observed in the cytotoxicity tests correlated with the paracortical (T-dependent) response, which decreased toward the end of 3 wk. The B-dependent cortex showed active lymphocyte follicles during the second week, and plasma cell production continued until the experiment was terminated. These changes occurred with progressive increase in lymph node mass. Serum antibody specific for the developing tumor was detected after day 20. (33 refs)

78-4581 **On the Mechanism of Reduction of Antiblastomogenic Resistance in Nephrogenic Hypertension.** (Rus) Litvitsky, P. F. (Dept. Pathophysiology, I. M. Sechenov First Medical Inst., Moscow, USSR); Kogan, A. Kh. *Patol Fiziol Eksp Ter* (1): 62-63; 1978.

The reduction of antiblastomogenic resistance due to nephrogenic hypertension was studied in random-bred rats. Nephrogenic hypertension was simulated by left nephrectomy with subsequent (10-14 days later) stenosing of the contralateral renal artery. On days 13-14 of hypertension, animals received one of the following: a paraffin disk implant containing 2 mg of benzo(a)pyrene (BP) or a transplant of carcinoma PC-1, sarcoma 45, Walker carcinoma 256, or Zajdela hepatoma cells. The functional state of the immune system was measured by the carcinolytic activity of the serum (CAS) and the number of lymphocytes and plasmacytes in the spleen specimens. The amount of 11-oxycorticosteroid (11- OCS) in the blood plasma was also assessed. The CAS decreased from 3.78 in intact (control) animals to 3.06 on day 28 of hypertension and 2.79 on day 100 of hypertension. The decrease was even more pronounced in animals with BP-induced tumors (0.63) and animals with carcinoma PC-1 (0.67). Rats with hypertension had a slightly decreased number of splenic lymphocytes: 321.6 on day 28 of hypertension, compared with 359.0 in controls, 273.6 in rats with hypertension + BP-induced tumors, and 280.0 in rats with hypertension + sarcoma 45. The blood plasma content of 11-OCS was increased in rats with hypertension and in those with hypertension + tumors. (22 refs)

78-4582 **Abrogation of Tumor Rejection by Trypan Blue.** (Eng) Kreider, J. W. (Dept. Pathology, Coll. Medicine, Coll. Medicine, Pennsylvania State Univ., Hershey, PA, 17033); Bartlett, G. L.; DeFreitas, E. *Cancer Res* 38(4): 1036-1040; 1978.

A determination was made of the effects of chronic trypan blue treatments on three animal tumor models in which strong, tumor-specific rejection immunity developed: a UV-induced cutaneous mouse tumor (UVT-2051), a diethylnitrosamine-induced line 1 guinea pig hepatoma, and a line 10 guinea pig hepatoma. UVT-2051 tumor fragments were implanted sc in the mice, and trypan blue was administered ip at a dose of 4 mg on day -1 and again 3 hr before tumor grafts; 1 mg 2x/wk was given thereafter. With line 1 cells, 3 x 10^6 cells were injected sc; with line 10, 1 x 10^5 cells were injected id in the flank, and a vaccine of irradiated cells and BCG was administered simultaneously in the other flank. For the two tumor models, guinea pigs received 30 mg/kg trypan blue sc on day 0 and 2x/wk thereafter, or 100 mg/kg on day 0 and a maintenance dose of 15 mg/kg 2x/wk thereafter, respectively. Trypan blue treatment prevented tumor-specific rejection in all three systems, but the secondary immune reactions to line 10 challenges were abolished. The mechanism by which tumor-specific rejection was inhibited is not known. (25 refs)

78-4583 **Hepatitis B Antigen in Black Patients with Hepatocellular Carcinoma: Correlation Between Orcein Stained Liver Sections and Serology.** (Eng) Cohen, C. (Dept. Anatomic Pathology, Hershey Medical Center, Hershey, PA 17033); Berson, S. D.; Geddes, E. W. *Cancer* 41(1): 245-249; 1978.

Formalin-fixed, paraffin-embedded liver and tumor tissue from 50 black mineworkers with hepatocellular carcinoma (HCC) were examined by orcein stain for the presence of cytoplasmic hepatitis B surface antigen (HBsAG). In 16/18 cases, HBsAG-positive liver tissue correlated with positive serum HBAg. The two false-positive serum HBAg results were obtained by immunodiffusion (ID), immunoelectrophoresis (IEP), and complement fixation (CF). Of the 32 HBsAG-negative cases, 14 had a negative serum HBAg upon ID, IEP, and CF, but a positive serum HBAg upon radioimmunoassay or hemagglutination. Six other negative orcein results appeared to be due to a sampling error. Although orcein staining of tumor cells was positive in three patients with serum HBAg, the staining reaction was equivocal. Provided the limitations of the technique are realized, orcein staining of liver tissue may be useful in retrospective studies of the prevalence of HBAg in HCC patients. (28 refs.)

78-4584 **Association Between HLA Antigens and Gastro-Intestinal Neoplasia (Meeting Abstract).** (Eng) Gennari, L. (Istituto Nazionale Tumorii, Universidad di Milano, Milan, Italy); Spinelli, P.; Pellegris, G.; Illeni, M. T.; Garotta, G. *Digestion* 16(3): 240; 1977. (no refs)

78-4585 **Quantitative Analysis of Immunogenic Activity of SV40 and of Tumor Cells Induced by This Virus.** (Rus) Klyuchareva, T. E. (Cancer Res. Center, USSR

Acad. Medical Sciences, Moscow, USSR). *Biull Eksp Biol Med* 86(8): 223-225; 1978.

Investigations of specific antitumor immunity induced in Syrian hamsters by oncogenic simian virus 40 (SV40) or by cells of SV40-induced tumors in Syrian hamsters indicated a direct correlation between the level of specific antitumor resistance and the immunizing virus or tumor cell dose. The minimal resistance-inducing dose of wild-type SV40 virus was 10 times higher than that of the temperature-sensitive mutant of this virus (tsA-30). The minimal resistance-inducing dose of irradiated tumor cells was about 9 x 10^5 cells; a 10-fold increase in this dose significantly increased the specific antitumor immunity level. (8 refs)

78-4586 **Immunity Against MOPC 104E Plasmacytoma: Effects of Tumor Size and Time Post Therapy on In Vivo Tumor Immunity.** (Eng) Lubet, R. A. (Dept. Biochemistry, Univ. Texas Health Science Center at Dallas, 5323 Harry Hines Blvd., Dallas, TX, 75235); Carlson, D. E. *J Natl Cancer Inst* 60(5): 1107-1111; 1978.

Ten- to 12-wk-old BALB/c mice with palpable sc MOPC 104E plasmacytomas (6-15 x 10^7 cells) were treated with cyclophosphamide (CP: 10 or 250 mg/kg ip) and, at various intervals, rechallenged with different doses of viable tumor cells, and the effects of tumor size and time posttherapy (PT) on in vivo tumor immunity were determined. When CP-treated ("cured") animals were given a secondary challenge of 3 x 10^3-2 x 10^6 tumor cells 22, 45, 120 days PT, they had a considerable increased ability to reject the challenge, and the av time of tumor appearance (in any "immune" animals that did permit tumor growth) was delayed. All low-CP-dose mice showed similar degrees of tumor immunity: a TD_{50} between 200,000 and 800,000 viable cells and a similar period of tumor latency (15.3 vs 10 days in controls with 200,000 cells). Similar tumor immunity levels were found in all high-CP-dose mice, but they were lower than those of the low-dose animals. Tumor latency was also delayed in these mice (20.8 vs 17.2 days with 1.25 x 10^4 cells). Thus, tumor immunity remained constant for at least 4 mo PT. A second group of animals was treated with 10 mg/kg CP 4, 11, or 20 days after tumor cell injection and then rechallenged with various numbers of viable tumor cells 30 days PT. These three subgroups rejected 6, 60, or 400 times as many tumor cells, respectively, as did controls. Thus, animals cured of large tumors and, by implication, exposed to greater amounts of tumor antigens showed greater residual tumor immunity than animals cured of relatively small tumors. (25 refs)

78-4587 **Cell-Surface Major Glycoprotein of BALB/c Mouse Plasmacytoma 58-8 Cells.** (Eng) Tokuyama, H. (Dept. Immunology Res., Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY, 14263); Migita, S. *J Natl Cancer Inst* 61(1): 203-208; 1978.

Alterations of cell-surface components of BALB/c mouse plasmacytoma 58-8 cells during successive ip transplantations in syngeneic mice were studied by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE). By SDS-PAGE and PAS staining, all 58-8 cells were found to have a common, unique, PAS-positive (PAS+) major membrane component (MMC) with a mol wt of about 170,000 daltons. The amount of this component increased with increasing transplants. The MMC appeared to be a major constituent of cell-surface membranes, since it was removed from cells by trypsinization and since its level in the crude membranes was 22-fold higher than that in the whole cell. An analysis of the SDS-PAGE patterns of surface-iodinated 58-8 cells revealed a prominent surface membrane component with the same mol wt (170,000 daltons) as that of the PAS+ MMC; the amount of this component also increased with increasing transplants. The PAS+ MMC and the iodinated major component appeared identical, as both were sensitive to tryptic digestion and the extraction percentage of the PAS+ MMC (29.8%) and the iodinated major component (28%) by 1 M urea was similar. The real mol wt of the PAS+ glycoprotein was estimated to be 120,000 daltons by SDS-PAGE. The PAS+ major glycoprotein contained a high amount of sialic acid residues. The sialoglycoprotein on the cell surface of 58-8 cells appeared to be the masking material that made the 58-8 cells unreactive with antibodies directed to their tumor-associated antigens or to the histocompatibility H-2 antigen. (15 refs)

78-4588 **Serologic Identification of Multiple Tumor-associated Antigens on Murine Sarcomas.** (Eng) Parker, G. A. (Surgery Branch, NCI, NIH, Public Health Service, U.S Dept. Health, Education, and Welfare, Bethesda, MD 20014); Rosenberg, S. A. *J Natl Cancer Inst* 58(5): 1303-1309; 1977.

Humoral immune responses to two transplanted chemically induced (3-methylcholanthrene) murine sarcomas (MCA-2 and MCA-3) were studied in C57BL/6N mice. These tumors were immunogenic, as evidenced by tumor amputation and rechallenge experiments, and they did not cross-react in in vivo challenge experiments. In a complement-dependent microcytotoxicity assay, antibodies to both MCA-2 and MCA-3 were detected in the sera of animals bearing MCA-3 and of those which had tumors removed. The sera of animals hyperimmunized to MCA-3 (MCA-3HI) were also cytotoxic with high titers to both MCA-2 and MCA-3 (50% cytotoxicity titers of 1:80 and 1:320, respectively). Sequential absorptions of sera from animals bearing MCA-3 and MCA-3HI sera with fresh MCA-2 cells completely removed the activity against MCA-2 but not the reactivity to MCA-3. Sequential absorptions with fresh MCA-3 cells produced stepwise reductions of activity against both tumors, but absorption with normal cells produced no loss of activity against either tumor. Thus, both specific and cross-reactive antigens were expressed on the surfaces of MCA-3 cells. Only the specific tumor antigen appeared to be involved in in vivo protection against tumor challenge. (28 refs.)

78-4589 **Failure of Neuraminidase Treatment to Influence Tumorigenicity or Immunogenicity of Syngeneically Transplanted Rat Tumour Cells.** (Eng) Pimm, M. V. (Cancer Res. Campaign Labs., Univ. Nottingham, Nottingham NG7 2RD, England); Cook, A. J.; Baldwin, R. W. *Eur J Cancer* 14(8): 869-878; 1978.

Vibrio cholerae neuraminidase (VCN) and *Clostridium perfringens* neuraminidase were examined for their ability to reduce the tumorigenicity of a range of seven different transplanted rat tumors. The enzymes released 0.2 to 1.2 micromoles of sialic acid/10^9 cells, comparable to levels reported by others. However, in no case did treatment of tumor cells with either enzyme consistently reduce in vivo tumorigenicity over and above that caused by the enzymes' buffer soln. Moreover, with a strongly immunogenic 3-methylcholanthrene-induced sarcoma, VCN treatment of cells did not improve their efficiency for active specific immunotherapy, and enzyme treatment of cells of an N-hydroxy-2-acetylaminofluorene-induced mammary carcinoma, nonimmunogenic by conventional criteria, did not render them immunogenic. These studies fail to confirm the reduced tumorigenicity of neuraminidase-treated cells or their increased immunogenicity. (36 refs.)

78-4590 **Lymphocytotoxic Autoantibodies Eluted from In Vivo Propagating Sarcoma Cells of Mice: Brief Communication.** (Eng) Ran, M. (Dept. Microbiology, George S. Wise Center Life Sciences, Tel-Aviv Univ., Tel-Aviv, Israel); Yaakubowicz, M.; Witz, I. P. *J Natl Cancer Inst* 60(6): 1509-1513; 1978.

Eluates of SEYF-a cells (from a polyoma virus-induced sarcoma grown in A.BY mice) containing cytotoxic anti-SEYF-a antibodies were found to mediate the complement-dependent cytotoxicity of normal lymph node cells. Some lymphocytotoxicity was also mediated by the sera of tumor-bearing mice, but the pattern of cytotoxicity was different from that exhibited by the tumor eluates. The titer of lymphocytotoxic antibodies in the serum was lower than the titer of anti-SEYF-a antibodies, but eluates exhibited similar titers toward both target cells. These findings indicate that the tumor cells selectively absorbed the lymphocytotoxic antibodies from the serum. Tumor eluates also had cytotoxic activity against thymocytes, splenocytes, and bone marrow cells. The strain distribution of sensitive thymocytes to the antibodies present in the eluates indicated that Thy 1 was not involved. Unfractionated eluates as well as IgG fractions isolated from eluates were active in mediating cytotoxicity. (24 refs)

78-4591 **Solubilized Tumour-Associated Antigens of Methylcholanthrene-induced Mouse Sarcomas. Comparative Studies by In Vitro Sensitization of Lymph-Node Cells, Macrophage Electrophoretic Mobility Assay and Transplantation Tests.** (Eng) Bubenik, J. (Inst. Molecular Genetics, Czechoslovak Acad. Sciences, 16610 Prague 6, Czechoslovakia); Indrova, M.; Nemeckova, S.; Malkovsky, M.; Von Broen, B.; Palek, V.; Anderlikova, J. *Int J Cancer* 21(3): 348-355; 1978.

The immunologic potential and antigenic specificity of solubilized tumor-associated antigens (TAA) extracted from methylcholanthrene-induced sarcomas in C57BL10/ScSn mice were examined by in vitro sensitization of lymph node cells (LNC), macrophage electrophoretic mobility (MEM) assay, and transplantation tests. The solubilized TAA elicited both primary and secondary immune responses in vivo, as judged from induction of transplantation resistance. The resistance to transplantation of the corresponding sarcoma cells was tumor-specific, partial, and weaker than that induced by immunization with irradiated tumor tissue. Treatment of mice with solubilized TAA prior to immunization with irradiated tumor tissue inhibited the induction of transplantation resistance by irradiated tumor cells. The interaction of the solubilized TAA preparations in vitro, either with LNC from animals immunized against the corresponding tumor or with LNC from tumor-bearing animals, gave rise to a macrophage-slowing factor detectable by the MEM test. TAA detected by the MEM assay in extracts of individual sarcomas were non-cross-reacting and individually distinct. Individual specificity of TAA present in the sarcomas was also demonstrated in vitro by sensitization of normal syngeneic LNC on monolayers of irradiated sarcoma cells, followed by detection of the generated cytotoxic LNC in a cytotoxicity assay. Although sensitization of LNC on layers of irradiated sarcoma cells gave rise to lymphocytes cytotoxic for cells of the sensitizing tumor, sensitization of LNC with solubilized TAA isolated from the same sarcoma gave negative results. (35 refs)

78-4592 **Tumor Inhibition by Effector Cells Cultured from Progressing Sarcomas.** (Eng) Berczi, I. (Dept. Immunology, Faculty Medicine, Univ. Manitoba, Winnipeg R3E OW3, Manitoba, Canada); Sehon, A. H. *Immunol Commun* 6(6): 617-632; 1977.

The effect of cytotoxic lymphoid cells emerging in primary cultures of an antigenic methylcholanthrene-induced sarcoma in inbred Sewall-Wright strain 13 guinea pigs was investigated on tumor growth in vivo. The growth of lethal tumor inocula was inhibited or the tumor cells were completely rejected in normal syngeneic recipients treated sc with 5 x 10^5 effector cells mixed with the tumor cells. A higher dose (2.5 x 10^6) of killer cells did not affect tumor growth significantly in normal recipients. Significant tumor inhibition was observed in x-irradiated recipients given 2.2 x 10^6 effector cells locally, although rejection did not occur with high frequency. Local treatment of irradiated recipients with 5 x 10^5 effector cells did not influence tumor growth. Systemic treatment of normal and irradiated recipients with effector cells had little effect at any dose, although tumor inocula were occasionally rejected. Sonication of the effector cells prior to administration inhibited their tumor-suppressing effect. Animals that rejected the first tumor inoculum were immune to a second lethal dose. These findings illustrate the complexity of the tumor-host interaction. (25 refs)

78-4593 **In Vitro and In Vivo Expression of Original and Foreign H-2 Antigens and of the Tumor-associated Transplantation Antigen of a Murine Fibrosarcoma.** (Eng) Carbone, G. (Div. Experimental Oncology A, Istituto Nazionale per lo Studio e la Cura dei Tumori, Via G. Venezian 1, 20133 Milan, Italy); Invernizzi, G.; Meschini, A.; Parmiani, G. *Int J Cancer* 21(1): 85-93; 1978.

The expression of original and foreign histocompatibility (H-2) antigens and tumor-associated transplantation antigen on the C-1 3-methylcholanthrene-induced BALB/c fibrosarcoma was investigated. The binding of C57BL/6J or C3Hf anti-BALB/c sera or that of monospecific alloantisera to H-2d specificities on plated C-1 cells indicated that the H-2d antigen expression was lower or undetectable on the in vitro tumor. The low expression of H-2d antigens on in vitro C-1 cells was confirmed by absorption experiments that allowed a direct comparison of in vivo and vitro cells. In these experiments, in vitro C-1 cells were unable to absorb the cytotoxicity of C57BL/6J antiBALB/c serum. H-2k antigens found on C-1 were examined using BALB/c anti-C3Hf, anti-AKR, anti-C57BL/6J polyspecific sera and also monospecific alloantisera against H-2k antigens. The expression of H-2k antigens was significantly lower in the in vitro than in the in vivo tumor, with antigen 5 being undetectable in the cultured cells. Allogeneic and syngeneic sera raised against in vitro or in vivo C-1 cells were tested on either BALB/c or C3Hf normal lymph node cells by the complement-dependent cytotoxicity assay. A significantly higher titer of cytotoxic antibodies to H-2d and to H-2k was obtained in the allogeneic and syngeneic sera, respectively, following immunization with the in vivo C-1 cells, compared with sera raised against the in vitro line. In vivo and in vitro C-1 cells had the same tumor-associated transplantation antigen, and its expression was not significantly reduced on cultured cells. (29 refs.)

78-4594 **Immunoprevention of Cancer in Experimental Animals: Strategies that Might be Useful in Prevention of Cancer in Man.** (Eng) Huebner, R. J. (NCI, Bethesda, MD); Gilden, R.; Fish, D.; Djurickovic, D.; Toni, R.; Stephenson, J.; Hill, P.; Trimmer, R.; Whitmore, C.; Rhim, J. *In: Carcinogens: Identification and Mechanisms of Action. Proceedings of the Thirty-First Annual Symposium on Fundamental Cancer Research Held at Houston, Texas, 1-3 March 1978.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute, NCI, American Cancer Society. (Austin, Texas). 18 pp.; 1978. (no refs)

78-4595 **Genetic Studies of Autoimmunity and Retrovirus Expression in Crosses of New Zealand Black Mice. I. Xenotropic Virus.** (Eng) Datta, S. K. (Hematology Service, New England Medical Center Hosp., Boston, MA, 02111); Manny, N.; Andrzejewski, C.; Andre-Schwartz, J.; Schwartz, R. S. *J Exp Med* 147(3): 854-871; 1978.

The role of xenotropic virus in the development of autoimmune disease was investigated in the progeny of crosses between New Zealand Black (NZB) and SWR mice. [(NZB x SWR)F_1 x SWR] mice and the F_2 progeny segregated into three phenotypes: high-virus, low-virus, and virus-negative; F_1 and (F_1 x NZB) progeny were always high virus. Autoantibodies, immune deposit nephritis, and lymphomas developed in the progeny of these crosses. The virological phenotype of the animal could be dissociated from the presence of either autoantibodies or nephritis: mice that expressed virus titers as high as the NZB parent failed to develop signs of autoimmunization, even up to 24 mo of age. However, some (F_1 x SWR) and F_2 mice that expressed low titers of virus developed autoimmune disease. Furthermore, a proportion of virus-negative mice produced autoantibodies and had typical immune deposit nephritis. No viral antigens could be detected in the renal lesions of such virus-negative animals. By contrast with the dissociation between expression of virus and occurrence of nephritis, the presence of antibodies to DNA correlated with the development of renal lesions. Thus, genes determining the expression of infectious xenotropic virus in NZB mice segregate independently from those involved in the autoimmune disease of these animals. (46 refs)

78-4596 **Genetic Studies of Autoimmunity and Retrovirus Expression in Crosses of New Zealand Black Mice. II. The Viral Envelope Glycoprotein gp70.** (Eng) Datta, S. K. (Hematology Service, New England Medical Center Hosp., Boston, MA, 02111); McConahey, P. J.; Manny, N.; Theofilopoulos, A. N.; Dixon, F. J.; Schwartz, R. S. *J Exp Med* 147(3): 872-881; 1978.

The viral envelope glycoprotein gp70 was measured in the serum of crosses of new Zealand Black (NZB) x SWR mice, and the levels were analyzed in relation to the expression of infectious xenotropic virus, presence of autoantibodies, and development of glomerulonephritis and lymphomas. The progeny of the crosses had serum gp70 values that segregated to NZB-like and SWR-like levels. A complex mechanism determined the inheritance of NZB-like serum gp70 levels. The factors determining the expression of this retroviral protein were independent of the genes (*Nzv-1* and *Nzv-2*) that determined the expression of infectious xenotropic virus. Autoimmune disease, including immune deposit nephritis, could be dissociated from the degree of expression of serum gp70. However, the presence of circulating immune complexes and anti-DNA antibody did correlate with the development of nephritis in these crosses. A significant correlation was found between high-grade expression of serum gp70 and the presence of lymphomas in these mice. (16 refs)

78-4597 **Antibody Stimulation of Benzo(a)pyrene Carcinogenesis.** (Eng) Curtis, G. L. (Univ. Nebraska Medical Center, Omaha, NB, 68105); Ryan, W. L.; Stenback, F. *Cancer Lett* 4(4): 223-228; 1978.

The possibility that chemical carcinogens induce a humoral antibody that plays an active role in carcinogenesis was investigated using female Swiss mice immunized with benzo(a)pyrene (BP). Antibody to BP was induced by inoculating the mice three times with BP conjugated to horse serum albumin (HSA) [1.6 (μmol) BP to 2.5 x 10^{-2} μmol protein/ip injection]. Three weeks after the final injection, 10 mg BP was applied to the shaved skin of these mice; mice that had been immunized with HSA only, and nonimmunized mice. The mice that had been immunized with BP-HSA had significantly more tumors than the nonimmunized mice. The slight increase in the HSA-immunized group could have ben due to the Freund's complete adjuvant injected with the HSA. A total of 2.5 mg BP was then incubated with diluted or undiluted rabbit anti-BP sera or normal rabbit sera and then injected into mice. Mice that received BP incubated with normal sera had three tumors regardless of whether the sera was diluted or undiluted. Mice that received BP incubated with rabbit anti-BP sera undiluted and diluted 1:5 had significantly more tumors than any group in which BP was incubated with normal rabbit sera. These results suggest that the immune response to carcinogens is an important component of carcinogenesis. (19 refs)

78-4598 **Antibody and Complement Modulation of Tumor Cell Growth In Vitro and In Vivo.** (Eng) Shearer, W. T. (Dept. Pediatrics, Washington Univ. Sch. Medicine, St. Louis, MO, 63110); Parker, C. W. *Fed Proc* 37(10): 2385-2389; 1978.

The results of recent experiments on antibody (ab) and complement (C) modulation of tumor cell growth in vitro and in vivo are reviewed. The transformed L cell line was covalently substituted with trinitrophenyl (TNP) groups and exposed to microgram amounts of highly purified rabbit and anti-TNP ab for various times. The ab specifically stimulated nucleoside uptake in the cells. Low doses of heated rabbit antiserum raised to membrane antigens on L cells also stimulated nucleoside incorporation into L cells, but the response was more marked than that seen in the TNP system. The anti-L cell ab also stimulated in vitro DNA synthesis and cell growth in the L cells. High concentrations of ab inhibited these processes. C activated through C3 augmented the cytostimulatory (CS) effect of low concentrations of ab; C activated through C9 augmented the cytoinhibitory effects of ab. A very early effect of CS concentrations of ab was activation of membrane carrier transport systems, as determined by measurements of $^{45}Ca^{2}+$ uptake in L cells treated with various doses of anti-L cell ab and C. This activation only took place when the cells were also stimulated to grow. Growth of L cells in tissue culture in the continuous presence of a CS concentration of ab selected for a variant cell line (LC_1) that was less responsive to ab, probably because of a blocking effect of increased amounts of sialic acid. LC_1 cells contained twice as much total cellular sialic acid residues as L cells. In vivo experiments documented that the same ab could modulate L-cell growth in T-cell-depleted C3H mice, depending on whether a low or high concentration of ab was given passively. It is concluded, however, that these effects of ab and C on tumor cell growth do not necessarily operate in autochthonous tumor systems, especially in human malignancies. (18 refs.)

78-4599 **True Plasma Corticosterone Levels of Mice in Cancer/Stress Studies (Meeting Abstract).** (Eng) Spackman, D. H. (Pacific Northwest Res. Foundation, Seattle, WA, 98104); Riley, V.; Bloom, J. *Proc Am Assoc Cancer Res* 19: 57; 1978. (no refs)

78-4600 **Antibody Response of Mice to Chemically Induced Tumors.** (Eng) Brown, J. P. (Div. Tumor Immunology, Fred Hutchinson Cancer Res. Center, Seattle, WA, 98104); Klitzman, J. M.; Hellstrom, I.; Nowinski, R. C.; Hellstrom, K. E. *Proc Natl Acad Sci USA* 75(2): 955-958; 1978.

The humoral immune response of BALB/c mice to 3-methylcholanthrene (3-MC)-induced sarcomas was examined to determine whether antibodies were produced against the unique tumor-specific transplantation antigens (TSTA) or against antigens of murine leukemia virus (MuLV). The 3-MC-induced sarcomas varied with respect to their expression of endogenous MuLV. Immunization of syngeneic mice with MuLV-free tumors failed to elicit antibodies against TSTA or MuLV antigens. Immunization with MuLV-producing tumors, however, induced a vigorous antibody response to the envelope proteins of MuLV. Antibodies formed against the MuLV-producing tumors cross-reacted in serological assays with antigens present on other MuLV-producing tumors, but they did not react with antigens on MuLV-free tumors. These data imply that the mechanisms leading to the rejection of chemically induced sarcomas are primarily cellular. (26 refs)

78-4601 **Antitumor Antibody Synthesis In Vitro Induced in Mouse Spleen Cells by Xenogeneic Immune RNA.** (Eng) Kern, D. H. (Dept. Surgery, Harbor General Hosp., Torrance, CA 90509); Pilch, Y. H. *J Natl Cancer Inst* 60(3): 599-603; 1978.

When incubated with xenogeneic immune RNA (I-RNA), globulin preparations from normal, nonimmune C3H mouse spleen cells contained antibody activity against 3-methylcholanthrene- or benzo(a)pyrene-induced fibrosarcomas. Globulins from spleen cells incubated with I-RNA extracted from the lymphoid organs of guinea pigs that had been immunized with either of the two cross-reacting chemically induced C3H mouse sarcomas reacted against target cells of both tumors, but not against target cells of a spontaneously syngeneic osteosarcoma. Globulins from spleen cells incubated with I-RNA from guinea pigs immunized with osteosarcoma cells or cells from a spontaneous C3H mouse mammary tumor reacted with either of the two chemically induced sarcomas, but globulins from spleen cells incubated with antiosteosarcoma I-RNA did not react with osteosarcoma target cells. Globulins from spleen cells incubated with I-RNA extracted from guinea pigs immunized with complete Freund's adjuvant only or with a pool of normal C3H mouse tissues exhibited no reactivity against any of the three target cells tested. The antitumor antibodies synthesized by xenogeneic I-RNA were apparently specific for the particular tumor used to immunize the I-RNA donor. (23 refs.)

78-4602 **Relationship Between the Production of Antibodies Against the Common Enterobacterial Antigen and Carcinogenesis Suppression.** (Rus) Montsevichyute-Eringene, E. V. (Scientific Res. Inst. Oncology, Vilnius, USSR). *Patol Fiziol Eksp Ter* (1): 58-61; 1978.

The formation of antibodies against a common enterobacterial antigen during carcinogenesis and the effect of these antibodies on the growth of induced tumors were studied in random-bred albino rats. Two series of the experiments were performed. In the first series, rats received a single injection of benzo(a)pyrene (BP) or 3-methylcholanthrene (3-MC) (both, 5 mg in 0.4 ml of peach oil, sc). Seven days later, the animals were inoculated with an extract of *Bacillus faecalis alcaligenes* (Bfa), and serum antibodies were assessed by double diffusion. Antibodies against Bfa were detected in 55.7% of the rats exposed to carcinogens, compared with 20.8% of the control (untreated) animals. In the second series, 25 rats with antibodies against Bfa and 25 rats without the antibodies were inoculated with BP (5 mg, sc). The former showed a significant inhibition of tumorigenesis: the av tumor wt and latent period in rats with antibodies against Bfa were 35.5 g and 159 days, compared with 90.9 g and 138 days in rats lacking the antibodies. (17 refs)

78-4603 **In Vivo Modulation of Thymus-Leukemia Antigens on Mouse Leukemia Cells and Thymocytes: Retention of Modulating Antibody on the Cell Surface.** (Eng) Jacobson, J. B. (Donald S. Walker Lab., Sloan-Kettering Inst. Cancer Res., 145 Boston Post Road, Rye, NY, 10580); Galuska, S.; Stackpole, C. W. *J Natl Cancer Inst* 61(3): 819-825; 1978.

Murine thymus-leukemia (TL) antigen modulation was investigated in vivo to determine whether modulation within the animal results in a total loss of modulating antibody and modulated antigens from the cell surface. Inoculation of RADA1, ASL1, and ERLD murine leukemia cells into the peritoneal cavities of (C57BL/6J x A/TL-)F$_1$ mice hyperimmunized against TL cell-surface antigens rendered most cells insensitive to in vitro lysis by guinea pig complement, even in the presence of TL antiserum. Thymocytes of A/J mice were similarly modulated by passive injection of TL antiserum. In both cases, the retention of some modulating antibody on the surfaces of most cells modulated in vivo for 1-27 days was indicated by: (1) acquisition of sensitivity of modulated cells to lysis by absorbed rabbit complement; (2) positive immunofluorescence reactions for mouse IgG on the surfaces of modulated cells; and (3) release of cytolytically active TL antibody from cells into the circulation of unimmunized mice following transfer of modulated cells. Reversal of modulation of RADA1 cells was complete in some experiments within 24 hr after transfer to unimmunized mice, by which time all indications of cell-bound TL antibody were lost. These findings indicate that even long-term modulation of TL antigenicity in vivo does not result in a complete loss of modulating antibody (presumably attached to TL antigens) from the cell surface. (14 refs.)

78-4604 **Comparison of the Mode of Immunopotentiating Action of BCG and Wax D. II. Effect on the Methylcholanthrene Carcinogenesis.** (Eng) Ishibashi, T. (Res. Inst. Diseases Chest, Faculty Medicine, Kyushu Univ., Maidashi 3-1-1, Higashi-ku, Fukuoka 812, Japan); Yamada, H.; Harada, S.; Harada, Y.; Takamoto, M.; Sugiyama, K. *Jpn J Exp Med* 47(6): 435-440; 1977.

The effects of BCG and wax D (extracted from *Mycobacterium tuberculosis* H$_{37}$Ra) on tumor development were investigated in male C3H/He mice treated with a single sc injection of 0.5 mg methylcholanthrene (MC). Doses of 1 mg BCG and 300 μg wax D were used. BCG given 8 wk after MC administration conferred significant protection against carcinogenesis. However, when given either on the same day as MC injection or 4 wk later, a slightly increased tumor incidence was noted. Wax D provided a marked protection against tumor development when given 4 wk after MC, but enhanced tumor development was noted when wax D was administered either on the day of MC treatment or 8 wk later. The protection against tumor development corresponded to a BCG- or wax D-induced increase in the level of delayed-type hypersensitivity at the beginning of tumor development. The enhancing of tumor development corresponded to an induced increase in antibody formation at the time of initiation of tumor development. Thus, the effects of BCG and wax D on tumor development vary with the time of administration. (18 refs)

78-4605 **Early Detection of Tumor-specific Antibodies to Mammary Carcinoma.** (Eng) Blair, P. B. (Dept. Bacteriology and Immunology, Cancer Res. Lab., Univ. California, Berkeley, CA, 94720); Lane, M. A. *Int J Cancer* 21(4): 476-481; 1978.

The antigenic uniqueness of the mammary tumor virus (MTV)-induced mammary tumors of female BALB/cfC3H mice was studied. Sera from 18 tumor-bearing females were used in a microcytotoxicity assay designed to detect immune responses directed against MTV-associated antigens. The sera were tested on both the autologous tumor and on two to three different isologous tumors. There was no evidence of cross-reactivity with 16/18 serum samples, but 2 of the samples did react with a target tumor other than their own. Neither of these two sera reacted with the two other isologous targets on which they were tested. Serial serum samples from multiparous and virgin BALB/cfC3H females were tested using the same assay. Serum activity against tumor-unique antigenicity was observed in samples taken from mice with visible tumors as well as in samples from multiparous females 1-4 mo before a tumor was detected. This antibody activity was not found in serum samples from virgin and nulliparous females before a tumor became grossly visible. The recruiting factor does not appear to be an antigen-antibody complex. The mechanism of tumor formation may involve a longer preneoplastic or latent neoplastic period in parous females than in virgin females. (25 refs)

78-4606 **Host Immunological Reactivity and Adrenal Cortex Function During the Early Period of Chemical Carcinogenesis.** (Rus) Iakimenko, L. V. (Dept. Immunology Carcinogenesis, Inst. Problems in Oncology, Kiev, USSR); Makarchuk, N. M.; Samundzhan, E. M.; Umanskii, Iu. A. *Biull Eksp Biol Med* 85(3): 349-351; 1978.

The associations between immunological reactivity and the functional state of the adrenal cortex were studied in BALB/c mice with 20-methylcholanthrene (20-MC)-induced immune depression. Animals received im injections of 0.2, 0.5, 1, 1.5, or 2 mg 20-MC in 0.1 ml peach oil; 7 days later, the number of antibody-producing cells (APC) in the spleen and the 11-oxycorticosteroid (11-OCS) content in the blood were assessed. Doses of 0.2, 0.5, and 1 g 20-MC increased the serum 11-OCS content 28, 11, and 9 times, respectively. At 0.2 mg, 20-MC decreased the number of APC by 44%; at 2 mg, it decreased the number of APC by nearly 3.5 times. These findings indicate that adrenal cortex hormones play a certain role in the genesis of carcinogen-induced immune depression. (14 refs)

78-4607 **Identification and Behavior of the Precipitating BLV Antibodies in Sera of Leukotic Cattle.** (Eng) Matthaeus, W. (Federal Res. Inst. Animal Virus Diseases, Tubingen, W. Germany); Kaaden, O. R.; Frenzel, B.; Schlotterer, E. *Int J Cancer* 22(2): 166-173; 1978.

Precipitating bovine leukemia virus (BLV) antibodies isolated from bovine leukemic sera by ion-exchange chromatography, gel filtration, and affinity chromatography were identified as immunoglobulins of the IgG_1 subclass and IgA class. They proved to be different with regard to molecular size and electrical charge. IgG_2 and IgM, as well as low-mol-wt components in the microglobulin regions ($\leq$4S) failed to precipitate BLV antigens. Individual bovine leukemic sera showed different precipitating activities against p24 and gp69 antigens. Precipitating monospecific gp69 antibodies represented the IgG_1 subclass exclusively. Some unidentified antigens in disrupted BLV preparations showed a serological relationship to p24 or gp69. (19 refs.)

78-4608 **Relationship Between Membrane Antigen of Human Leukemic Cells and gp70 Oncornavirus Structural Components. Search for Antibodies to the gp70 Antigen.** (Eng) Toth, F. D. (Inst. Microbiology, Univ. Medical Sch., P.O. Box 17, 4012 Debrecen, Hungary); Vaczi, L.; Rak, K.; Balogh, M.; Jako, J. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 148-149; 1978.

Twelve patients with acute myeloid leukemia (AML), 15 with preleukemic disorders (PL), and 44 healthy controls were tested for the major envelope glycoprotein, gp70, of mammalian leukemia viruses and for antibody with gp70 specificity. WBC from six patients with advanced AML and four with PL reacted with gp70 antisera, indicating the presence of gp70 antigen. Remission of leukemia resulted in the disappearance of the antigen and the appearance of gp70 antibody in 7/12 patients. All serum samples from AML patients with advanced disease were negative for the antibody. Of the nine seronegative PL patients, four developed overt leukemia within 1 yr. No gp70-like antigen was present on the surface of normal WBC, but antibody was detected in the sera of 17 healthy persons. (3 refs)

78-4609 **Intra- and Interspecies Reactivity of Human and Mouse Natural Killer (NK) Cells.** (Eng) Hansson, M. (Dept. Tumor Biology, Karolinska Institutet, S 104 01 Stockholm 60, Sweden); Bakacs, K. K.; Kiessling, R.; Klein, G. *J Immunol* 121(1): 6-12; 1978.

Natural killer (NK) cells can kill certain syngeneic, allogeneic and xenogeneic tumor targets in short-term ^{51}Cr-release assays. In the present study, intra- and interspecies NK activity was analyzed. Ten mouse and five human tissue culture lines were used. In direct cytolytic assays with mouse spleen cells or human peripheral blood lymphocyte (PBL) effectors, intraspecies reactivity was much stronger than interspecies reactivity, as a rule. A certain interspecies activity was obtained, stronger in mouse antihuman (M α H) than in human antimouse (H α M) combinations. In the H α M system, activity was associated with the same type of PBL-derived non-B-non-T cell fraction as in the intraspecies H α H system. The non-B-non-T cell nature of the M α H killer cell has been demonstrated previously. Nonlabeled tumor cells were allowed to compete with isotope-labeled targets in intra- and interspecies cytolytic NK tests. NK-sensitive tumor lines of the same species were superior to xenogeneic competitors in both M α M and H α H tests. In the M α M assay, the competing ability of the same human tumors varied, depending on the genotype of the mouse effector cells. None of the human lines tested competed effectively with strain CBA effectors, but some showed a certain competition with C57BL effectors. In the H α H assay, strong competition was seen with 2/10 xenogeneic mouse tumors tested. (25 refs)

78-4610 **Periodic Loss of Reactivity of a Myeloma Tumor with Cytotoxic Thymus-Derived Lymphocytes.** (Eng) Russell, J. H. (Dept. Biology and Center Cancer Res., MIT, Cambridge, MA, 02139); Hale, A. H.; Ginns, L. C.; Eisen, H. N. *Proc Natl Acad Sci USA* 75(1): 441-445; 1978.

During each transplantation passage of a BALB/c mouse plasmacytoma (MOPC-315) through syngeneic mice, the tumor cells lost their reactivity with cytotoxic T lymphocytes directed against the products of the BALB/c major histocompatibility complex (*H-d*). However, this reactivity was regained upon tumor transfer to fresh hosts. In contrast to this cyclical change, MOPC-315 cells remained uniformly reactive with anti-H-2d alloantisera throughout the transplantation cycle. It is not clear whether the periodic loss of reactivity with anti-H-2 cytotoxic T cells represents a mechanism by which the tumor evades destruction by cell-mediated immune responses or reflects a normal differentiation sequence in the maturation of B lymphocytes. (19 refs)

78-4611 **Genetic Resistance to Friend Leukemia Virus in Mice: Masking of *Fv-2* Phenotype by an Epistatic Gene, *Fv-4*^{r}.** (Eng) Odaka, T. (Dept. Genetics, Inst. Medical Science, Univ. Tokyo, Shirokanedai, Minato-ku, Tokyo 108, Japan); Ikeda, H. *Jpn J Exp Med* 47(6): 515-521; 1977.

The *Fv-2* genotype of G mice was investigated by crossing G mice with *Fv-2*^{rr} mice. Both of these strains resist the induction of spleen foci or splenomegaly by Friend leukemia virus, but NB-tropic Friend virus replicates well in the latter and not at all in the former. The (G x *Fv-2*^{rr})F$_1$ hybrids were similar to G. In the F$_1$ x *Fv-2*^{rr} backcross generation, three types of mice appeared: (1) G-type, (2) *Fv-2*^{rr}-type, and (3) *Fv-2*^{SS}-type. Mice of the last type supported virus replication and developed splenomegaly. Mice of types 1, 2, and 3 appeared at the expected ratio of 2:1:1. It is concluded that in G mice, the *Fv-4*^{r} gene suppresses NB-tropic Friend virus replication and thus masks the expression of the *Fv-2*^{S} gene. Studies of the F$_2$ generation support this conclusion. (14 refs)

78-4612 **Immunological Aspects of Mycosis Fungoides.** (Rus) Lezvinskaya, E. M. (Dept. Dermatology, Central Skin and Venereologic Inst., Moscow, USSR). *Vestn Dermatol Venerol* (2): 21-24; 1978.

The blast transformation reaction (BTR) and autoradiography were used to evaluate the functional state of lymphocytes in 30 patients with mycosis fungoides (MF). The indices of lymphocyte transformation were significantly reduced in patients with Stage III MF (30.0%, compared with 55.5% in healthy controls); BTR in patients with Stages I and II MF was similar to that in controls. Patients with an erythrodermoid variant of MF had a marked inhibition of cellular immunity. (29 refs)

78-4613 **Evaluation of the Efficiency of Immune Surveillance in Mice (Meeting Abstract).** (Eng) Patek, P. Q. (Salk Inst. for Biological Studies, San Diego, CA, 92112); Collins, J. L.; Cohn, M. *Fed Proc* 37(6): 1280; 1978. (no refs)

78-4614 **Cellular Interaction Between Cytotoxic and Suppressor T Cells Against Syngeneic Tumors**

in the Mouse. (Eng) Fujimoto, S. (Labs. Immunology, Chiba Univ. Sch. Medicine, Chiba, Japan); Matsuzawa, T.; Nakagawa, K.; Tada, T. *Cell Immunol* 38(2): 378-387; 1978.

The activities of suppresor (sup) and cytotoxic (cyt) T cells against syngeneic tumors (spontaneous lymphoma L1117, methylcholanthrene-induced sarcomas S1509a and SaI) and the modes of interaction between these two cell types in the efferent stage of tumor immunity were determined using A/J mice. An in vitro system was established in which both activities could be estimated simultaneously. Cyt T cells against the tumor lines were induced in vitro by culturing normal spleen and lymph node cells with mitomycin C-treated syngeneic tumor lines. After in vitro stimulation, L1117 lymphoid cells were activated to kill specifically the homologous and closely related tumor cell lines. In addition, the addition of spleen cells from mice bearing the homologous, but no heterologous, tumor to the mixture of cyt and target cells specifically inhibited the T-cell-mediated cyt reaction. With the sarcomas, however, the cyt T cell activated by S1509a or SaI could kill both tumors to the same extent, but the sup T cells from hosts bearing either one of these tumors specifically inhibited the cyt reaction against the homologous tumor but not the cross-reactive one. Sup T cells from tumor bearers possessed Ia antigen controlled by a gene in the *I-J* subregion of the major histocompatibility complex (*H-2*). Cyt T cells generated by some but not all syngeneic tumors were, like sup cells, killed by anti-Ia and complement; however, the Ia antigen on these cyt T cells was controlled by a locus in the *I-A* subregion. The newly established L1117 and S1509a lines induced Ia-positive cyt T cells, whereas the older SaI line, which was capable of growing across the *H-2* barrier, induced Ia-negative cyt T cells. The results indicate that tumor-specific resistance depends on the balance of activation of cyt and sup T cells with different specificities and phenotypic expressions. (21 refs)

78-4615 **Serologic and Immunochemical Characterization of an Immunosuppressive Soluble Factor from Mastocytoma-bearing Mice.** (Eng) Watanabe, M. (Dept. Microbiology, Albert Einstein Medical Center, York and Tabor Roads, Philadelphia, PA, 19141); Pan, S. H.; Friedman, H. *J Natl Cancer Inst* 61(1): 249-254; 1978.

An immunosuppressive (IS) soluble factor from P815-X2 mastocytoma-bearing DBA/2 mice was characterized by serologic and immunochemical tests. The ascitic fluid from mastocytoma-bearing mice depressed the antibody response to sheep RBC. Antisera prepared against ascitic fluid in rabbits neutralized the IS property of the ascitic fluid. Furthermore, antisera prepared in rabbits immunized with concentrated cell-free culture fluid also neutralized the IS activity of ascitic fluid, which indicated some immunologic relationship between the ascitic fluid and the culture supernatants in terms of IS material. Antisera prepared against extracts of intact tumor cells grown in vivo similarly neutralized the ascitic fluid from tumor-bearing mice, also suggesting a relationship between the IS moiety directly extractable from tumor cells grown sc and/or ip and the cell-free ascitic fluids. Immunoelectrophoretic studies with rabbit serum absorbed with normal DBA/2 serum proteins indicated that a distinct precipitin band could be obtained with the ascitic fluid from the tumor-bearing mice. This precipitin band could be dissociated into two separate precipitin arcs by treatment with proteolytic enzymes, but not with other enzymes. Sephadex G-200 gel filtration at neutral pH indicated that the ascitic fluid could be separated into three peaks. Testing for IS activity showed that all plaque-forming cell-impairing activity had shifted from the first peak (containing high-mol-wt globulins, especially α-globulins) to the third peak (containing lower-mol-wt material) when pH 3.6 buffer was used. After acid dissociations, the mol wt of the IS material was $\leq$5,000, a range similar to that of dialyzable IS material found in culture fluids of mastocytoma cells grown in serum-free medium. It is possible that this low-mol-wt material may be complexed with larger proteins, such as the high-mol-wt carrier α-globulins. (16 refs)

78-4616 **Serum-blocking Factors in Human Prostatic Carcinoma.** (Eng) Kouttab, N. M. (Dept. Molecular Carcinogenesis and Virology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX); Bowen, J. M.; von Eschenbach, A. C.; Johnson, D. E.; Bracken, R. B.; Dmochowski, L. *Cancer Bull* 30(4): 115-118; 1978.

Over 50 sera from patients with prostatic cancer and at least 30 sera from normal donors were analyzed for the presence of blocking (factor(s) that abrogate T-cell-mediated immunity against a target cell). The identity of the factor has not been established and it is not known whether a single factor or multiple factors may be involved. Lymphocyte stimulation by phytohemagglutinin (PHA) and mixed leukocyte cultures (MLC) were used as in vitro assays of T-cell function. In the first assay, the sera of patients with prostatic carcinoma were significantly more inhibitory than normal sera. Fractionation of the sera from the patients and normal donors on Sephadex G200 columns demonstrated at least two factors, one of high mol wt (> 160,000) and one of lower mol wt (50,000-70,000). In the MLC assay, a 60% mean inhibition was obtained with sera from the patients, but the mean inhibition with normal sera did not exceed 14%. It is evident that prostatic cancer is responsible for the presence of significant concentrations of an inhibitory factor(s). However, not all sera of prostatic cancer patients contain high concentrations of this factor. It was concluded that most of the inhibitory activity was due to a high-mol-wt factor that is neither an antibody nor an antigen-antibody complex. (5 refs.)

78-4617 **Hormonal Environment of Immunosuppressed Mice.** (Eng) Williams, G. (Urological Unit, Dept. Surgery, Royal Postgraduate Medical Sch., Hammersmith Hosp., London W12 OHS, England); Ghanadian, R.; Papadopoulos, A. S.; Castro, J. E. *Br J Cancer* 37(1): 123-125; 1978.

Serum concentrations of estradiol, progesterone, and testosterone were measured by radioimmunoassay in normal male and female mice aged 9 and 17 wk and compared with those in mice of similar age who had been made deficient in cell-mediated immunity when 3-4 wk old. In 9-wk-old female mice, there was no significant alteration of serum hormone concentrations in the immunodeficient and normal groups. In 17-wk-old immunosuppressed female mice, there were significantly lower serum estradiol concentrations but no difference in serum testosterone or progesterone concentrations. In 9-wk-old immunodeficient male mice, there was a significant increase in serum progesterone and a significant decrease in serum testosterone concentration compared with normal male mice. Both these differences disappeared by 17 wk. Serum hormone concentrations of male and female mice varied widely in comparison with similar human values, which may account for the failure of certain hormone-dependent tumors to grow in immunosuppressed mice. (12 refs)

78-4618 **Friend Virus-induced Immunodepression: Effect of Neuraminidase Treatment on Thy-1.2 Antigen Expression and Cytotoxic Potential of Splenocytes from Virus-infected Mice.** (Eng) Mascio, A. A. (Dept. Zoology, Drew Univ., Madison, NJ, 07940); Ceglowski, W. S. *Infect Immun* 19(2): 486-492; 1978.

Infection of susceptible female C57Bl/6 and DBA mice with Friend leukemia virus (FLV) resulted in a profound depression of cell-mediated immunity, as assessed by lymphocyte-mediated cytotoxicity. This depression occurred early in the disease, before the onset of splenomegaly, and it was associated with a decline in the susceptibility of splenocytes from FLV-infected mice to lysis by anti-Thy-1.2 serum and complement. Treatment of splenocytes from FLV-infected mice with neuraminidase restored, in large part, their susceptibility to anti-Thy-1.2 serum as well as their cytolytic capacity. These studies suggest that one early immunosuppressive consequence of infection with FLV involves alteration of the effector T-lymphocyte cell surface. (32 refs)

78-4619 **Mechanism of Genetic Resistance to Friend Virus Leukemia in Mice. IV. Identification of a Gene (*Fv-3*) Regulating Immunosuppression In Vitro, and Its Distinction from *Fv-2* and Genes Regulating Marrow Allograft Reactivity.** (Eng) Kumar, V. (Dept. Pathology, Boston Univ. Sch. Medicine, Boston, MA, 02118); Goldschmidt, L.; Eastcott, J. W.; Bennett, M. *J Exp Med* 147(3): 422-433; 1978.

Friend murine leukemia virus (FLV) suppresses the proliferative response of normal lymphocytes to mitogens. The suppression of the mitogenic response is mediated by T suppressor cells and is under genetic control. Genetic analyses using resistant and susceptible parental strains F_1 hybrids, and F_2 intercrosses indicated that susceptibility to in vitro suppression is regulated by a single autosomal gene, *Fv-3*. This gene, which is not linked to the *H-2* histocompatibility complex, segregated independently of the *Fv-2* gene that controls resistance to spleen focus formation in vivo. The gene is also not linked to the immune response genes that regulate the ability of *H-2d* mice to reject *H-2b* bone marrow grafts. *Fv-3* may regulate the numbers and/or functions of T suppressor cells. (24 refs)

78-4620 **Murine Lymphoma-induced Immunosuppression: Requirement for Direct Tumor Cell Contact.** (Eng) Cimprich, R. S. (Dept. Microbiology and Immunology, Albert Einstein Medical Center, Philadelphia, PA, 19141); Specter, S.; Friedman, H. *Science* 200(4337): 60-61; 1978.

FBL-3 lymphoma cells, derived originally from leukemia virus-resistant C57BL/6 mice inoculated at birth with Friend leukemia virus, were examined for their effects on the murine immune response system. Ip injection of as few as 10 FBL-3 cells into C57BL/6 mice resulted in death from lymphoma within 30 days. Depression of antibody formation to challenge with sheep RBC occurred after tumor cell inoculation: tumor-bearing mice showed a progressive decline in antibody formation as a function or time after FBL-3 injection. Spleen cells from normal C57BL/6 mice were then cultured to determine the nature of the immunosuppression. Immunization of the spleen cells with 0.1 ml of a 0.1% suspension of sheep RBC resulted in the rapid appearance of hemolytic plaque-forming cells (PFC). Cocultivation of the normal spleen cells with FBL-3 cells resulted in a marked reduction in the antibody response: 500,000 tumor cells produced a 95% suppression of this response. Prior treatment of intact tumor cells with ≥1,000 rads or with heat (56 C) for 20-30 min completely abolished in vivo and in vitro immunosuppressive activity. Tumor cell homogenates had little effect on immunosuppressive activity. Cell-free filtrates showed no suppressive effect. The direct interaction of tumor cells with normal spleen cells appeared to be necessary for suppression of the hemolytic antibody response. (10 refs)

78-4621 **Effect of the *Fv-1* Locus In Vivo: Host Range Pseudotypes of Murine Sarcoma Virus.** (Eng) Otten, J. A. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Myer, F. E.; Tennant, R. W.; Brown, A. *J Natl Cancer Inst* 60(4): 875-880; 1978.

Fv-1-specific host-range pseudotypes of murine sarcoma virus (MuSV) were developed by rescue from nonproducer cells with N- or B-tropic leukemia viruses. The MuSV(B) and MuSV(N) pseudotype viruses were tested in vitro and were restricted specifically by the *Fv-1* gene locus. When the pseudotype viruses were tested in vivo in mice of specific *Fv-1* genotypes, tumor induction was completely restricted in nonpermissive animals, including athymic nude mice. However, all tumors grew progressively in permissive animals. All tumors regressed in adult heterozygous mice, but not in adult athymic nude mice. These results confirm that the MuSV(B) and MuSV(N) host-range pseudotype viruses are subject to the same intracellular restriction as the N- or B-tropic helper leukemia viruses. Furthermore, the *Fv-1* gene restricts the MuSV(N) pseudotype virus in the absence of a competent thymic function. (19 refs)

78-4622 **Are Tumor-associated Transplantation Antigens of Chemically Induced Sarcomas Related to Alien Histocompatibility Antigens?** (Eng) Pliskin, M. E. (Sch. Dental Medicine, Univ. Pennsylvania, 4001 Spruce St., Philadelphia, PA, 19104); Prehn, R. T. *Transplant Proc* 26(1): 19-24; 1978.

The hypothesis that tumor-specific transplantation antigens of chemically induced tumors cross-react with allogeneic histocompatibility antigens was tested. This hypothesis makes several predictions that can be tested experimentally. First, tumors should grow better and be less immunogenic in certain F_1 hybrids than in their syngeneic parents, owing to the hypothecated cross-reactivity of the tumor-specific transplantation antigens with F_1 antigens. This is in contrast to the more common observation that parental strain tumors grow worse in the F_1 hybrids than they do in the parent. Also, certain allogeneic skin grafts might immunize the parental strain mice against their syngeneic tumors, and, finally, immunizing parental mice with syngeneic tumors might cause accelerated rejection of certain skin allografts. The results show that certain methylcholanthrene-induced sarcomas grew better in C/C3H F_1 mice than they did in the BALB/c parents but that the tumors were not less immunogenic in the F_1 hybrids. Mice immunized against alloantigens showed a dose-dependent enhancement of syngeneic tumor growth. Finally, mice immunized with syngeneic tumors demonstrated an apparent prolongation of certain skin allografts. Possible alternatives explaining these results are considered. (50 refs.)

78-4623 **Temperature Sensitivity of the Specific Transplantation Antigens of Tumor Cell Membrane.** (Rus) Kliuchareva, T. E. (Lab. Tumor Immunology, Cancer Res. Center, Moscow, USSR). *Biull Eksp Biol Med* 85(3): 351-353; 1978.

A study was made of the effect of temperature on the immunogenic properties of the tumor-specific transplantation antigen (TSTA) in simian virus 40 (SV40)-induced tumors. The tumor cells were incubated at 37 C and 56 C for 30 min, irradiated at 12,000 rads, and then injected into syngeneic hamsters. The immunogenic properties of the TSTA were assessed by a transplantation test. Administration of irradiated cells incubated at 37 C to hamsters significantly increased their specific resistance; incubation of cells at 56 C resulted in complete inactivation of TSTA. (7 refs)

78-4624 **Protection Against Syngeneic Tumor Grafts Induced by Inoculation with Normal Allogeneic Tissues.** (Eng) Bear, R. H. (Dept. Immunology Res., Roswell Park Memorial Inst., Buffalo, NY, 14263); Roholt, O. A.; Pressman, D. *Immunol Commun* 6(6): 547-558; 1977.

The protection offered by sc injection of normal allogeneic kidney and liver tissues against syngeneic tumor grafts was investigated in DBA/2Cr mice. Female mice were given x χ 10^4 viable methylcholanthrene-induced L5178Y lymphoma cells ip and 0.5 ml of an allogeneic mixture of liver and kidney tissues from BALB/cCr, A/J, C57BL/6Ha, ARK/Sn, and DBA/2Ha-DD mice. Following challenge and immunization, 42% of the mice were alive after 100 days; controls immunized with syngeneic DBA/2Cr kidney and liver tissue survived a mean of 13 days. X-irradiation of the allogeneic cell mixture (10^4 R) prior to immunization gave the same protection as the unirradiated mixture. The allogeneic mixture did not confer protection against 10^4 L1210 cells in DBA/2Cr mice, but it did confer partial protection against 10^6 L1210 cells in DBA/2Ha-DD mice. A combination of 2 ml of the allogeneic mixture and 8 ml of the syngeneic cells was also administered, and 86% of the mice were alive 100 days after challenge. The protection was comparable to that obtained by immunization with irradiated L5178Y tumor cells. Analysis of the protection conferred by each individual strain indicated that none of the strains alone were as effective as the allogeneic mixture from the five different strains. Therefore, the protection is potentiated when the tissues are combined. It is suggested that this protection is the result of a cross-reactivity or identity between L5178Y tumor-associated transplantation antigens and nonhistocompatibility alloantigens normally expressed by some allogeneic tissues. (16 refs)

78-4625 **Loss of Oncogenicity and Concomitant Increased Immunogenicity of Murine Plasmacytoma Cell Lines.** (Eng) Sorenson, G. D. (Dept. Pathology, Dartmouth Medical Sch., Hanover, NH, 03755); Pettengill, O. S.; Cate, C. C. *Am J Pathol* 90(3): 565-582; 1978.

Long-term observations of BALB/c mouse MOPC-21 and MOPC-315 plasmacytoma cells showed that the cells lost their oncogenicity with increasing time in culture. Newly established cultures were oncogenic and caused lethal tumors in syngeneic mice. Later, although the cultures were still oncogenic, some of the tumors regressed. Eventually, the cultures became nononcogenic in normal mice, but they did cause tumors in irradiated mice. Clones from the cultures varied from being very oncogenic to nononcogenic. It appears that the nononcogenic cells have a selective growth advantage and that they become the dominant cell type in the cultures. Nononcogenic cultures were more effective for immunization to subsequent challenge with the original tumor than were the tumor cells or oncogenic cultures. They appeared to confer complete protection against lethal tumor challenge in adoptive transfer experiments. Mixtures of nononcogenic and oncogenic cells decreased the tumor-forming ability of the latter when they were injected together in one site or separately in two sites. (39 refs)

See also:
*(Rev.): 78-4212.
*(Chem.): 78-4284, 78-4287.
*(Viral): 78-4387, 78-4391, 78-4400, 78-4403, 78-4406, 78-4414, 78-4420, 78-4431, 78-4441, 78-4443, 78-4448, 78-4467, 78-4474, 78-4478, 78-4480, 78-4498, 78-4501, 78-4505, 78-4507, 78-4508, 78-4512, 78-4513, 78-4527, 78-4529, 78-4533.
*(Path.): 78-4627, 78-4636, 78-4638, 78-4644, 78-4647, 78-4650, 78-4651.
*(Epid.-Biom.): 78-4673, 78-4676, 78-4688.

78-4626 **Chromosomes of Lymph Node Metastases Derived from Ehrlich Ascites Carcinoma Cells Inoculated into Rats.** (Eng) Olinici, C. D. (Inst. Oncology, R-3400 Cluj-Napoca, Romania); Risca, R.; Todorutiu, C. *Z Krebsforsch* 90(3): 281-284; 1977.

Chromosomes from lymph node metastases in female Wistar rats that had received 100 x 10^6 Ehrlich ascites tumor cells ip were examined. The rats were given 600 R whole-body irradiation 1 day before inoculation and five ip injections of 5 mg cortisone (total dose 25 mg) starting on the day of inoculation. The animals were sacrificed 7 and 15 days after grafting. The incidence of rat cells in the ascites fluid increased from 16% on day 7 to 36% on day 15. The incidence of rat metaphases was considerably greater in the lymph nodes: 29% on day 7 and 69% on day 15. Chromosome-banding analysis indicated that the tumor cells in the lymph nodes had the typical constitution of Ehrlich cells, as ascertained by three marker chromosomes. No host-derived chromosomes were noted in these cells. The rat cells contained 42 species-characteristic chromosomes. (8 refs)

78-4627 **Genetic Studies of the X-linked Recessive Lymphoproliferative Syndrome (XLRLS) (Meeting Abstract).** (Eng) Paquin, L. A. (Univ. Massachusetts Medical Sch., Amherst, MA); Purtilo, D. T. *Genetics* 88(4, part 2, Suppl): s76; 1978. (1 ref)

78-4628 **Genetic Analysis of Tumorigenesis: Expression of Tumor-Forming Ability in Hamster Hybrid Cell Lines.** (Eng) Sager, R. (Sidney Farber Cancer Inst., Dept. Microbiology, Harvard Medical Sch., Boston, MA); Kovac, P. E. *Somatic Cell Genet* 4(3): 375-392; 1978.

The characteristics of two new fibroblast cell lines and hybrids formed between these lines were studied. The two cell lines, which were established from a Chinese hamster embryo, were designated CHEF 16-2 and CHEF 18-1. The cell lines differed in colony morphology, plating efficiency, anchorage dependence of growth, tumorigenic potential in nude mice (16-2 was tumorigenic and 18-1 was not), and modal chromosome number. The most striking difference was that the 16-2 cells cloned at high frequencies (30%-60%) in methylcellulose but the 18-1 cells did not form colonies at a detectable frequency ($< 10^{-5}$). Mutagenesis in ethylmethane sulfonate (EMS) and selection of 6-thioguanine (TG)-resistant 18-1s and 5-bromo-2-deoxyuridine (BUdR)-resistant 16-2s lines did not alter the principal distinguishing properties of the parental lines. The 22 chromosomes in both parental lines consisted of 10 pairs of homologous autosomes plus an X and a Y. As few as 10 CHEF 16-2 cells gave rise to tumors in nude mice, provided that they were coinjected with x-irradiated 18-1s cells, which fulfilled an unknown function. Hybrid colonies more closely resembled the nontumorigenic parent than the tumorigenic parent, and each hybrid clone was a mixture of cells with different levels of anchorage dependence. The 19 hybrid clones tested were unambiguously tumorigenic in the nude mouse, although they showed a heterogeneity in rate of tumor formation. Nine subclones derived from three hybrid lines showed a decrease in chromosome number from the tetraploid range of the hybrids to the diploid range. The tumor subclones appeared to be tumorigenic subsets in the suppressed hybrid populations. Evidence that the hybrids were of hybrid rather than parental origin is provided. (30 refs.)

78-4629 **Chromosome Reduction in Selected Mouse and Chinese Hamster Cell Hybrids (Meeting Abstract).** (Eng) Howell, N. (Sidney Farber Cancer Inst., Boston, MA); Kovac, P.; Sager, R. *Genetics* 88(4, part 2, Suppl): s45-s46; 1978. (no refs)

78-4630 **A New Cytogenetic Finding in Bloom's Syndrome: Terminal Association of Homologous Chromosomes at Metaphase (Meeting Abstract).** (Eng) Schonberg, S. (New York Blood Center, New York, NY); German, J.; Chaganti, R. S. *Genetics* 88(4, part 2, Suppl): s88-s89; 1978. (no refs)

78-4631 **A Mammalian Cellular System for the Concomitant Study of Neoplastic Transformation and Somatic Mutation.** (Eng) Barrett, J. C. (Natl. Inst. Environmental Health Sciences, NIH, P.O. Box 12233, Research Triangle Park, NC, 27709); Bias, N. E.; Ts'o, P. O. *Mutat Res* 50(1): 121-136; 1978.

Neoplastic transformation and somatic mutation were studied in strain LSH/ssLAK Syrian hamster embryo cells. Chemically induced somatic mutation was assayed at two genetic loci: mutants deficient in hypoxanthine phosphoribosyltransferase (HPRT) were detected by the production of colonies resistant to 40 μg/ml 8-azaguanine (AG-r) or 2 μg/ml 6-thioguanine (TG-r); mutants with an altered Na^+/K^+ ATPase were detected by the production of colonies resistant to 1.25 mM ouabain (Oua-r). The results showed that AG-r/TG-r cells possess a mutation in a structural or regulatory gene for HPRT and that the Oua-r cells have an altered Na^+/K^+ ATPase. This finding is based on the evidence that: (1) the isolated clones had stable, heritable resistance to the selective drugs; (2) the AG-r clones were cross-resistant to 6TG; (3) the reversion frequency of the AG-r and TG-r clones was $< 10^{-6}$; (4) the HPRT activity of the AG-r/TG-r clones was $< 1\%$ of the parental cells; (5) Oua-r clones showed decreased sensitivity to Oua inhibition or ^{86}Rb uptake (compared to wild-type cells) and they maintained their resistance to Oua; and (6) the frequency of resistant clones was increased by known mutagens in a dose-dependent manner. Factors involved in quantification of the mutation frequencies of the embryo cells were determined following exposure of the cells to benzo(a)pyrene (BP: 1 or 10 μg/ml) or N-methyl-N′-nitro-N-nitrosoguanidine (MNNG: 1 or 5 x 10^{-6} M). BP or MNNG treatment before selection increased the frequency of resistant colonies significantly. This system allows a quantitative comparison of the frequencies of somatic mutation and morphological transformation. (42 refs)

78-4632 **Presence of Retinoic Acid-Binding Protein in Experimental Tumors and in Tissues with Metastatic Tumor (Meeting Abstract).** (Eng) Sani, B. P. (Southern Res. Inst., Birmingham, AL, 35205). *Proc Am Assoc Cancer Res* 19: 19; 1978. (no refs)

78-4633 **Some Mechanisms Involved in Cancer Cell Detachment by Necrotic Material.** (Eng) Weiss, L. (Dept. Experimental Pathology, Roswell Park Memorial Inst., Buffalo, NY, 14263). *Int J Cancer* 22(2): 196-203; 1978.

Previous studies of cylinders punched from Walker 256 (W-256) tumors growing in rats showed that detachment of viable cancer cells was facilitated by their proximity to necrotic regions and exposure to a necrotic extract. In these experiments, pure cultures of W-256 cells were used to show that necrotic extracts that are rich in lysosomal hydrolases act directly on W-256 cells, facilitating their in vitro detachment. Lysosomal enzymes are known to facilitate cell detachment. The partial inhibition of this process by hydrocortisone suggests that the extracts also acted by labilizing the W-256 cell lysosomes; this enzyme release was also demonstrable by direct assay. Although secondary lysosomes could not be visualized in the W-256 cells, both they and macrophages released lysosomal hydrolases upon freeze-thawing. Both could account for the high enzyme content of the necrotic regions, by a process of continuous secretion and/or by cell input. Histologic examination of the perinecrotic and peripheral regions of tumors showed a higher proportion of macrophages in the former, probably because of differential trapping of the macrophages. The results emphasize the functional properties of necrotic tumor regions and the necessity of referring to specific regions of the same tumor when alluding to its metastatic and other properties. (35 refs.)

78-4634 **Suppression of Malignancy in Intraspecific Human Cell Hybrids: The Generality of the Phenomenon (Meeting Abstract).** (Eng) Weissman, B. E. (Dept. Medical Microbiology, Univ. California, Irvine, CA, 92717); Stanbridge, E. J. *Proc Am Assoc Cancer Res* 19: 51; 1978. (no refs)

78-4635 **Identification of Mouse Chromosomes Which Cannot Suppress Malignancy in Interspecific Hybrid Cells (Meeting Abstract).** (Eng) Moss, W. (Medical Oncology Branch [DCT, NCI], Veterans Admin. Hosp., Washington, DC, 20422); Lalley, P.; Francke, U. *Proc Am Assoc Cancer Res* 19: 117; 1978. (no refs)

78-4636 **Spontaneous Tumors and Human Tumor Metastases in Nude Mice (Meeting Abstract).** (Eng) Fogh, J. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Filippa, D.; Sharkey, F. E. *Proc Am Assoc Cancer Res* 19: 211; 1978. (no refs)

78-4637 **Splenectomy in CML and Suppression of Leukemic Clones: Cytogenetic Studies (Meeting Abstract).** (Eng) Venuat, A-M. (INSERM, Hopital Paul-Brousse, 94800 Villejuif, France); Pico, J-L.; Mathe, G. *Proc Am Assoc Cancer Res* 19: 179; 1978. (no refs)

78-4638 **Characterization of a Suppressor-Cell Leukemia. Evidence for the Requirement of an Interaction of Two T Cells in the Development of Human Suppressor Effector Cells.** (Eng) Broder, S. (Metabolism Branch, NCI, Bethesda, MD 20014); Poplack, D.; Whang-Peng, J.; Durm, M.; Goldman, C.; Muul, L.; Waldmann, T. A. *N Engl J Med* 298(2): 66-72; 1978.

The suppressor-cell activity of neoplastic T cells from an 11-mo-old boy with acute lymphoblastic leukemia and hypogammaglobulinemia was investigated. The patient's leukemic cells did not secrete detectable immunoglobulin (Ig) in vitro, but the lymphocytes of 12 normal persons secreted 3,200 nanogram (ng) of IgM, 1,825 ng of IgA, and 2,447 ng of IgG. The addition of neoplastic or irradiated neoplastic T cells to normal B cells did not stimulate Ig synthesis, indicating that the T cells were not committed to express helper activity. However, the cells were committed to suppressor activity, as the addition of 10^6 neoplastic lymphocytes profoundly suppressed Ig synthesis in equal numbers of lymphocytes from six normal persons. Suppressor cell activity was nullified by prior irradiation (2,000 R), and it was a function of the number of the neoplastic cells added to the normal cells. In a pokeweed mitogen-stimulated system of Ig secretion, neoplastic T cells caused suppression only when unirradiated normal T cells were provided. When irradiated or unirradiated normal T cells were added as a source of helper activity, Ig production returned to normal for the unseparated lymphocytes. These findings indicate the existence of two populations of radiosensitive cells: the neoplastic T cells from the patient and a subset of cooperating normal T cells. (28 refs.)

78-4639 **Studies of Genetic Suppression of AKR Mouse Leukemia.** (Eng) Lilly, F. (Dept. Genetics and Pathology, Albert Einstein Coll. Medicine, Bronx, NY, 10461); Duran-Reynals, M. L.; Mayer, A. J.; Bosch, A. *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 55-59; 1978.

Crosses between various low-leukemic mouse strains and the high-leukemic AKR strain were studied to determine relationships between the occurrence of spontaneous leukemia and the genetically controlled expression of ecotropic and/or xenotropic murine leukemia viruses (MuLV) in lymphoid tissues. The incidence of leukemia in the F_1 progeny resulting from AKR x ST/b crosses essentially paralleled that in AKR mice, indicating that ST/b mice possess no dominant gene(s) capable of suppressing the AKR phenotypes under consideration. However, F_1 mice of the AKR x BALB/c cross showed a leukemia incidence of $<5\%$, indicating that one or more dominant genes of the BALB/c complement must be capable of suppressing this phenotype. The phenotype appears to be a function of the Fv-1b allele. F_1 progeny of the AKR x RF/J cross showed high levels of infectious ecotropic MuLV but a low incidence of leukemia. The results of backcrosses of (AKR x RF) with AKR suggested that the Fv-1 allelle of RF mice is capable of suppressing the AKR leukemia phenotype. The allele may exert its effect by suppressing the expression of xenotropic MuLV. The data suggest that the expression of both ecotropic and xenotropic viruses is necessary for AKR leukemogenesis. (15 refs.)

78-4640 **Changes in the Karyotype of Bone Marrow Cells in the Progeny of Leukemic Cows.** (Rus) Burba, L. G. (All-Union Inst. Experimental Veterinary Medicine, Moscow, USSR); Dun, E. A.; Simonian, G. A. *Veterinariia* (3): 43-46; 1978.

The bone marrow and blood cells of 22 calves born to leukemic cows were investigated at 3-mo intervals. Changes in the chromosome complement of the bone marrow cells as well as subleukemic and leukemic changes in the blood were found in 27% of the calves. Considerable deviation from the diploid cells, which ranged from 40% to 70%, tended to rise with age. The number of hypodiploid and hyperdiploid metaphases increased significantly, but the percentage of aneuploid metaphases decreased with age. The findings confirm the significance of genetic disposition in leukemogenesis. (no refs)

78-4641 **Karyotypic Analysis of Lymphoid Blood Cells in Leukemic Cows.** (Rus) Birbin, S. S. (Veterinary Res. Station, Saratov, USSR). *Veterinariia* (5): 37-40; 1978.

The incidence of chromosome aberrations was studied in 39 leukemic cows, 24 in the initial (hematologic) stage and 15 in the advanced stage (hyperplasia of lymphoid tissue and the spleen). All of the latter had chromosome aberrations (predominantly in lymphoid blood cells). The aberrations included hyperploidy (85%-99% of examined metaphases; 11/15 cows had 61 chromosomes (compared with 60 chromosomes in the normal complement)) and chromosome rearrangements (metacentric and submetacentric chromosomes). (no refs.)

78-4642 **Cellular Maturation in Human Preleukemia.** (Eng) Koeffler, H. P. (Div. Hematology-Oncology, Dept. Medicine, UCLA Sch. Medicine, Los Angeles, CA, 90024); Golde, D. W. *Blood* 52(2): 355-361; 1978.

Bone marrow cells from three preleukemic patients (1 woman aged 25, 2 men aged 78 and 79) with prominent marrow karyotypic abnormalities were studied in liquid culture to determine if the neoplastic clones were capable of maturation. Parallel cytogenetic and cytologic studies were performed in sequentially harvested bone marrow cultures. Maturation, albeit delayed, occurred in cultures from all three patients. By 14 days of culture in vitro, morphologic, cytochemical, and functional evidence of maturation was observed in about 70% of the cells. By day 21, 85% of the cells were mature. All but 2 of 249 metaphases from the cultured cells contained the cytogenetic abnormality of the neoplastic clone. It is concluded that some preleukemic cells identified by a chromosomal abnormality can mature in vitro. Preleukemia may be viewed as a syndrome of 'early leukemia' in which the neoplastic clone is established and manifested functionally as ineffective hematopoiesis. Hematopoietic cell differentiation becomes progressively abnormal, terminating in the nearly complete maturational block characteristic of acute myelogenous leukemia. (30 refs.)

78-4643 **Blood Dyscrasias and Childhood Tumors and Exposure to Chlordane and Heptachlor.** (Eng) Infante, P. F. (Office Carcinogen Identification and Classification, Occupational Safety and Health Admin., U.S. Dept. Labor, 3rd and Constitution Ave., NW, Washington, DC, 20210). *Scand J Work Environ Health* 4(2): 137-150; 1978.

Pathological conditions possibly associated with exposure to chlordane- and heptachlor-containing pesticides are discussed. Five recently diagnosed cases of neuroblastoma associated with chlordane exposure during prenatal and postnatal development are summarized, as are three cases of aplastic anemia and three cases of acute leukemia associated with chlordane formulations. The neuroblastoma cases are of interest in view of recent data on the carcinogenicity and mutagenicity of chlordane and heptachlor and data on environmental distributions and body burdens. The aplastic anemia and leukemia cases are of interest in view of previous reports on associations between chlordane and similar chlorinated hydrocarbon pesticides and blood dyscrasias and reports indicating that some subjects with aplastic anemia convert to leukemia. There is need for a formal epidemiologic study to evaluate the health risks associated with domestic and occupational exposure to chlordane. (63 refs)

78-4644 **Acute Myeloid Leukaemia with Monosomy-7 Follows Acute Lymphoblastic Leukaemia.** (Eng) Secker Walker, L. M. (Dept. Cytogenetics and Immunology, Royal Marsden Hosp., Fulham Road, London SW3 6JJ, England); Sandler, R. M. *Br J Haematol* 38(3): 359-366; 1978.

The case report of an 8-yr-old girl who developed acute myeloid leukemia (AML) following the successful treatment of acute lymphoblastic leukemia (ALL) is presented. She presented with ALL at the age of 5. Remission was induced with prednisolone, vincristine, radiation (2,400 rads to the cranium, 1,000 rads to the spine), and intrathecal methotrexate. She remained well on maintenance therapy, including 600 mg/m^2 cyclophosphamide every 12 wk, for 2 yr, at which time she became pancytopenic with marrow depression. Bone marrow remission was induced by the cessation of chemotherapy, but the pancytopenia remained. Marrow cells monosomic for chromosome 7 (45 XX-7) were first noted 2 mo before the onset of progressive pancytopenia when blood parameters were normal. During both the pre-leukemic and frankly leukemic states of the myeloid neoplasm, cells with monosomy-7 constituted the dominant cell line in the marrow. This suggests that the pancytopenia was part of the malignant change and that the chromosomal change was an early event in the neoplastic process. Family studies indicated that the patient's paternal grandmother had died of acute leukemia and that there had been a high cancer incidence in her maternal grandfather's generation. It is suggested that genetic factors may have been at least as important as environmental conditions in triggering the second malignancy. (31 refs)

78-4645 **Metamorphosis of Chronic Granulocytic Leukaemia Arising in an Extramedullary Site.** (Eng) Kwan, Y. L. (Medical Res. Dept., Kanematsu Memorial Inst., Sydney Hosp., Sydney, New South Wales 2000, Australia); Singh, S.; Vincent, P. C.; Gunz, F. W. *Leuk Res* 1(4): 301-307; 1977.

The metamorphosis of chronic granulocytic leukemia was followed in a 52-yr-old man. The diagnosis was based on typical leukocytosis, a hypercellular marrow with myeloid hyperplasia, and a single Philadelphia (Ph^1) chromosome in 100% of the marrow metaphases. Elective splenectomy was performed early in the disease because of persistent mild splenomegaly and a tendency toward thrombocytopenia. As the disease progressed, extramedullary leukemic deposits were noted in the stomach, skin, and lymph nodes. Marked chromosome abnormalities (hyperdiploidy and doubling of the Ph^1 chromosome) were noted in a biopsied axillary lymph node at a time when marrow chromosomes showed only a 46XY Ph^1 positive pattern typical of the chronic phase. With disease progression, cells with related chromosome abnormalities appeared in the blood. The sequence of events and cytogenetic changes suggested that the cells responsible for the metamorphosis appeared first in an extramedullary site and spread from there to the marrow and blood. (5 refs)

78-4646 **Genetic and Pathologic Findings in a Kindred with Hereditary Sarcoma, Breast Cancer, Brain Tumors, Leukemia, Lung, Laryngeal, and Adrenal Cortical Carcinoma.** (Eng) Lynch, H. T. (Dept. Preventive Medicine/Public Health, Creighton Univ. Sch. Medicine, Omaha, NB, 68178); Mulcahy, G. M.; Harris, R. E.; Guirgis, H. A.; Lynch, J. F. *Cancer* 41(5): 2055-2064; 1978.

Pathologic and genetic studies were carried out on a family with a hereditary tumor complex characterized by sarcomas, brain tumors, leukemias, and carcinomas of the breast, larynx, lung, adrenal cortex, and other sites. Based on sibships segregating for cancer, the genetic segregation parameter was estimated as 45.5% ± 11%, which is consistent with that expected for a rare deleterious autosomal gene showing complete dominance. A microscopic review of 16 tumors revealed intranuclear cytoplasmic invaginations, intranucleolar bodies, or acidophilic inclusions in 8 lesions. Two tumors showed both intranuclear cytoplasmic invaginations and intranucleolar inclusions. It is hypothesized that the cancer-prone genotype interacts with one or more exogenous factors, including an oncogenic virus, in causing this familial tumor aggregation. (39 refs)

78-4647 **Kaposi's Sarcoma in Renal Allograft Recipients.** (Eng) Stribling, J. (Dept. Surgery, Univ. Mississippi Medical Center, 2500 N. State St., Jackson, MS, 39216); Weitzner, S.; Smith, G. V. *Cancer* 42(2): 442-446; 1978.

Kaposi's sarcoma developed in a 61-yr-old man who had been a renal allograft recipient 9 mo previously. Eight similar cases in the literature are reviewed. In all nine patients, the interval between transplantation and diagnosis of the sarcoma varied from 8 to 43 mo; however, in six patients the interval was 8-12 mo. It was hypothesized that chronic immunologic interaction between normal and antigenically altered or transformed lymphocytes (as in a graft-vs-host reaction) results in the production of an angiogenesis factor that produces intense proliferation of mesenchymal and endothelial cells, eventually leading to Kaposi's sarcoma. It has also been suggested that a chronic low-grade immune response rather than severe crises are important, thus invoking the role of enhancing or blocking factors. Viral etiologies have also been postulated. The patients presented met the criteria of chronic immunologic stimulation, probably had altered lymphocytes because of suppressive therapy, and nearly half developed viral infections. However, development of malignant neoplasms following renal transplantation appears to depend on immunosuppressive therapy, since the oral lesions in three patients and the cutaneous lesions in one regressed completely upon cessation of this therapy. (11 refs.)

78-4648 **Metastatic Heterogeneity of Cells from an Ultraviolet Light-induced Murine Fibrosarcoma of Recent Origin.** (Eng) Kripke, M. L. (Cancer Biology Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501); Gruys, E.; Fidler, I. J. *Cancer Res* 38(9): 2962-2967; 1978.

The parent line and 21 cell clones isolated from fibrosarcoma 2237 (recently produced by chronic UV irradiation in a female C3H- mouse) were tested in specific-pathogen-free C3H/HeN(MTV-) mice to determine if any variations existed in their metastatic potential. Three different in vivo tests were used: (1) mice were given iv injections of 10^5 tumor cells and killed 18 days later, and tumor colonies in the lungs were counted; (2) mice were given sc injections of 10^6 tumor cells and examined at death for the presence of spontaneous metastases; and (3) mice were given iv injections of 10^6 tumor cells and autopsied at death. The time of death was recorded, and the number and site of metastases were determined. The clones varied greatly in their ability to grow and metastasize following sc or iv inoculation. The three tests gave similar results in that clones judged to be of high or low metastatic potential in one test generally exhibited the same behavior in the other two tests. It was concluded that the parent tumor was heterogeneous and that cells with widely different metastatic potential preexisted in the parental population. The correspondence among the three tests also permitted the conclusion that for this tumor, the quantitative lung colony assay approximated rather well the more tedious measurements of spontaneous metastasis. (24 refs.)

78-4649 **Malignant Melanoma and Genetic Heterogeneity: A New Familial Melanoma Syndrome (Meeting Abstract).** (Eng) Lynch, H. T. (Creighton Univ. Sch. Medicine, Omaha, NB, 68178); Frichot, B. C.; Guirgis, H. A.; Lynch, P. M.; Follet, K. *Proc Am Assoc Cancer Res* 19: 348; 1978. (no refs)

78-4650 **Metastasis, An Experiment with a Transplantable Tumor in F1 Hybrids of C3H x AJ Mice (Meeting Abstract).** (Eng) Pringgoutomo, S. (Dr. Cepto Mangunkusumo Hosp., JL, Diponegoro 71, Jakarta, Indonesia); Suprapti, S. P. *In: Summary Program and Abstracts of Papers. Proceedings of the 3rd Asian Cancer Conference Held by the Philippine Cancer Society under the Auspices of the Asian Federation of Organizations for Cancer Research and Control in Manila, 26-30 September, 1977.* Philippine Cancer Society. (Manila, Philippines): 184 pp.; 128-129; 1977. (no refs)

78-4651 **Serum Genetic Markers in a Newfoundland Isolate with a Familial Aggregate of Hodgkin's Disease.** (Eng) Carter, N. D. (Dept. Child Health, St. George's Hosp., Medical Sch., Cranmer Terrace, London, SW17, ORE, England); van Loghem, E.; Marshall, W. H.; Newton, R. M.; West, C. M. *Hum Hered* 28(5): 372-379; 1978.

Genetic markers [immunoglobin (Ig) allotypes Gm, Am, and Km and three other serum proteins, group-specific component (Gc), transferrin, and haptoglobin] were investigated in members of an isolated Newfoundland community, most of whom (> 1,200) were related. A familial aggregate of lymphoma and immunodeficiency diseases was discovered among individuals that belonged to this extended family. Blood samples from nearly 1,000 individuals were studied. Gene frequencies for the serum proteins showed no significant deviation from European frequencies. The finding of a variant transferrin in six individuals is in accord with the ancestral origin (English) of the community. However, the occurrence of rare phenotypes [A2m(2) and Gm (zfngb)] pointed toward a specific admixture from American Indians and Scandinavians. An American Indian admixture has been documented in the pedigrees. Transmission of the Ig marker Gm (zfngb) to Newfoundland from Scandinavia via the Orkney Islands has been proposed. A possible contribution of these rare alleles and of others to pathogenesis in a familial aggregate of Hodgkin's disease within this community is discussed. The hypothesis is that predisposition to Hodgkin's disease is likely to be based on a complex genotype with contributions from many different loci. This aggregate of cases in an inbred, genetically isolated community suggests that, due to chance, a number of alleles capable of contributing to a predisposition for Hodgkin's disease have come together in this family. (10 refs.)

78-4652 **Intestinal Lymphoma Associated with Malabsorption (Letter to Editor).** (Eng) Cooper, B. T. (Dept. Medicine, Hammersmith Hosp., London W12, England); Holmes, G. K.; Cooke, W. T. *Lancet* 1(8060): 387; 1978.

The results of an unpublished review of 27 patients with lymphoma and celiac disease are in agreement with previous studies, and they indicate that patients with flat jejunal mucosa, malabsorption, and lymphoma should be evaluated for preexisting celiac disease. However, by the time a lymphoma becomes clinically manifest, it is usually too late for a response to a gluten-free diet. (1 ref)

78-4653 **Prostaglandins and Actinic Cancer (Letter to Editor).** (Eng) Smith, E. B. (Dept. Medicine, Sch. Medicine, Univ. New Mexico, Albuquerque, NM, 87131); Mason, W. V.; Goodwin, J. S. *Lancet* 1(8063): 561; 1978.

A role for prostaglandin E_2 (PGE_2) in the pathogenesis of skin tumors is suggested. PGE_2 released in the skin following UV exposure may suppress immune responses in the skin and interfere with immunological surveillance. This effect would offset any protective effects of PG formation. It could also explain the manifestations of viral exanthems in light-exposed skin. (11 refs)

78-4654 **Bone Metastases from Occult Primary Cancer (Meeting Abstract).** (Eng) Shank, B. (Dept. Radiation Therapy, Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Batata, M. A.; Chu, F. C. *Proc Am Assoc Cancer Res* 19: 314; 1978. (no refs)

78-4655 **Extraneuraxial Canine Meningioma. A Light and Electron Microscopic Study.** (Eng) Vuletin, J. C. (Dept. Pathology, Downstate Medical Center, 450 Clarkson Avenue, Brooklyn, NY, 11203); Friedman, H.; Gordon, W. *Vet Pathol* 15(4): 481-487; 1978.

Light and electron microscopic features of an sc extra-neuraxial meningioma removed from the shoulder of a 7.5-yr-old German Shepherd crossbred male dog are described. No local recurrence or metastases were found 6 mo after surgery. The tumor probably arose from arachnoid cap cells displaced during embryogenesis. (23 refs.)

78-4656 **Granular Cell Tumor of the Brain.** (Fre) Pialat, J. (Laboratoire d'Anatomie Pathologique et Neuropathologie, Faculte de Medecine A. Carrel, Rue Guillaume Paradin, F 69372 Lyon Cedex 2, France); Carrier, H.; Pierluca, P.; Chazot, G.; Trillet, M.; Kopp, N. *Lyon Med* 239(5): 269-274; 1978.

A case is reported of primary granular cell tumor of the brain in a 61-yr-old man. Concerning the pathogenesis of these tumors, a Schwann cell origin seems most probable. (no refs)

78-4657 **Connexions Between Nerves and Intradermal Nevi (Meeting Abstract).** (Eng) Jurecka, W. (Dept. Electron Microscopy, Univ. Vienna, Vienna, Austria); Lassmann, H.; Gebhart, W. *Arch Dermatol Res* 261(1): 91; 1978. (no refs)

78-4658 **Tumor Caused by a Foreign Body in the Nasal Fossa and Maxillary Sinus.** (Pol) Lukomski, M. (Klinika Laryngologiczna, Instytut Chorob Ukladu Nerwowego i Narzadow Zmyslow AM, ul. Kopcinskiego 22, 90-153 Lodz, Poland); Makowski, A. *Otolaryngol Pol* 31(1): 73-75; 1977.

Diffuse squamous cell carcinoma, with chronic inflammation and focal erosion, and a corroded metal object measuring nearly 4 cm in length were found in the left nasal fossa and left maxillary sinus of a 68-yr-old man. He had presented with recurrent headache and exophthalmus of the left eye 3 yr earlier. The carcinoma is believed to have developed from the chronic irritation caused by the metal object and the compounds formed by its corrosion. (13 refs)

78-4659 **Estrogen Profiles in Young Women: Effect of Maternal History of Breast Cancer.** (Eng) Morgan, R. W. (Dept. Preventive Medicine, Univ. Toronto, Toronto, Ontario M5S 1A8, Canada); Vakil, D. V.; Brown, J. B.; Elinson, L. *J Natl Cancer Inst* 60(5): 965-967; 1978.

Estrone (E_1), estradiol (E_2), and estriol (E_3) concentrations were determined in the urine of daughters of breast cancer patients and their matched controls. These findings were then used to compute the estriol proportions (EP; $E_3/E_1 + E_2 +$

E$_3$). For all women, the EP rose to a peak at age 26-30 yr and then declined slightly. Parity had no consistent effect on EP. Study of the daughters and their matched controls showed no differences in EP. It is concluded that the estrogen profile is not an important determinant of familial breast cancer. (10 refs)

78-4660 **Identic Viral Infections in Four Cases of Malignant Lymphoepithelioma.** (Eng) Arnold, W. (ENT Dept., Univ. Dusseldorf, Moorenstrasse 5, D-4000 Dusseldorf, W. Germany). *Arch Otorhinolaryngol (NY)* 220(4): 287-289; 1978.

Electron microscopy of tumor tissue from four patients with malignant lymphoepithelioma of the nasopharynx and tonsil revealed herpesviruslike nucleocapsids in the nucleoplasm and empty capsids in the cytoplasm. These particles were similar to those found in tissue culture lines of Burkitt's lymphoma and nasopharyngeal carcinoma. These findings, together with the observation that Epstein-Barr virus antibody levels change during the course of lymphoepithelioma, suggest a role for a specific viral infection in the disease. (5 refs.)

78-4661 **Clinical Findings and Morphology of Precancerous States and Early Cancers of the Larynx.** (Pol) Szlezak, L. (Oddzial Otolaryngologiczny, Szpital im. Fr. Raszeji ZOZ Poznan-Jezyce, Grodziska 18a m. l., 60-363 Poznan, Poland); Przybora, L.; Ryglewicz, J. *Otolaryngol Pol* 31(3): 305-316; 1977.

Precancerous and cancerous changes of the larynx were studied in 151 patients. Polyps with pachyderma or leukoplakia were found in 28 patients; all smoked 10-20 cigarettes daily. Polyps with pachyderma, cellular dysplasia and early cancer were found in one patient. Pachyderma and leukoplakia were found in 44 patients; all smoked 10-20 cigarettes a day. Pachyderma, leukoplakia with cell proliferation, and dysplasia were seen in 26 patients; all smoked 20-40 cigarettes a day. Pachyderma, leukoplakia with cellular dysplasia, and early cancer were seen in 19 patients. Malignant transformation was found in 11/33 adults with papilloma. (21 refs)

78-4662 **Histogenesis of Adenomas of the Parathyroid Gland.** (Rus) Lebkova, N. P. (Central Inst. Advanced Medical Training for Physicians, Moscow, USSR); Uranova, E. V. *Arkh Patol* 40(5): 9-25; 1978.

The similarity of adenomas of the parathyroid gland with other tumors of the APUD (amine precursor uptake and decarboxylation) system was investigated. The ultrastructural characteristics and the histology of 10 tumors were compared. Various histological variants of these tumors were shown to have a similar ultrastructure. The tumor tissue was made up of clear transitional cells and dark cells whose cytoplasm contained osmiophilic granules, filaments, laminar structures resembling Nissl bodies, fine granular and fibrous substances, and lipid inclusions. The adenomas were structurally similar to other endocrine-active tumors developing from cells of neuroectodermal origin (carcinoids, paragangliomas, etc). It is suggested that parathyroid gland adenomas belong to the APUD system. (21 refs)

78-4663 **Agglutinability, Cytoskeleton, and Surface Topography of Normal, Preneoplastic, and Neoplastic Mouse Mammary Cells In Vitro (Meeting Abstract).** (Eng) Asch, B. B. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX, 77030); Brower, M. E.; Brinkley, B. R. *Proc Am Assoc Cancer Res* 19: 67; 1978. (no refs)

78-4664 **Occurrence and Preneoplastic Significance of Ductal Dysplasia in the Normal Mammary Tissue of the Strain GR Mouse (Meeting Abstract).** (Eng) Miyamoto, M. J. (Cancer Res. Lab., Univ. Calif., Berkeley, CA, 94720); DeOme, K. B.; Osborn, R. C. *Proc Am Assoc Cancer Res* 19: 32; 1978. (no refs)

78-4665 **The Incidence of Mammary Tumours in a Group of 40 8-Year-Old Female Beagle Dogs Used in a Toxicity Study.** (Eng) Wadsworth, P. F. (Huntingdon Res. Centre, Huntingdon, Cambridgeshire, England); Heywood, R.; Prentice, D. E.; Van Abbe, N. J. *Toxicol Lett* 1(5/6): 285-289; 1978.

The incidence of mammary tumors was determined in 40 purebred, nulliparous, 8-yr-old beagle dogs (Appleton strain) that had been used in a 7.5-yr toxicity study of chloroform in toothpaste. Since the treatment did not increase the incidence of mammary tumors, data from treated and control dogs were combined. Periodic clinical examination during this time revealed four palpable mammary gland abnormalities. After macroscopic and histological evaluation, two benign mammary tumors, a pericanalicular fibroadenoma and a papillary cystadenoma, a papillary complex cyst, and a fibroma were identified. Histological examination of the mammary glands from all dogs indicated an intracanalicular fibroadenoma, a complex adenoma, and a tubular adenoma in addition to the four palpable tumors. The incidence of benign mammary tumors was thus 7.5%. Furthermore, 11 areas of nodular hyperplasia were found in 9 dogs, and foci of epitheliosis were found in 35 mammary glands in 12 dogs. It is concluded that (1) malignant mammary tumors are uncommon in these dogs, (2) benign mammary tumors are present in only a small percentage of these dogs, and (3) mammary gland dysplasia is very frequent in these dogs. (12 refs)

78-4666 **Lobular Neoplasia (So-called Lobular Carcinoma In Situ) of the Breast.** (Eng) Haagensen, C. D. (Columbia Univ. Coll. Physicians and Surgeons, 630 W. 168th St., New York, NY, 10032); Lane, N.; Lattes, R.; Bodian, C. *Cancer* 42(2): 737-769; 1978.

A total of 211 cases of lobular neoplasia of the breast (lobular proliferation without coexisting infiltrating carcinoma) were identified among 5,560 benign epithelial mammary lesions diagnosed in one laboratory between 1930 and 1972. Of the 211 patients, who followed for a mean time of 14 yr, 190 were premenopausal at the time of diagnosis and 21 were postmenopausal. After menopause, the lobular phase of the neoplasia was considerably diminished and the ductal phase sharply increased. The left breast was slightly more affected (57%) than the right (43%). The lobular neoplasia was associated with gross cysts (≥3 mm in diameter) in 153 patients and benign palpable tumors in another 42. Of 209 patients (1 lost to follow-up and another excluded because of carcinoma prior to the neoplasia), 35 subsequently developed breast carcinoma compared with 4.85 expected. The risk of subsequently developing carcinoma appeared to increase up to 25 yr after the original diagnosis. In patients who subsequently developed carcinoma, half of the carcinomas were entirely or partially small cell, and another nine were intraductal. Study of the factors that predispose to breast carcinoma (history of the disease in a mother or sister, gross cystic disease, and lobular neoplasia) in the 209 patients indicated that these three factors predispose to cancer development in a cumulative manner. Findings from previous studies on lobular neoplasia are reviewed, as is the histological basis for the classification of this neoplasm. (20 refs.)

78-4667 **Anovulation and Increased Androgenic Activity as Breast Cancer Risk in Women with Fibrocystic Disease of the Breast.** (Eng) Grattarola, R. (Hormone Res. Lab., Istituto Nazionale dei Tumori, Via Venezian 1, Milan, Italy). *Cancer Res* 38(9): 3051-3054; 1978.

The androgenic activity of 26 women with fibrocystic disease of the breast was investigated to determine whether this activity is a factor in the hormonal etiology of breast cancer. Urine samples (24-hr) were collected on days 20-22 of the menstrual cycle and assayed by gas chromatography for testosterone and for androstanediol (5α-androstane-3α,17β-diol), the major metabolite of dihydrotestosterone. The mean values for both androgens were significantly higher in these women than in 18 normal women in the same age range. Samples of endometrium taken on the same days indicate that only 9/26 women had normal progestational patterns 16 had a hyperplastic pattern and 1 showed a simple proliferative pattern. Since the endometrial specimens were obtaine in the premenstrual period, the presence of hyperplasi proved that the menstrual cycle in over two-thirds of th patients was nonovulatory. This study shows that patient with fibrocystic disease have the same hormonal status a breast cancer patients, ie, anovulatory menstrual cycle, increased androgenic activity, and endometrial hyperplasia. (3 refs.)

78-4668 **Malignancy of Human Lung Cancer Cells i Suppressed in Hybrids Formed with Mouse Fibroblast Cells (Meeting Abstract).** (Eng) Carney, D. N (Medical Oncology Branch [DCT, NCI, NIH], Vetera Admin. Hosp., Washington, DC, 20014); Edgell, C. J Gazdar, A. F.; Minna, J. D.; Chopra, H. *Proc Am Asso Cancer Res* 19: 117; 1978. (no refs)

See also:
*(Rev.): 78-4203, 78-4204, 78-4206, 78-4207, 78-420 78-4209, 78-4210, 78-4211, 78-4212, 78-421 78-4214.
*(Chem.): 78-4256, 78-4269, 78-4270, 78-427 78-4274, 78-4284, 78-4294, 78-430 78-4321, 78-4334, 78-4352.
*(Phys.): 78-4368, 78-4373, 78-4375, 78-438 78-4476.
*(Viral): 78-4410, 78-4421, 78-4436, 78-4441, 78-450 78-4512.
*(Immun.): 78-4542, 78-4545, 78-4552, 78-455 78-4560, 78-4569, 78-4570, 78-457 78-4595, 78-4596, 78-4619.
*(Epid.-Biom.): 78-4670, 78-4696, 78-4697, 78-470 78-4718, 78-4723, 78-4728, 78-472 78-4731, 78-4735, 78-4736, 78-474 78-4749, 78-4752, 78-4757.

78-4669 Epidemiology of C57BL/6M Mice: Actuarial Data for a Control Population. (Fre) Mewissen, J. D. (Laboratoire de Radiobiologie, Universite Libre de Bruxelles, 109, boulevard de Waterloo, 1000 Brussels, Belgium); Rust, J. H.; Haren, J.; Clutin, M. J. *C R Soc Biol (Paris)* 171(5): 1140-1144; 1977.

An actuarial analysis of the causes of death, tumor incidence, and tumor/host index in 503 male and 497 female control C57BL/6M mice is presented. The animals were kept under controlled, hygienic conditions. The overall spontaneous tumor incidence was found to be 66.7% in males and 84.4% in females over the max life-span of 1,300 days. The age-adjusted Gompertzian incidence rates were found to be nonlinear. For males, the tumor/host index had two maxima at 650-700 days (1.35) and 850-900 days (1.3). The Gompertzian regression curves showed three incidence peaks, the first between days 350 and 400 due to the relative max of lymphoid lymphosarcoma, the second between days 650 and 700 due to the relative peak of histiocytic reticulum-cell sarcoma, and a third between days 850 and 900 due to the relative max of histiocytic reticulum-cell sarcoma and reticulocytic reticulum-cell sarcoma. The tumor/host index had two peaks for the females, one between days 800 and 850 (1.62) and a second between days 950 and 1,000 (1.6). The Gompertzian regression curves showed two peaks in the respective age brackets of 300-350 days and 800-850 days. The first was due to the relative max of lymphoid lymphosarcoma, the second to reticulocytic reticulum-cell sarcoma. (5 refs)

78-4670 Neurofibromatosis and Childhood Leukemia. (Eng) Bader, J. L. (Clinical Epidemiology Branch, NCI, A-521, Landow Building, Bethesda, MD); Miller, R. W. *J Pediatr* 92(6): 925-929; 1978.

Twelve new cases of childhood leukemia and neurofibromatosis (NF) were evaluated in conjunction with 17 previously documented cases. Among all 29 patients, 9 had acute ymphocytic leukemia (ALL), 8 had acute myelomonocytic eukemia (AMML), 8 had chronic myelogenous leukemia, 3 had acute myelogenous leukemia, and 1 had acute nonlymphocytic leukemia, type unspecified. There were 18 boys and 11 girls, all Caucasian, and 2 AMML patients were siblings. No consistent cytogenetic abnormalities were found in eight patients studied. NF was present in 70% of the patients, compared with the usual 50% observed in NF patients. All pa- ients with leukemia diagnosed at <2 yr of age had familial NF. Familial NF was present in 83% of patients with non- ymphocytic leukemia and in only 44% of patients with ALL. The ratio of ALL:nonlymphocytic leukemia among patients with sporadic NF was 5:3, compared with the 4:1 ratio observed for childhood leukemia in general. The ratio was 4:15 among patients with familial NF. For all cases combined, the ratio was 9:20. Two possible variants of the NF-leukemia association were NF with 'transient leukemia' and nonlymphocytic leukemia with multiple skin xanthomas and normal cholesterol levels. Abnormal radiologic findings occasionally associated with therapy in childhood leukemia were observed unusually early in one patient, possibly because of the presence of NF. The addition of NF to the array of genetic diseases and environmental factors associated with leukemia may help to explain the genesis of the neoplasm. (28 refs.)

78-4671 Occupational Exposure to Petroleum Products in Men with Acute Non-lymphocytic Leukaemia. (Eng) Brandt, L. (Dept. Internal Medicine, Univ. Hosp., S-221 85 Lund, Sweden); Nilsson, P. G.; Mitelman, F. *Br Med J* 1(6112): 553; 1978.

Occupational exposure to petroleum products was investigated in 50 men (aged 20-65 yr) with acute nonlymphocytic leukemia (ANLL) and in three groups of controls: 100 men (aged 20-65 yr) treated for nonmalignant disorders; 100 men (aged 20-65 yr) treated for allergic diseases; and 22 men (aged 20-62 yr) with chronic myeloid leukemia (CML) and 10 men (aged 41-63 yr) with chronic lymphocytic leukemia (CLL). Eighteen of 50 ANLL patients were occupationally exposed to petroleum products or their combustion residues. In each of the first two control groups, 10/100 patients were so exposed; 3/22 CML patients and none of the CLL patients were occupationally exposed to petroleum products. It is concluded that occupational exposure to motor fuel is unduly common in ANLL patients and that some leukemogenic factor may be present. (5 refs)

78-4672 Some Clues to Human Leukemia Expression from Comparison of Age-Standardized Incidence Rate Data in Man, Cat and Dog. (Eng) Schneider, R. (Animal Registry, Dept. Epidemiology and Preventive Medicine, Univ. California, Davis, CA, 95616). *In: Advances in Comparative Leukemia Research 1977. Proceedings of the VIIIth International Symposium on Comparative Research on Leukemia and Related Diseases, held in Amsterdam, The Netherlands on 22-26 August, 1977.* Bentvelzen, P.; Hilgers, J.; Yohn, D. S., eds. (New York: Elsevier/North Holland Biomedical Press): 503 pp.; 37-40; 1978.

The incidence rate patterns of leukemias-lymphomas in cats, dogs, and humans were compared with the use of population-based animals and human tumor registries functioning in the same area of California. Incidence rates for dogs and cats were calculated using cases diagnosed from July 1967 to June 1974, and population estimates were obtained by survey for the middle of the case-collecting period. Incidence rates for humans were calculated using the leukemia and lymphoma cases reported in the San Francisco-Oakland Standard Metropolitan Statistical Area during 1969-1971. The leukemia female/male ratios were 0.6 for humans and cats and 1.1 for dogs, and the male/female ratios for all other cancers were 0.9 for humans, 1.0 for cats, and 1.3 for dogs. The dog did not show a consistent pattern of changing leukemia male/female ratios according to age, whereas the sex ratio changed with age according to a similar pattern in humans and cats. Both humans and cats showed a bimodal age-specific leukemia incidence rate that was not seen in dogs. The early peak for Hodgkin's disease in humans was at 20-29 yr and the early peak for leukemia in cats was at an equivalent age. The incidence of leukemia in dogs increased with increasing size of the dog, from about 10-15/100,000 for breeds < 10 kg to about 150/100,000 for the Great Dane and St. Bernard. Rates of all other cancers combined also increased with size, but to a lesser extent (one-half). This may be due to differences in rate of growth among breeds. The data indicate that of the three species, the dog may be the most efficient in protecting itself against these diseases and the cat the least efficient. (6 refs.)

78-4673 **The Effect of Neonatal BCG Vaccination on the Incidence of Leukemia.** (Ger) Ambrosch, F. (Institut fur Spezifische Prophylaxe und Tropenmedizin, Universitat Wien, Kinderspitalgasse 15, A-1095 Vienna, Austria); Krepler, P.; Wiedermann, G. *Munch Med Wochenschr* 120(8): 243-246; 1978.

Data from a literature review as well as current investigations were analyzed with respect to the possible nonspecific influence of BCG vaccinations on leukemia. Data from the five largest provinces of Austria indicate an inverse correlation between leukemia mortality and neonatal BCG vaccination. A protection rate of 0.88 for the prevention of leukemia deaths was calculated for the age group 0-5 yr; this corresponded to the literature protection rate of 0.85. Further analysis of the data indicated a deficit in positive tuberculin reactions at the onset of the disease in BCG-vaccinated children. A mathematical model was designed in order to calculate the frequency of positive tuberculin reactions in a population of 16 BCG-vaccinated leukemic children (ages 1-5 yr). There were only five positive tuberculin reactions, a finding that indicated a hypothetical influence of BCG vaccination on leukemia. (12 refs.)

78-4674 **Epidemiologic and Histologic Patterns of Hodgkin's Disease in Blacks.** (Eng) Vianna, N. J. (Cancer Control Bureau, New York State Dept. Health, Empire State Plaza, Tower Building, Room 523, Albany, NY 12237); Thind, I. S.; Louria, D. B.; Polan, A.; Kirmss, V.; Davies, J. N. *Cancer* 40(6): 3133-3139; 1977.

The epidemiologic, social, and histologic patterns of Hodgkin's disease (HD) in blacks were examined in Newark, New Jersey, and New York State (excluding New York City). The number of HD deaths among the Newark blacks was significantly smaller than expected for the young adult and older age groups. The number of deaths in the childhood age group was too small for statistical evaluation. In New York, a similar pattern was observed for the adult age groups, but the number of cases in children < 14 yr was greater than expected. The relative risk ratios of blacks to whites was 0.2 in Newark and 0.6 in New York. There was a general male predominance in both races, especially in blacks. A significantly greater number of black patients belonged to low-income groups. A comparison of these findings with those for blacks in Southern Transvaal, South Africa, and other countries suggests that the natural history of HD might be strongly influenced by social milieu. Age-related environmental factors might be important in the histologic reactivity of the host. (15 refs.)

78-4675 **Incidence of Hematological Neoplasms among Physicians.** (Eng) Gil-Extremera, B. (Dept. Developmental Therapeutics, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX, 77025); Rodriguez, V.; Bodey, G. P. *Rev Esp Oncol* 24(1): 207-213; 1977.

The incidence of hematologic malignancies (acute leukemia, lymphoma, Hodgkin's disease, and multiple myeloma) among physicians was reviewed in a retrospective study covering 1969-1974. During this period, 435 physicians died of hematologic malignancies, an incidence of 2%, which was constant over the 6-yr period. No significant difference was detected in the distribution of deaths due to leukemia, lymphoma, Hodgkin's disease, or myeloma among the various specialty groups. Comparison of the mortality incidence of the physicans with that of the general population indicated that there was a higher incidence of death from acute leukemia among the physicans than among the general population. The difference in the number of deaths due to other hematological malignancies among physicans and the general population was not significant during 1969 and 1970. However, an increased number of deaths due to these diseases was observed among physicians in 1971, 1972, and 1974. Prospective studies should be undertaken to determine whether populations of physicians, such as oncologists, who are frequently exposed to these diseases are especially likely to contract them. (9 refs)

78-4676 **A Cluster of Hodgkin's Disease in a Small Community. Evidence for Environmental Factors.**

(Eng) Schwartz, R. S. (Dept. Internal Medicine, Univ. Michigan Medical Center, Ann Arbor, MI, 48104); Callen, J. P.; Silva, J. *Am J Epidemiol* 108(1): 19-26; 1978.

A cluster of Hodgkin's disease (HD) cases occurred in a small rural central Michigan town of 1,250 people. Ten cases of HD and three cases of non-Hodgkin's lymphoma were identified within or linked to this town since 1954. Therefore, this town had an av annual incidence and mortality for HD of 29.3 and 16.7 cases per 100,00 population respectively. Most HD cases demonstrated case-contact associations and a distinct geographic distribution. The data suggest that an environmental agent was responsible for the elevated rates of HD. One unique aspect of this cluster is that the town has only one industry, a large grain elevator. The cases closely surrounded this elevator. It is postulated that the residents are subject to chronic immune stimulation from mitogenic substances in the local environment. These agents may alter the immunity in the residents and predispose them to HD. (40 refs.)

78-4677 **Pesticides and Lymphoma in Iowa.** (Eng) DeKraay, W. H. (Ottumwa, IA). *J Iowa Med Soc* 68(2): 50-53; 1978.

Iowa counties with higher than expected lymphoma incidences were clustered along the Des Moines and Nishnabotna Rivers. Water samples from these and other rivers indicated seasonal dieldrin concentrations as high as 65 parts per trillion. The lowest dieldrin concentrations were found in the drinking water of cities that obtained water from wells, and these cities did not have high lymphoma rates. Dieldrin levels in catfish (toxic level established at 300 ppb) ranged as high as 1,600 ppb in some rivers, but no attempt was made to determine catfish consumption by lymphoma patients. A study of 110 lymphoma patients in one county revealed that most were 60-80 yr old. Approx 34% had Hodgkin's, 66% non-Hodgkin's lymphoma. The age pattern of Hodgkin's disease did not follow the bimodal curve expected in the third and sixth decades. A 5-yr survival rate of 29% and a 5-yr recurrence-free rate of 15% were observed. With the banning of dieldrin in 1974, a follow-up study of lymphoma incidence will be of interest. (6 refs.)

78-4678 **Pre- and Post-treatment Second Malignant Neoplasms (SMN) in Hodgkin's Disease (HD) (Meeting Abstract).** (Eng) Ayoub, J. I. (Hopital Hotel-Dieu de Montreal, Montreal, Quebec, Canada H2W IT8); Dubois, A.; Cosendai, A. M.; Tetreault, L.; Pretty, H. *Proc Am Assoc Cancer Res* 19: 322; 1978. (no refs)

78-4679 **Growth Rates of Small Cell Bronchogenic Carcinoma (SCBC) (Meeting Abstract).** (Eng) Brigham, B. (Div. Cancer Treatment, NCI-VA Medical Oncology Branch, Washington, DC, 20422); Bunn, P. A.; Minna, J. D.; Cohen, M. H.; Ihde, D. C.; Shackney, S. E. *Proc Am Assoc Cancer Res* 19: 357; 1978. (1 ref)

78-4680 **Environmental Contaminants of Foods for Infants (Meeting Abstract).** (Eng) Gladen, B. (Biometry Branch, NIEHS, Research Triangle Park, NC, 27709); Rogan, W. *Environ Health Perspect* 20: 248; 1977. (no refs)

78-4681 **Risk Factors of Breast Cancer in Burma.** (Eng) Hlaing, T. (Epidemiology Div., Dept. Medical Res., Ministry Health, Zafar Shar Road, Dagon P.O., Rangoon, Burma); Myint, T. M. *Int J Cancer* 21(4): 432-437; 1978.

A hospital-based case-control study of breast cancer was conducted in Rangoon, Burma. One hundred ninety-three breast cancer cases and 403 controls were interviewed. The median age of the breast cancer patients was 52.1 yr, and that of the controls was 50.4 yr. The risk of breast cancer was significantly increased among more educated women ($p = 0.0003$) and among women who had their menopause after the age of 47 ($p = 0.00003$). It was also increased in women who had their menarche prior to age 15. There was no association between risk and lactation. A strong inverse relation was found between risk and parity: women who had six or more children had only one-third the risk of cancer of married women who had less than four children. Risk was increased in women aged >30 yr at first birth. The age-standardized incidence of breast cancer in Rangoon, 25.1/100,000 women-years, is higher than that of other Asian populations and lower than that in North America and northern Europe. The predomination of parity rather than age at first birth as a risk factor in Rangoon is unique and should be explored further. (18 refs)

78-4682 **Cell Kinetics of Large Cell Lung Tumors and Protocol Design (Meeting Abstract).** (Eng) Jacob, H. E. (Allegheny General Hosp., Pittsburgh, PA, 15212); Braunschweiger, P. C.; Stragand, J. J.; Novak, J.; Schiffer, L. M. *Proc Am Assoc Cancer Res* 19: 397; 1978. (no refs)

78-4683 **The Prevalence of Sjogren's Syndrome in Non-Hodgkin's Lymphoma. (Meeting Abstract).** (Eng) Pritchard, K. I. (Ontario Cancer Inst. M4X 1K9, Univ. Toronto, Toronto, Ontario, Canada); M'Seffar, A. M.; Weinstein, A.; Katz, A.; Gordon, D. A.; Baker, M. A. *Proc Am Assoc Cancer Res* 19: 418; 1978. (no refs)

78-4684 **Ethnic-related Incidence of Ovarian Carcinoma in New York City.** (Eng) Soichet, S. (New York Hosp.-Cornell Medical Center, New York, NY). *Isr J Med Sci* 14(3): 363-369; 1978.

A two-part statistical study of ovarian cancer incidence in New York City was conducted in an effort to explain the striking racial differences in mortality from this disease. In the first part, an examination of 874 death certificates from case reports for 1964 (487) and 1974 (387) revealed (1) a lower incidence of ovarian cancer among nonwhite than among white women and (2) a higher incidence among Jewish than among non-Jewish women. For Jewish women, the mortality from ovarian cancer was similar in 1964 and 1974. The second part entailed a review of 143 records from New York City hospitals from which more precise data, such as history and treatment, were available. Clinical features related to the different ethnic and racial incidences were analyzed, but no etiologic factors could be correlated with the disease. (23 refs)

78-4685 **Cohort Mortality for Prostatic Cancer among United States Nonwhites.** (Eng) Ernster, V. L. (Dept. Biomedical and Environmental Health Sciences, Univ. California, Berkeley, CA, 94720); Selvin, S.; Winkelstein, W. *Science* 200(4346): 1165-1166; 1978.

To determine if a cohort or a generational effect is wholly or partially responsible for the increase in prostatic cancer mortality among blacks, prostatic cancer mortality rates among men aged 45-84 yr were analyzed for separate birth cohorts of US nonwhites extending from 1846 to 1925. Since blacks make up the majority of nonwhites, only small effects could be due to other nonwhites. Rates for all ages peaked in the 1896-1900 in the cohort and declined thereafter, at least for men under the age of 70 for whom data were available. This presages an arrest and reversal of the time trend in summary mortality rates as more recent nonwhite cohorts reach the ages of max risk. Research into the historical experience of the 1896-1900 and earlier nonwhite birth cohorts may provide clues to agents associated with prostatic cancer. (7 refs)

78-4686 **Association of Colo-Rectal and Prostatic Cancer (Meeting Abstract).** (Eng) Paladine, W. J. (Oncology Section, Albany Veterans Admin. Hosp., Albany, NY, 12208); Chalmers, P. *Proc Am Assoc Cancer Res* 19: 340; 1978. (1 ref)

78-4687 **Fecal Microbial Flora in Seventh Day Adventist Populations and Control Subjects.** (Eng) Finegold, S. M. (Infectious Disease Section, Wadsworth Hosp. Center, Veterans Admin., Los Angeles, CA 90073); Sutter V. L.; Sugihara, P. T.; Elder, H. A.; Lehmann, S. M.; Phillips R. L. *Am J Clin Nutr* 30(11): 1781-1792; 1977.

The fecal microbial flora content of vegetarian Seventh Da Adventists was investigated and compared to nonvegetaria Adventists, non-Adventists on a conventional American diet Japanese-Americans on either a Western or Japanese diet and Caucasians on a conventional American diet. Ther was little difference among the Adventist groups. Group at high risk for colon cancer (Japanese-Americans o Western diet and Caucasians on conventional Americar diet) had significant differences as compared to lo risk populations (Japanese-Americans on Japanese die and Adventists). (22 refs.)

78-4688 **Further Biologic Characteristics of a Huma Carcinoembryonic Antigen-producing Colon Carcinoma Cell Line.** (Eng) Drewinko, B. (Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., 6723 Bertner Ave., Houston, TX, 77030); Yang, L. Y.; Barlogie, B.; Romsdahl, M.; Meistrich, M.; Malahy, M. A.; Giovanella, B. *J Natl Cancer Inst* 61(1): 75-83; 1978.

Human carcinoembryonic antigen (CEA)-producing colon adenocarcinoma cells (LoVo cells) grown in vitro were characterized in terms of several kinetic and biological parameters. Doubling times of exponentially growing cells ranged from 34 to 38 hr for initial cell concentrations of 10^5 to 7 x 10^5/petri dish. The generation time was 29.3 hr. Cultures exposed continuously to tritiated thymidine had a growth fraction of 90%. Compartment distribution calculated in reference to cell cycle transit times measured by pulse-labeled mitosis techniques was similar to that defined directly by pulse cytophotometry (PCP). PCP analysis of cells in the stationary growth phase revealed a significant fraction of cells with S-phase DNA content (15%), even though the simultaneously defined labeling index was only 1%. Adequate synchronization in the S- and G_2-phases was achieved by treating the cells with 7 mM thymidine for 24 hr. Centrifugal elutriation and mitotic selection were inferior in terms of reproducibility and cell yield. Colony formation was most efficient for cells plated in fresh medium and incubated for 2 days. The tumorigenic potential of LoVo cells wa established by their transplantation into nude mice Inoculated cells grew relatively slowly, and visibl tumor nodules appeared after 3 wk. The transplante cells retained the morphologic features of the origina tumor. Sera from three LoVo cell-bearing mice assayed for CEA when the animals were killed contained 31.3, 40.0, and 87.5 nanograms of CEA/mg, but no CEA was detected in control mice. The LoVo line may be a unique candidate for an in vitro model of colon carcinoma. (37 refs)

78-4689 **An Investigation of the Use of Asbestos Cemen Pipe for Public Water Supply and the Incidenc**

of Gastrointestinal Cancer in Connecticut, 1935-1973. (Eng) Harrington, J. M. (Environmental Hazards Activity, Chronic Diseases Div., Bureau Epidemiology, Center Disease Control, Atlanta, GA 30333); Craun, G. F.; Meigs, J. W.; Landrigan, P. J.; Flannery, J. T.; Woodhull, R. S. *Am J Epidemiol* 107(2): 96-103; 1978.

Determination was made of the effects of the use of asbestos cement pipe for public water supply on the incidence of stomach, colon, and rectal cancer in Connecticut between 1935 and 1973. Tumor incidence per township was obtained from the Connecticut Tumor Registry. For all towns, the date(s) of installation of the asbestos cement pipe, the length of this type of pipe compared to other types, and the population serviced by the pipe were determined. These factors were incorporated into two composite indices: the Assessment of Exposure and Risk Factors. Observed to expected (O/E) ratios of all three types of cancer tended to increase as the population density increased. Furthermore, O/E ratios for colon and rectal cancer were opposite those for stomach cancer. However, there was no pattern of association between cancer incidence and the use of asbestos cement pipe. (26 refs)

78-4690 **An Occupational Group with a High Risk of Large Bowel Cancer.** (Eng) Vobecky, J. (Faculte de Medicine, Universite de Sherbrooke, Sherbrooke, Quebec, Canada J1H 5N4); Devroede, G.; Lacaille, J.; Watier, A. *Gastroenterology* 75(2): 221-223; 1978.

Over a 1.5-yr period, five individuals employed in the same unit of a carpet factory that manufactured synthetic threads developed large bowel cancer. Workers stated that there was a strong odor in this unit while a powdery ingredient was being liquefied to produce the material. The tumors were of similar histological type, the patients were 31-58 yr, and there was no family history of cancer. Duration of exposure ranged from 10 to 32 yr. A detailed study of the occurrence of colorectal cancer in the area from 1965 to 1975 was undertaken to determine if this outbreak of cancer was a chance occurrence or the result of occupational exposure. Review of 1,000 medical records of patients with colorectal cancer revealed that 30 patients had been employed in the carpet factory. Further analysis was restricted to 12 men whose cancers occurred from 1971 to 1975 and who were working in the factory at the time of diagnosis. Most cancers were in the left colon, and the most frequent histopathological type was adenocarcinoma. The observed age- and sex-adjusted relative risk ratio was 10 times higher among workers in the carpet factory than among workers in other occupations. The morbidity in the studied group was 11.4 times higher than the expected one. (18 refs.)

78-4691 **Fecal Constituents of a High-Risk North American and a Low-Risk Finnish Population for the Development of Large Bowel Cancer.** (Eng) Reddy, B. S. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY, 10595); Hedges, A.; Laakso, K.; Wynder, E. L. *Cancer Lett* 4(4): 217-222; 1978.

Feces were collected from age-matched, healthy, middle-aged male volunteers in Kuopio, Finland (a low-risk population for the development of colon cancer), and the New York metropolitan area (a high risk population for colon cancer), and their constituents were compared. The av daily intake of fat and protein was essentially the same in the two populations, but the intake of carbohydrate was higher in Kuopio. The main sources of the fat were from meat in New York, from dairy products in Kuopio. The latter group also had a higher intake of fish, whole-rye bread, and whole-grain cereals. The number of stools passed per day was higher in Kuopio; the percentage of dry matter was lower, but the daily stool output was three times higher. Fecal bacterial β-glucuronidase activity per gram of dry feces was lower in Kuopio, but there was no difference in the daily output of enzyme activity between the two groups. A significantly lower concentration of secondary bile acids was found in Kuopio, but the total daily output of these bile acids was the same in the two groups. There was no difference in the concentration of neutral sterols, but the daily excretion of neutral sterols was higher in Kuopio. These findings suggest that the greater fecal bulk in Kuopio may dilute tumorigenic compounds, and tumor promotors (in the bile acids) that come in direct contact with the colonic mucosa. (26 refs)

78-4692 **Colo-rectal Carcinoma in Young Adults.** (Eng) Koyama, Y. (Dept. Surgery, Natl. Cancer Center Hosp., 5-1-1, Tsukiji, Chuo-ku, Tokyo 104, Japan); Hojo, K.; Hirota, T.; Kodaira, S. *Jpn J Clin Oncol* 8(1): 21-26; 1978.

Twenty-five patients <29 yr old (Group 1) and 693 patients >30 yr old (Group 2), all with carcinoma of the large intestine, were compared to determine if there are any factors connected with the etiology and prognosis in young patients. Localized lesions were found in 9 Group 1 patients, and 6 had distant metastases at the time of surgery; 19 underwent curative resection. Study of the crude cumulative survival rate indicated that only 37% of the Group 1 patients survived 5 yr, compared with 56% of the Group 2 patients. Histologically, the mucinous type of tumor, especially signet ring cell carcinoma was observed more frequently in Group 1 than in Group 2. Cancer cells permeating into the lymph vessels were found in most of the Group 1 patients. A comparison of the frequencies of coincidental cancerous and/or precancerous lesions at the time of surgery indicated that synchronous multiple cancers were present in 3/23 young patients treated by resection compared with 22/677 older patients; this difference was statistically significant. The incidence of familial polyposis coli, which accompanied invasive carcinoma, was also significantly higher in the younger patients. The cumulative no evidence of disease rate after curative surgery was lowest among Group 1 patients; it increased stepwise with each successive decade. (20 refs.)

78-4693 **Topographic Study of Glycoconjugates in Proximal and Distal Rat Colonic Mucosa (Meeting Abstract).** (Eng) Freeman, H. J. (Veterans Admin. Hosp., Univ. California, San Francisco, CA); Kim, Y. S.; Lotan, R. *Clin Res* 26(3): 319A; 1978. (no refs)

78-4694 **Spectrum of Fecal Bacterial Flora in Colon and Non-Gastrointestinal Cancer Subjects (Meeting Abstract).** (Eng) Floch, M. H. (Norwalk Hosp., Norwalk, CT); Moskovitz, M.; Vargo, D.; Ramos, R.; May, S.; Fuchs, H. M. *Clin Res* 26(3): 318A; 1978. (no refs)

78-4695 **Statistical Studies in the Aetiology of Malignant Neoplasms. V. Trends and Risks. Denmark 1943-72.** (Eng) Clemmesen, J. (Cancerregisteret, Finsen Inst., Strandboulevard 49, Copenhagen 2100, Denmark). *Acta Pathol Microbiol Scand [A]* (Suppl. 261): 1-286; 1977.

Statistical studies on malignant neoplasms in humans are compiled using data obtained from the Danish Cancer Registry. In Denmark, there was a gradual increase in cervical carcoma from 1943 to 1967, followed by a decrease from 1968 to 1972. In Connecticut, however, a steady decrease in incidence was noted. In both areas, an increase in the number of in situ cancers was accompanied by a decrease in invasive cancers. Endometrial carcinomas in Denmark have increased with time. Clinical data on 8,078 patients treated with phenobarbital and followed until their death failed to reveal any carcinogenic effects. Data from air pollution studies indicate that pollution may play an etiological role in lung cancer, but the absence of data on local smoking habits makes any definite conclusions difficult. Gastric cancer incidence decreased from 1943 to 1972, although some of this decrease could be attributed to more effective diagnostic assessment of a previously overrated diagnosis. A study of the survival of breast cancer patients revealed that the 7-yr survival for women $<$ 45 yr old improved between 1949 and 1954; the improvement was less marked in women $>$ 45 yr old. However, morbidity changed little between 1950 and 1970 for women $<$ 45 yr old, and it increased for women $>$ 45 yr old. Tables of morbidity rates for various malignant neoplasms for the years 1943-1972 are also presented. (50 refs)

78-4696 **Incidence of Endometrial Carcinoma in King County, Washington: A Standardized Histologic Review: Brief Communication.** (Eng) Szekely, D. R. (Dept. Epidemiology, SC-36, Sch. Public Health and Community Medicine, Univ. Washington, Seattle, WA, 98195); Weiss, N. S.; Schweid, A. I. *J Natl Cancer Inst* 60(5): 985-989; 1978.

A total of 150 endometrial cancer specimens from women aged 35-74 yr who were residents of King County, Washington, during the first 6 mo of 1975 were reviewed by on
pathologist who used uniform criteria to assess the cance
The routinely reported incidence of this tumor was judge
to be inflated, falling from 108.2 to 88.5/100,000 women afte
the exclusion of cases found not to be unequivocally mali
nant. Nonetheless, this rate represented a large increase ove
the rate of 47.3/100,000 observed in the US Third Nationa
Cancer Survey 5 yr previously. The proportion of wome
who used noncontraceptive estrogen preparations for a mini
mum of 2 yr varied between 70% and 81% for any grade c
cancer. More women with cancer confined to the endometri
um were estrogen users than were women with cancer pen
trating into the myometrium. (14 refs)

78-4697 **Epidemiology and Pathophysiology of Corp**
Carcinoma. (Eng) Kjellgren, O. (Dep
Gynaecological Oncology, Univ. Umea, S-901 85 Ume
Sweden). *Acta Obstet Gynecol Scand [Suppl]* (65
77-82; 1977.

The incidence of endometrial carcinoma in Sweden is re
viewed. Between 700 and 800 new cases are being reporte
annually for an incidence of approx 19/100,000; th
death rate from endometrial carcinoma accounts fo
0.5% of all female deaths. Most cases occur between th
ages of 50 and 70 yr. Many of these patients are ove
weight, and comparison with a control population i
dicated that the former are approx 13% heavier tha
the latter. A person who is 21 to 50 pounds overweig
between the ages of 25 and 29 has a risk factor c
approx 3; being 50 pounds overweight carries a ris
factor of 10. Late menopause, nulliparity, uterine f
broids, and diabetes are frequent in endometrial carcinom
patients. A review of the experimental evidence linking estro
gens with cancer indicated that cancer can only be induce
in animal strains with a high spontaneous rate of cancer an
under certain circumstances. Although endometrial cance
can be induced in about 8% of the patients with gonada
dysgenesis, this induction results from diethylstilbestrol an
not steroids. The literature contains controversial results:
is not known whether changes in the uterus following estro
gen treatment are really cancerous or just a hyperstimulatio
of the endometrium. (22 refs.)

78-4698 **Relationship Between Epidemiologic and Endo**
crinologic Aspects of Cervical Cancer. (Eng
Kodama, M. (Lab. Chemotherapy, Aichi Cancer Center Re
Inst., Chikusa-ku, Nagoya, Japan); Kodama, T.; Totani, R
Aoki, K. *J Natl Cancer Inst* 61(1): 35-39; 1978.

Epidemiologic and endocrinologic data were used to dete
mine the etiologic relevancy of steroid deviations in the urin
of cervical cancer patients. It was found that th
premenopausal cervical cancer patient had her first child a
a significantly earlier age than did an age-matched urban con

rol, despite their similar ages at menarche. However, when he same patient was compared with a premenopausal rural ontrol, this difference was not found. The same parameter lso failed to differentiate a postmenopausal patient from a orresponding urban control. The premenopausal patient vho was indistinguishable from the rural control by age at irst delivery was, however, distinguished from the rural conrol by reduced excretions of adrenal steroids, including 1-deoxy-17-ketosteroids and some corticosteroid metaboites. The spectrum and degree of deviation of urinary steoids for the cervical cancer patient coincided with the specrum and degree of age dependency of the urinary steroids or a normal woman during and after adolescence. These indings indicate that the inability of the reproductive parameter to discriminate a cervical cancer patient rom a rural control was associated with growth retardaion in a population and that a maturation deficiency of the adrenal gland (arrest of adrenarche) is implicated n the genesis of cervical cancer. (10 refs)

8-4699 **Carcinoma of the Female Breast in Jamaica.** (Eng) Harris, M. (Dept. Pathology, Univ. Manchester, Manchester M13 9PT, England). *Trop Geogr Med* 29(3): 213-222; 1977.

The incidence of breast cancer in Jamaican women is intermediate between the low levels of African countries and the high levels of the US, Great Britain, and other Western industrial societies. The annual incidence in Jamaica has shown an upward trend from 1958 to 1974. Compared with Uganda, breast cancer in Jamaica conforms to a westernized pattern in terms of the age-specific incidence curves and tumor stage at presentation. However, there are no marked histologic differences among the breast cancers in Uganda, Jamaica, and the U.S. The Jamaican incidence is higher than expected in view of the fact that early first pregnancies (purported to have a protective effect) are common in this country. (20 refs.)

8-4700 **Barrier Contraceptive Practice and Male Infertility as Related Factors to Breast Cancer in Married Women.** (Eng) Gjorgov, A. N. (Dept. Res. Medicine, Dept. Obstetrics and Gynecology, Univ. Pennsylvania Sch. Medicine, Philadelphia, PA, 19104). *Med Hypotheses* 4(2): 79-88; 1978.

A case-control study of 153 consecutive mastectomy cases of married or never-married white women, 35-60 yr of age at diagnosis, and 168 controls was conducted to test the hypothesis that a reduced exposure to human seminal factors in early reproductive life is a risk factor in breast cancer. It was found that 71.2% of the breast cancer patients used barrier methods of contraception (condom, withdrawal, abstinence, celibacy) compared with 34.5% of the controls. Preliminary results indicate that the relative risk of exposure to the hypothetical semen-factor deficiency is 4.69 times greater for breast cancer patients than for controls, which suggests that barrier contraception and male infertility may play more than a chance role in this disease in married women. When contraceptive methods alone are taken into consideration, the relative risk is increased up to 5.3 times. These data also indicate that 16.1% of the women using barrier contraception would develop breast cancer compared with 3.4% of the women using nonbarrier techniques. Elimination of the barrier method of contraception would result in a >50% reduction in breast cancer incidence among married women. (36 refs)

78-4701 **Results of Case-Control Study of Breast Cancer.** (Eng) Krajinovic, S. (Inst. Epidemiology, Medical Faculty, Belgrade, Yugoslavia); Udicki, S.; Radovanovic, Z.; Merkas, Z.; Jankovic, S.; Miljkovic, V. *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 187-190; 1977.

Results of an interview of 447 women with breast cancer from hospitals in Belgrade, Hungary, and 447 age-matched controls revealed that the most significant difference between the two groups was the time between successive periods: the interval was much shorter in the cancer patients. In addition, the women with cancer were more often nulliparous, had later deliveries, fewer children, shorter duration of lactation, and a shorter duration of menstruation. (7 refs.)

78-4702 **The Possible Role of Hormones, Marital Status and Gestation in Breast Cancer.** (Eng) Juhasz, L. (Cancer Registry, Szabolcs-Szatmar County, Nyiregyhaza, Hungary). *In: Characterization and Treatment of Human Tumours. Proceedings of the Seventh International Symposium on the Biological Characterization of Human Tumours held in Budapest, April 13-15, 1977.* Davis, W.; Harrap, K. R., eds. (Amsterdam: Excerpta Medica): Advances in Tumour Prevention, Detection and Characterization. Vol. 4, 332 pp.; 191-193; 1978.

A retrospective analysis of data on 505 patients with breast cancer and 866 controls from two counties in East Hungary indicated that marriage, longer duration of marriage, greater number of deliveries, younger age at first birth, longer duration of lactation, and earlier menopause may be associated with a decreased risk of breast cancer. (2 refs.)

78-4703 **Breast Cancer in a Psychiatric Hospital Before and After the Introduction of Neuroleptic Agents (Meeting Abstract).** (Eng) Wagner, S. (Norristown State Hosp., Norristown, PA, 19401); Mantel, N. *Proc Am Assoc Cancer Res* 19: 180; 1978. (no refs)

78-4704 **Familial Breast Cancer: Risk to the Contralateral Breast.** (Eng) Harris, R. E. (Dept. Preventive Medicine and Public Health, Sch. Medicine, Creighton Univ., Omaha, NB, 68178); Lynch, H. T.; Guirgis, H. A. *J Natl Cancer Inst* 60(5): 955-960; 1978.

The pathology reports of 198 women with familial breast cancer were reviewed to determine the chronologic features of tumorigenesis in the breast contralateral to the site of the initial mastectomy. The actuarial data revealed two major risk periods with an intervening gap in which no malignant tumors of the contralateral breast were detected. The cumulative cancer risk to the contralateral breast increased annually at 2.8% in the first 6 postoperative yr, showed no increase in the next 7 yr, and finally increased at an annual rate of 6.8% between years 13 and 16. The corresponding annual rates of increase for second primary breast cancer in the two risk periods were 1.6% and 5.9%, respectively. A total of 37 patients manifested bilateral breast cancer, of whom 21 had pathologically confirmed second primary carcinomas. Of these patients, 27 exhibited a relatively short interval between mastectomies (1.7 yr), but 10 had a much longer disease-free interval (16.5 yr). It is suggested that tumors manifested in the contralateral breast within 6 yr of the initial breast cancer were already developing but were not clinically discernible at the time of the first mastectomy. The extended disease-free interval in many patients suggests that the host defense mechanisms may have been stimulated by the first tumor, which in turn had a persisting suppressive effect on de novo carcinogenesis in the contralateral breast. These findings indicate the need for vigorous cancer surveillance of long-term survivors of an initial breast cancer, particularly in familial patients. (12 refs)

78-4705 **Racial Trends in Urban Breast Cancer Incidence and Cancer Control (Meeting Abstract).** (Eng) Brennan, M. J. (Michigan Cancer Foundation, Detroit, MI, 48201); Wilt, G. M.; Albert, S. *Proc Am Assoc Cancer Res* 19: 391; 1978. (no refs)

78-4706 **Reproductive History and Risk of Breast Cancer. A Case-Control Study in an Unselected Swedish Population.** (Eng) Adami, H. O. (Dept. Surgery, Univ. Hosp., S-750 14 Uppsala, Sweden); Rimsten, A.; Stenkvist, B.; Vegelius, J. *Cancer* 41(2): 747-757; 1978.

The reproductive history of 179 women with breast cance and 179 age-matched controls was compared in an attemp to link this history with breast cancer risk. There was no significant difference between the two groups with respect to age at menarche, age at first birth, age at menopause, or number of children. Selection of the controls from the genera population and not from hospital patients could account for the differences between these findings and previous reports (46 refs)

78-4707 **Characteristic Species Dependent Growth Patterns of Mammalian Neoplasms.** (Eng) Brunton, G. F. (Dept. Clinical Physics and Bio-Engineering, West of Scotland Health Boards, 9-11 W. Graham St., Glasgow G4 9LF, Scotland); Wheldon, T. E. *Cell Tissue Kinet* 11(2) 161-175; 1978.

Data on tumor growth in mice, rats, and hamsters and on the growth of human multiple myeloma support the idea of a species-specific relation between two supposedly independent parameters in the Gompertz equations frequently used to quantify tumor growth curves. These findings support previous conjectures, based on observations of the growth kinetics of a murine melanoma and a rat mammary carcinoma, that such a relation may be a general feature of tumor growth. Data on the growth of xenografts of human colorectal tumors in immune-deprived mice suggest that the growth relation reflects the ability of a particular species to support a tumor of certain max size. The existence of this relation simplifies the task of predicting complete patterns of undisturbed neoplastic growth in these species. (20 refs)

78-4708 **The Contributions of Diet and Childbearing to Breast-Cancer Rates.** (Eng) Hems, G. (Dept. Community Medicine, Univ. Medical Buildings, Foresterhill, Aberdeen, Scotland). *Br J Cancer* 37(6): 974-982; 1978.

The 1970-1972 mean, age-standardized, breast cancer mortality rates for women of 41 countries, were closely correlated with dietary data for 1964-1966. Partial correlation analysis indicated that breast cancer rates were positively correlated with total fat, animal protein and animal calories, independently of other components of diet. These three components were correlated with one another so closely that it was not possible, with available data, to say whether any one was associated with breast cancer independently of the other two. In addition to, and independently of, these correlations, breast cancer was associated with consumption of refined sugar. In 26 countries, breast cancer mortality rates at 50-54 yr during 1964-1967 were closely correlated with childbearing, expressed as mean family size for women aged 45-49 yr in 1960-1961. However, this correlation was not independent of the correlations with diet, and it was concluded that variations of breast cancer rates among countries arose predominantly from differences in diet. The variation of breast cancer risk with childbearing, observed in clinical studies, seemed best regarded as a second gradient of risk, seen more readily as variation of breast cancer rates within a population, in which differences in diet would be relatively small. The physiological basis for the association between breast cancer and diet was not clear. The dietary associations did not correlate in an obvious way with height, obesity, and estrogen levels, factors observed in clinical studies to influence risk of breast cancer. That the observed statistical associations were real was supported by published findings on the effects of diet on mammary cancer in experimental animals, as well as the lower rates of breast cancer among vegetarians. (44 refs)

78-4709 **Incidence of Salivary Gland Tumours in Scotland: Accuracy of National Records.** (Eng) Lennox, B. (Dept. Pathology, Western Infirmary, Glasgow G11 6NT, Scotland); Clarke, J. A.; Drake, F.; Ewen, S. W. *Br Med J* 1(611): 687-689; 1978.

The accuracy of the Information Services Division files on the incidence of malignant and benign salivary gland tumors in Scotland was evaluated by comparing the files with case notes and pathology department records from selected hospitals. Because of miscoding of benign tumors as malignant, the true incidence of the two tumor types could not be ascertained. The study did indicate, however, that the incidence of all tumors of major salivary glands exceeds 40/million/yr. This incidence is higher than that in any other nationality except Canadian Eskimos. Analysis of pathology records indicated that eastern Scotland has an unusually high incidence and that the reported figures in other areas are likely to be too low. (8 refs)

78-4710 **Occupational Hazards to Reproduction and Health in Anaesthetists and Paediatricians.** (Eng) Rosenberg, P. H. (Dept. Anaesthesiology, Helsinki Univ. Central Hosp., SF-00290 Helsinki 29, Finland); Vanttinen, H. *Acta Anaesthesiol Scand* 22(3): 202-207; 1978.

The results of a questionnaire sent to Finnish anesthetists and pediatricians (controls) indicated that exposure to halothane anesthetic vapor in an operating room does not produce a higher risk of reproductive or teratogenic complications or malignancies compared with that in the control group. (19 refs)

78-4711 **Mesothelioma and Asbestos in the Province of Quebec, 1969-1972.** (Eng) Theriault, G. P. (Dept. Social and Preventive Medicine, Laval Univ., Quebec, Canada); Grand-Bois, L. *Arch Environ Health* 33(1): 15-19; 1978.

A retrospective case-control study of deaths from mesothelioma in the Province of Quebec during 1969-1972 was undertaken to determine the relationship between this tumor and industrial exposure to asbestos. Fifty-three confirmed cases and 15 doubtful cases of mesothelioma occurred during the study period. Most of the patients died between the ages of 40 and 70 yr, and the overall male-to-female ratio was 2 to 1, being especially high for pleural mesothelioma. The incidence was highest in urbanized areas. Among the 32 confirmed mesothelioma patients for whom occupational histories were obtained, 34.4% had histories of direct exposure to asbestos compared with only 1.6% in the control group. Exposure was related to processing in more cases than to mining. None of the women who died of mesothelioma had a history of direct occupational exposure. No relationship was found between cigarette consumption and mesothelioma. Chrysotile fibers, which are primarily associated with mining, appear to be less related to mesothelioma than other types of asbestos fibers, which are more involved in processing. (17 refs)

78-4712 **A Summary of Asbestos Fibre Counting Experience in Seven Countries.** (Eng) Gibbs, G. W. (Occupational Health and Safety Unit, McGill Univ., Montreal, Quebec, Canada); Baron, P.; Beckett, S. T.; Dillen, R.; du Toit, R. S.; Koponen, M.; Robock, K. *Ann Occup Hyg* 20(4): 321-332; 1978.

The membrane filter method for evaluating airborne concentrations of asbestos in the work place was evaluated in laboratories in Belgium, Canada, Finland, Germany, South Africa, the United Kingdom, and the United States. A comparison of the fiber counts in the different countries revealed large differences for single samples, but laboratories that were in close contact and regularly exchanged samples had better agreement of their results. Methods for improving the reproducibility of the counts are outlined. (9 refs)

78-4713 **A Cohort Study on Trichloroethylene Exposure and Cancer Mortality.** (Eng) Axelson, O. (Dept. Occupational Medicine, Regional Hosp., 701 85 Orebro, Sweden); Andersson, K.; Hogstedt, C.; Holmberg, B.; Molina, G.; de Verdier, A. *J Occup Med* 20(3): 194-196; 1978.

A cohort of 518 Swedish men exposed to trichloroethylene (TCE) was studied with respect to cancer mortality. The men were divided into high-/and low-exposure categories as estimated by the av trichloroacetic acid level in the urine: those with levels > 100 mg/liter were in the high exposure group, and those with levels < 100 mg/liter were in the low-exposure group. Requiring 10 yr of latency time after the date of first employment (assumed to correspond to the time of first exposure) resulted in 3,643 person-yr of observation. Subdividing the cohort with regard to 10 yr of latency time as to high-/and low-exposure categories resulted in 548 and

3,095 person-yr of observation, respectively. There was a close agreement between expected and observed number of cancer cases in both the high-/and low-exposure groups. No particular type of cancer was overrepresented, although there were two leukemias and two stomach cancers. Both the leukemia and stomach cancer cases were in the low-exposure category. It seems justified to consider TCE as a possible human carcinogen, but one that is not more potent than many other industrial compounds. There is probably no serious hazard at low exposures. (21 refs)

78-4714 **Association of Cancer Sites with Tobacco and Alcohol Consumption and Socioeconomic Status of Patients: Interview Study from the Third National Cancer Survey.** (Eng) Williams, R. R. (Dept. Internal Medicine, Univ. Utah Coll. Medicine, Salt Lake City, UT 84121); Horm, J. W. *J Natl Cancer Inst* 58(3): 525-547; 1977.

From personal interviews with 7,518 invasive cancer patients obtained in the Third National Cancer Survey, the quantitative lifetime use of cigarettes, cigars, pipes, unsmoked tobacco, wine, beer, hard liquor, and combined alcohol were recorded, as well as education and family income level. In an initial screening analysis of these data, Mantel-Haenszel 2 x 2 contingency tabulations and multiple regression analyses were used to compare each specific cancer site with controls from other sites to test for associations with exposure variables. Significant positive associations with cigarette smoking were found for cancers of the lung, larynx, oral cavity, esophagus, stomach, pancreas, bladder, kidney, and uterine cervix. Other forms of tobacco were associated with cancers of the oral cavity, larynx, lung, and cervix. Consumption of wine, beer, hard liquor, and all combined showed positive association with neoplasms of the oral cavity, larynx, esophagus, colon, rectum, breast, and thyroid gland. College education and high income showed positive associations with cancers of the breast, thyroid gland, uterine corpus, and melanomas in men. These indicators of high socioeconomic status showed inverse associations with invasive neoplasms of the uterine cervix, lung, lip-tongue, and colon in women. College attendance (but not income) showed an inverse association with stomach cancer and positive association with pancreatic cancer in men. Still other tumor sites showed "suggestive" associations with each of these exposure variables. In this study, age, race, sex, smoking, drinking, education, income, parity, foreign birth, marital status, and geographic location were used as stratification variables separately or in combination, to assess and control their potentially confounding effects and to examine interactions between exposure and control variables within a stratum. (41 refs.)

78-4715 **Tobacco Smoking, Occupation, and Malignant Tumors in the Rural Population of Vratsa, Bulgaria.** (Bul) Nikolov, I. (Oncology Res. Center, Medica Acad., Bulgaria); Stoianov, I.; Stoichev, I.; Draganov, I.; Pet kova-Bocharova, T.; Chernozemski, I. *Onkologiia* 14(2) 61-66; 1977.

Cancer incidence and its relationship to tobacco smoking an occupation were studied in 27 villages of the district of Vratsa, Bulgaria, during 1965-1974. Only subjects aged > 16 y were considered. Tumors were found in 315/46,913 smoker and in 239/61,244 nonsmokers. The relative risk of tumor of the mouth, stomach, urinary bladder, and, especially, o the bronchi and lungs was significantly higher among smokers than nonsmokers ($p < 0.001$), but there was no significant difference between these groups in the incidence cancer of the renal pelvis, ureter and urinary trac Compared with subjects of other occupations, far workers were found to have increased risk of skin canc (5.1x), cancer of the renal pelvis and ureter (3.3x bladder cancer (3.3x), gastric cancer (2.1x), and labi cancer (2.1x). (18 refs)

78-4716 **Energy Expenditure, Cigarette Smoking, an Blood Pressure Level as Related to Death fro Specific Diseases.** (Eng) Paffenbarger, R. S. (Dept. Famil Community and Preventive Medicine, Stanford Univ. Sc Medicine, Stanford, CA, 94305); Brand, R. J.; Sholtz, R. I Jung, D. L. *Am J Epidemiol* 108(1): 12-18; 1978.

In a 22-yr follow-up of 3,686 San Francisco longshoreme the roles of physical activity, cigarette smoking habit, an systolic blood pressure level were evaluated independently i relation to risk of death from a broad range of diseases. Smoking pattern and blood pressure status were established i 1951, and job activity was assessed annually during the fo low-up period. Heavy cigarette smoking predicted increase risk of death from heart attack (2.09), cancer (1.71; lung cancer, 3.19), chronic obstructive respiratory disease (2.63), an pneumonia (2.27). (12 refs.)

78-4717 **Mining, Lung Cancer and Smoking.** (Eng) Axelson, O. (Dept. Occupational Medicine, Regiona Hosp., Linkoping, Sweden); Sundell, L. *Scand J Work Environ Health* 4(1): 46-52; 1978.

Mortality from lung cancer during 1956-1976 was studied i smokers and nonsmokers who had worked in two adjacen Swedish zinc-lead mines. The local register of deaths an burials was used as a source of material, which included 2 cases of lung cancer and 174 controls who had died from causes other than lung cancer. (Controls were those 3 person who had died before and after each case.) There was a 16-fol increase in lung cancer mortality among the miners. Significantly more of the lung cancer cases (72%) had been expose to underground mining than the controls (11%). Althoug the risk of dying from lung cancer was higher for nonsmoker han for smokers, smoking miners had a higher risk of deveoping lung cancer than would be expected from smoking lone. Furthermore, the av induction-latency time for lung ancer was about 9 yr shorter for the smokers than for the onsmokers, and the smokers died from lung cancer approxinately 11 yr earlier than the nonsmokers. It is possible that moking increases the thickness of the bronchial mucus layer, hereby protecting the bronchial epithelium from alpha radiaion due to Radon daughters. However, after the radiogenic nduction of carcinogenesis, tobacco smoke might have a pronoting effect. (20 refs)

78-4718 **Incidence of Salivary Gland Neoplasms in Greenland with Special Reference to an Anaplastic Carcinoma.** (Eng) Nielsen, N. H. (Univ. Inst. Forensic Medicine, Frederik V's vej 11, DK-2100 Copenhagen 0, Denmark); Mikkelsen, F.; Hansen, J. P. *Acta Pathol Microbiol Scand (A)* 86(2): 185-193; 1978.

The incidence and histopathology of 42 salivary gland neoplasms (17 benign, 25 malignant) diagnosed between 1955 and 1974 in Greenland are reviewed. The 25 malignant tumors occurred in 10 men and 15 women 12-68 yr of age. The yearly incidence rates between 1965 and 1974, age-adjusted to the European standard population, were 4.5/100,000 for men and 9.6/100,000 for women; the observed/expected ratios during this period were 7.5 for men and 9.2 for women. Twenty-two malignant tumors were located in the parotid gland, 2 were in the submandibular gland, and 1 was in the palate. Histological classification indicated 1 mucoepidermoid tumor, 1 adenoid cystic carcinoma, and 23 undifferentiated, anaplastic carcinomas histologically identical to malignant lymphoepithelial lesions. The 5-yr determinate survival rate was only 14%. It is suggested that virus infection and/or dietary deficiencies may be etiological factors in these tumors; however, secondary nasopharyngeal carcinoma could not always be excluded. (50 refs.)

78-4719 **Environmental Factors in the Pathogenesis of Thyroid Cancer.** (Ita) Gubetta, L. (Instituto di Anatomia Patologica, Centro Studi sulla Endemia di Gozzo, Ospedale Mauriziano, Torino, Italy); Costa, A. *Minerva Med* 69(34): 2313-2320; 1978.

Between 1957 and 1974, 227 cases of thyroid cancer were diagnosed at a Northern Italian hospital. The increasing incidence of this neoplasm in the area served by the hospital is connected with a geographical factor. The disease has been shown to be most prevalent in two mountainous areas of endemic goiter (1.46 and 2.16 cases/100,000 inhabitants, respectively, as opposed to an av national incidence of 0.74/100,000). High levels of thyrotropin have also been linked to thyroid cancer. Animal experiments on the effects of iodine-deficient diets, thyrotropin administration and partial thyroidectomy are cited. Histologically, papillary carcinomas, the most common type of thyroid cancer, were less common than follicular carcinomas; however, since 1969, the number of papillary lesions increased, possibly due to population changes (immigration from the South, migration to the cities). Undifferentiated forms of tumor increased least. Ionizing radiation may also contribute to the development of thyroid cancer: of 15 young people in the hospital series, 7 had received therapeutic irradiation 10-12 yr prior to tumor appearance. (61 refs.)

78-4720 **Development of Thyroid Carcinoma in Residents of a Region Endemic for Goiter.** (Rus) Neimark, I. I. (Dept. Surgery, Second Railroad Clinical Hosp., Barnaul, USSR); Timoshnikov, V. M. *Probl Endokrinol (Mosk)* 24(3): 28-32; 1978.

An attempt was made to evaluate the role of various factors in the etiology of thyroid cancer. Of 338 patients with malignant thyroid tumors, 106 were residents of a region of the Altai Mountains, USSR, that is endemic for goiter. Ninety-nine patients had eu- and hypothyroidism; only 7 had symptoms of hyperthyroidism. A history of endemic goiter was found in 86.6% of the patients. The cancer patients had significantly decreased levels of iodine in the thyroid tissue (1.9725 g% compared with 3.9676 g% in healthy subjects and 2.2897 g% in patients with goiter only), and increased levels of copper (27.011 mg%, compared with 14.97 mg% in healthy subjects and 18.54 mg% in patients with goiter) and cobalt (6.204 mg%, compared with 2.65 and 4.42 mg%, respectively). (14 refs.)

78-4721 **Thyroid Carcinoma in Kenya.** (Eng) McGill, P. E. (Westwood Hosp., Beverley, N. Humberside, England). *Trop Geogr Med* 30(1): 82-86; 1978.

Thyroid carcinoma was diagnosed between 1964 and 1969 in 63 Kenyans of African origin. Most of the tumors were histologically differentiated; the female:male ratio was approx 5:1. Follicular tumors were found mainly in highland tribes in which endemic goiter is known or presumed to exist; papillary carcinoma predominated in lowland tribes. (25 refs.)

78-4722 **Dose Assessment Analogies.** (Eng) Lindell, B. (Swedish Natl. Inst. Radiation Protection, Fack, S-104 01 Stockholm, Sweden). *Environ Health Perspect* 22: 75-78; 1978.

Assessments of radiation risks are based on the absorbed dose in the organs and tissues of interest, and they assume a linear, nonthreshold dose-response relationship for cancer induction or genetic effects at low doses. Similar assessments may be made for nonradioactive, mutagenic or carcinogenic substances. The quantity that would correspond to the absorbed radiation dose as the basis for risk assessment would be time integral of the local tissue concentration of the carcinogen, which may not be identical for inhaled or ingested substances. The assessment involves calculations that are very similar to those made in the assessment of radiation risks. Such estimates for nonradioactive pollutants would provide a more appropriate basis for comparisons with radiation risks. In the cases of lung cancer, the risk is assumed to be proportional to the total amount of carcinogen inhaled. (5 refs)

78-4723 **Offspring of Childhood-Cancer Survivors (Meeting Abstract).** (Eng) Li, F. P. (NCI, Bethesda, MD, 20014); Holmes, F. F.; Holmes, G. E.; Jaffe, N. *Proc Am Assoc Cancer Res* 19: 218; 1978. (no refs)

78-4724 **Studies of the Mortality of A-Bomb Survivors. 6. Mortality and Radiation Dose, 1950-1974.** (Eng) Beebe, G. W. (Clinical Epidemiology Branch, NCI, Bethesda, MD, 20014); Kato, H.; Land, C. E. *Radiat Res* 75(1): 138-201; 1978.

This updated (through 1974) study of cancer deaths among atomic bomb survivors provides more evidence of the carcinogenic effect of radiation. The relative and absolute risk estimates for 1971-1974 (1.48 and 4.19) were similar to those for 1967-1970 (1.28 and 3.32); the overall estimates for 1950-1974 were 1.29 and 2.21, respectively. In addition to leukemia and thyroid, breast, and lung cancer, cancer of the esophagus, stomach, and urinary organs and the lymphomas should be included among the cancers caused by the ionizing radiation from the explosions. Evidence of radiation carcinogenesis is much stronger for Hiroshima than for Nagasaki victims of the bombs. The leukemogenic effect was still present during 1970-1974, having lasted longer than anticipated, but it is declining. The av absolute risk for other malignant neoplasms now exceeds that for leukemia. Accumulating evidence of radiation-induced cancer in an increasing number of tissues shows that most tissues may have been sensitive to the radiation delivered by the bombs. The minimal latent period for most of the carcinogenic effects was <15 yr and it depended on age and cancer site. Age in 1945 played an important but variable role in the carcinogenic effect, but one that cannot be understood fully without further study. This study rejects the hypothesis that radiation accelerates natural aging, and it concludes that the results of exposure to large, single doses of ionizing radiation are focal and largely confined to cancer. (36 refs)

78-4725 **Army Technologists: 29-Year Follow up for Cause of Death.** (Eng) Jablon, S. (Medical Follow-up Agency, Natl. Acad. Sciences-Natl. Res. Council, Washington, DC); Miller, R. W. *Radiology* 126(3): 677-679; 1978.

A previous 18-yr (1946-1963) follow-up study revealed no significant excess of cancer among men who served in the Army during World War II as radiological technologists (6,560), compared with men who served as medical, laboratory, or pharmacy technologists (6,826). Extension of the follow-up by 11 yr (1946-1974) revealed that 145 former radiological technologists had died of cancer compared to 158 controls. There were no statistically significant differences between these groups for individual sites of cancer or for deaths from other causes. There is, therefore, no evidence that military radiological technologists have suffered adverse effects as a consequence of their occupational exposures. (10 refs)

78-4726 **The Queensland Melanoma Project.** (Eng) Beardmore, G. L. (Queensland Melanoma Project, Princess Alexandra Hosp., Ipswich Road, Woolloongabba, 4102, Queensland, Australia). *Int J Dermatol* 16(10): 831-835; 1977.

Epidemiological data on 1,500 patients treated for malignant melanoma in Queensland, Australia, between 1963 and 1969 are presented. The yearly incidence was 16/100,000, with a sex distribution of 55% women and 45% men. No patient was $<$ 10 yr old; the smallest number of patients was in the 10- to 19-yr age group, the largest in the 40- to 49-yr age group. About 23% of the melanomas were $<$ 1.0 cm in diameter, 50% were 1.0-1.9 cm, 17% were 2.0-2.9 cm, and the remaining 10% were $\geq$3.0 cm. In men, 44% of the lesions were on the back; in women, 36% were on the lower limbs. Multiple primary cutaneous melanomas were observed in 4.8% of the patients. The 5-yr survival rate for the 1,500 patients was about 74%, with women having a better chance of survival than men (82% vs 65%). (8 refs.)

78-4727 **Skin Tumours in White South Africans. Part I. Patients, Methods and Incidence.** (Eng) Whiting, D. A. (Dermatology Section, Dept. Internal Medicine, Southwestern Medical Sch., Univ. Texas Health Science Center, 5323 Harry Hines Blvd., Dallas, TX, 75235). *S Afr Med J* 53(3): 98-102; 1978.

The incidence of skin tumors seen in whites recorded in a dermatological practice in the southern Transvaal region of South Africa between 1968 and 1975 is reported. A total of 4,861 skin tumors were diagnosed in 3,597/15,153 patients seen during this period. Solar keratosis (1,523 cases), seborrheic keratosis (624), basal cell carcinoma (574), intradermal nevus (428), polyps and skin tags (312), epidermal cysts (257), and keratoacanthomas (139) made up the majority of the lesions. Only 18 cases of malignant melanoma (versus an expected number of 30) were noted; this could be due to a low referral rate. (27 refs)

78-4728 **Various Epithelial and Non-Epithelial Tumors Spontaneously Occurring in Long-Lived Mice of A/St, CBA, C57BL/6 and Their Hybrid Mice.** (Eng) Kawada, K. (Dept. Pathology, Gifu Univ. Sch. Medicine, Tsukasa-machi 40, Gifu 500, Japan); Ojima, A. *Acta Pathol Jpn* 28(1): 25-39; 1978.

The incidence and pathological nature of various kinds of epithelial and nonepithelial tumors and tumorlike lesions that develop spontaneously in aged mice of strains A/St, C57BL/6, and CBA and of (C57BL/6 x CBA)F_1 hybrids were investigated. Mice were sacrificed at ages 13 to > 36 mo old. Tumor incidence was high in the CBA strain (55%) and in the hybrids (46.7%). In the A/St strain, epithelial tumors exceeded lymphoreticular neoplasms. Most of the neoplasms found in the C57BL/6 strain were lymphoreticular, and they occurred in the youngest age group in contrast to the other strains or the hybrids. Some tumors were successfully transplanted into the same strain of mice. A tumor line showing phagocytic activity and growing only in the liver, even when injected sc, was obtained from a reticulum cell neoplasm (Dunn type A) that had originated in the liver of a (C57BL/6 x CBA)F_1 hybrid male mouse. Additional tumors that were identified in this population included liver cell, mammary, pulmonary, renal, small intestinal, and islet cell carcinomas, ovarian and oviduct tumors, a cavernous hemangioma, angiosarcomas, and pheochromocytomas. (18 refs)

78-4729 **Incidence of Cancer in Relatives of Children with Retinoblastoma.** (Eng) Fedrick, J. (Unit Clinical Epidemiology, Univ. Oxford, Oxford, OX3 7LF, England); Baldwin, J. A. *Br Med J* 1(6105): 83-84; 1978.

The families of 11 patients with retinoblastoma (1 with bilateral disease) were interviewed to determine the incidence of cancer among the members. Five cancers were found compared to the 1.5 expected. Other family members reportedly had developed cancer, but the diagnosis could not be verified. (5 refs)

78-4730 **Relative Importance of Risk Factors in Bladder Carcinogenesis.** (Eng) Miller, C. T. (Health Protection Branch, Health and Welfare Canada, Ottawa, Canada); Neutel, C. I.; Nair, R. C.; Marrett, L. D.; Last, J. M.; Collins, W. E. *J Chronic Dis* 31(1): 51-56; 1978.

Responses from outpatients with urological complaints were recorded in a detailed questionnaire exploring potential risk factors in bladder cancer. The subjects were subsequently classified on the basis of hospital records as cancer cases or controls. Each of the 265 cases was matched to 2 controls of the same age and sex. History and treatment of tuberculosis of all subjects were verified through the four geographically adjacent tuberculosis registers. Analysis of exposures of matched triples revealed six statistically significant associations with bladder cancer in men [smoking, gout, allergies (self), allergies (family), occupational radiation exposure, and occupational exposure to chemicals.] In women, there was one significant positive association (coffee consumption) and one significant negative association (allergies, self). Although the relative risk of isonicotinic acid hydrazide (INH) exposure in women was the highest observed (3.0), the number of exposed individuals was too small to permit any meaningful conclusion. Discriminant analysis was used to rank the variables in order of their importance as potential risk factors. Statistically significant ability to discriminate cases from controls was observed in men for (1) allergy (family), (2) occupational exposure to radiation, (3) occupational exposure to chemicals, and (4) smoking. In women, the significant discriminators were (1) coffee consumption, (2) history of tuberculosis and INH treatment, and (3) allergy (self, protective effect). The suggestion that INH is a potential carcinogen indicates that further study of the association of the drug with bladder cancer is in order. (23 refs)

78-4731 **On the Endemic Occurrence of Papillomas. Evaluation of Environmental Factors in the Occurrence of Urothelial Tumors in the Province of Steiermark.** (Ger) Scholl, H. (Urologische Abteilung, Landeskrankenhaus, Auenbruggerplatz 1, A-8036 Graz, Austria); Narath, P. *Acta Chir Austriaca* 9(4): 99-103; 1977.

The features of benign bladder papillomas were evaluated in 888 patients (185 women, 703 men) from the province of Steiermark, Austria, and the effects of environmental pollutants (such as contamination of water with nitrosamines, bacteria, and minerals) on the incidence of these tumors were determined. There was a 2.5-fold higher morbidity in industrial regions than in agricultural areas. These higher morbidity areas also had increased sulfur dioxide and dust levels in the atmosphere. There were no indications that bladder papillomas were endemic to the Steiermark region. Industrial air pollution had a greater influence on the incidence of papillomas than groundwater pollution. The highest morbidity rate was observed around Graz, and it was attributed to an increase in air pollution as a result of automobile exhausts and poor air circulation. Because of the threefold increase in the incidence of bladder papillomas in areas with excessive pollution, it is suggested that the benign nature of the papillomas be reevaluated. (10 refs.)

78-4732 **Epstein-Barr Virus Infections in Brazil. II. Hodgkin's Disease.** (Eng) Evans, A. S. (Dept.

Epidemiology and Public Health, Sch. Medicine, Yale Univ., 60 College St., New Haven, CT, 06510); Carvalho, R. P.; Frost, P.; Jamra, M.; Pozzi, D. H. *J Natl Cancer Inst* 61(1): 19-26; 1978.

Antibody levels to Epstein-Barr virus (EBV) and to eight other viral antigens were tested in 67 Brazilian patients with Hodgkin's disease (HD) and matched healthy controls. Antibody to the viral capsid antigen (VCA) of EBV was tested with the use of the Jijoye line as a source of antigen. Antibody was present in the sera (diluted 1:10) of all 67 HD patients, and it was elevated to 1:320 or greater in 31.3%; the geometric mean titer (GMT) was 1:146. In 46 patients tested with the use of the EB-3 cell line as a source of antigen, all sera again showed the presence of antibody; 28% had elevated titers of 1:320 or greater, and the GMT was 1:110. Titers were highest in men, in patients aged 30-39 yr, in severe forms of HD, and in Stage 2 disease, although none of these differences was statistically significant. There was a statistically higher GMT and a higher frequency of elevated titers in the patients than in the controls. Heterophile antibody and EBV-specific IgM antibody levels were not elevated in HD patients with respect to controls, but 20.5% of the HD patients had antibody to the early antigen of EBV in their sera. Antibody levels for herpes simplex virus, cytomegalovirus, rubella, measles, papovavirus, and parainfluenza viruses in HD patients were not significantly elevated over those of the controls. It appears that enough epidemiologic and virologic evidence points to a relationship between EBV and some cases of HD to merit further evaluation of the role of EBV in the pathogenesis of HD. (39 refs)

78-4733 **Geographical Distribution of Respiratory Tract Tumors in Upper Silesia.** (Pol) Zemla, B. (Instytut Onkologii, ul. Wybrzeze Armi Czerwonej 15, 44-101 Gliwice, Poland). *Nowotwory* 27(3): 249-254; 1977.

The results of an epidemiological survey of the incidence of malignant respiratory tract tumors among the urban population of Upper Silesia, Poland, are presented. Tumor incidence per 100,000 individuals in different localities ranged from 9.3 to 24.2, and the tumors were more frequent among men than women (ratio 4.64:1). There was a higher incidence in men above age 45 and in women over age 64. (no refs.)

78-4734 **The Epidemiology of Primary Liver Cancer Based on Tests of Autopsy Material over the Period 1962-1975.** (Pol) Zielinska, K. (Inst. Pathomorphological Diagnostics, Medical Center Postgraduate Education, Warsaw-Bielany Hosp., Warsaw, Poland); Czorniuk, A.; Kobuszewska-Faryna, M. *Pol Tyg Lek* 32(43): 1669-1672; 1977.

A review of autopsy material at a Warsaw hospital revealed 61 cases of primary liver cancer (1.2% of all autopsies performed over a 13-yr period). The cancer cases included 49 men and 12 women. Cirrhosis (excluding biliary and cardiac cirrhosis) was present in 58 of the cases, and it was four times more frequent in men than in women. The patients lived in a district in which the av mortality rate from liver cancer in the period 1970-1975 was 6.03 for men and 1.68 for women. (18 refs.)

78-4735 **Hepatocellular Carcinoma and Hepatic Cirrhosis in the West of Scotland: A 25-Year Necropsy Review.** (Eng) Burnett, R. A. (Dept. Pathology, Univ. Glasgow, Glasgow G11 6NT, Scotland); Patrick, R. S.; Spilg, W. G.; Buchanan, W. M.; MacSween, R. N. *J Clin Pathol* 31(2): 108-110; 1978.

The incidence of hepatocellular carcinoma and hepatic cirrhosis in five hospitals in Western Scotland between 1950 and 1974 was studied retrospectively, based on a necropsy review of 313 cases. During this period, the necropsy incidence of the tumor rose to twice its original level, and this trend was noted for both men and women. However, the tumor was found four times more often in men than in women. A comparison of the cirrhosis and tumor incidence in three hospitals indicated that the incidence of cirrhosis remained stable until the last half-decade, when it increased. It is suggested that cirrhosis acts as a promoting agent in the presence of a primary carcinogenic agent(s); this does not exclude the possibility that the same agent(s) may express both a cirrhogenous and/or carcinogenic effect in some persons. (9 refs)

78-4736 **The Changing Pattern of Liver Disease in South African Blacks.** (Eng) Isaacson, C. (Dept. Pathology, Sch. Pathology, South African Inst. Medical Res., Johannesburg, S. Africa). *S Afr Med J* 53(10): 365-368; 1978.

The pattern of liver disease in South African Blacks was studied by reviewing the results of 201 liver biopsies performed between 1959 and 1960 and 729 biopsies performed between 1975 and 1976. Changes were noted in the histological appearances of micronodular cirrhosis. During the latter period, there were more instances of fatty change, alcoholic hepatitis, alcoholic hyalin, and alcoholic cirrhosis. These changes are presumably related to the liberalization of the liquor laws in 1962. Prior to that time, alcohol consumption by blacks was confined to home-brewed beverages containing large quantities of iron derived from the containers in which the liquor was brewed. No change in the histological pattern or incidence of macronodular cirrhosis and hepatocellular carcinoma was observed. A total of 22 cases of hepatocellular carcinoma were diagnosed during the first period, 77 during the second. Twenty-one cases showed an associated macronodular cirrhosis, 10 an associated micronodular cirrhosis. (13 refs)

78-4737 **Occupational Cancers of the Ethmoid Region.** (Fre) Haguenauer, J. P. (Societe de medecine du travail et d'ergonomie de Lyon, 8, avenue Rockefeller, 69373 Lyon Cedex 2, France); Romanet, P.; Duclos, J. C.; Guinchard, R. *Arch Mal Prof* 38(9): 819-823; 1977.

In a series of 115 patients with cancer of the ethmoid region, 32 worked in a variety of occupations related to wood. Thirty of these patients had an adenocarcinoma and 2 had either an adenocarcinoma or a reticulosarcoma. The usual duration of exposure was 14-15 yr. At the time of diagnosis, 27 were still working, 2 had retired (2 and 12 yr, respectively), and 3 had been in other occupations for 11, 15, and 30 yr, respectively. An additional patient in the series was a woman with adenocarcinoma who was the wife of a cabinet maker; she had lived above the shop for 10 yr. The etiological agent responsible for the cancers is thought to be tannin, because: (1) workers involved with high-tannin-content wood such as oak and chestnut are more likely to develop cancer of the ethmoid and (2) workers in tanneries and leather factories also have a high incidence of ethmoid cancer. Other agents thought to be responsible for cancers of the ethmoid region or nasal fossae are nickel, radium, asbestos, and hexamethylphosphoramide. (no refs.)

78-4738 **Usefulness of Population Controls in Retrospective Studies of Alcohol Consumption. Experience from a Case-Control Study of Esophageal Cancer in Ille-et-Vilaine, France.** (Eng) Jensen, O. M. (Unit Epidemiology and Biostatistics, International Agency Res. Cancer, 150 cours Albert Thomas, 69008 Lyon, France); Tuyns, A. J.; Pequignot, G. *J Stud Alcohol* 39(1): 175-182; 1978.

In a study of the association of alcohol consumption with esophageal cancer, controls were taken from patients hospitalized for non-alcohol-related diseases and from the general population, and the effectiveness of the two groups as controls was compared. Both control groups had similar rates of alcohol consumption. (23 refs)

78-4739 **General and Etioepidemiological Characteristics of Cancer of the Upper Respiratory and Digestive Tracts.** (Fre) Cachin, Y. (Departement de chirurgie cervico-faciale et ORL, Institut Gustave-Roussy, 16 bis, avenue Paul-Vaillant-Couturier, 94800 Villejuif, France). *Rev Prat* 28(31): 2359-2364; 1978.

According to statistics compiled in 1967, cancers of the upper respiratory and digestive tracts (URD) represent 12% of all cancers and 8% of cancer deaths in France. The epidemiology of URD cancers is discussed on the basis of 5,748 patients observed at the Gustave-Roussy Institute from 1959 to 1970. Approx 95%-97% of these patients were men. A high proportion of patients with cancers of the tongue, oropharynx, hypopharynx, and esophagus were alcoholics. Alcoholics did not have a high incidence of cancers of the nasal cavity and sinuses or the rhinopharynx. Cancers at these sites were more likely to occur among persons in certain occupations, such as woodworkers, carpenters, asbestos miners, and nickel workers. Heavy smokers tended to develop cancers of the same areas as alcoholics in addition to cancers of the lung, lip, and buccal cavity. Again, tobacco usage was not strongly associated with cancers of the upper respiratory tract, such as the nasal cavities and rhinopharynx. URD cancers have a varied incidence in relationship to geographical region and sex. Examples of these variations are provided. (no refs)

78-4740 **Oesophageal Carcinoma in India. Some Epidemiologic and Morphologic Considerations.** (Eng) Mehrotra, M. L. (Dept. Pathology, Inst. Medical Science, Banaras Hindu Univ., Varanasi, India); Lal, H.; Pant, G. C.; Vaidya, M. P.; Gupta, I. M. *Trop Geogr Med* 29(4): 353-358; 1977.

The frequency of esophageal carcinoma in relation to gastrointestinal tract and total malignancies was investigated in a hospital population in Varanasi, India. The 202 patients who presented with this carcinoma comprised 2.39% of all malignancies and 17.11% of all gastrointestinal tract malignancies noted between 1964 and 1975. The highest tumor incidence occurred in the sixth decade of life with a male: female ratio of 2.2:1. This ratio decreased with age from 3:1 in the fourth decade of life to 1.7:1 in the seventh. The tumor involved the middle third of the esophagus in 104 patients, the lower third in 72, and the upper third in 26. Squamous cell carcinoma was the most frequent histologic type (193 cases), with 3 cases of adenocarcinoma and 6 of anaplastic carcinoma also being reported. The gross appearance of the lesion was fungating in 188 patients, fibromatous in 8, and crateriform in 6. (30 refs)

78-4741 **Epidemiology of Primary Cancer of the Bronchus in the African Negro.** (Fre) Schmidt, D. (Service de Pneumophtisiologie, CHU de Treichville, BPV 13, Abidjan, RCI, Ivory Coast, W. Africa); Sanchez, J.; Tiendrebeogo, H.; Roudaut, M. *Nouv Presse Med* 7(21): 1827-1830; 1978.

Epidemiological data are presented on 86 Ivory Coast African negroes (81 men, 5 women) with primary cancer of the lung observed over a 5-yr period (1972-1976). The av age of the patients was 50.4 yr, and 68% were >45 yr of age. Risk appeared higher in the rural population, as 56% of the patients were from rural areas. Tobacco use was definitely related to the development of cancer: 70/86 patients were smokers. Precise data on tobacco use were obtained in 63 patients: 51 smoked for >20 yr and 44 smoked >20 cigarettes per day. The Ivory Coast is free of industrial pollution; however, brush fires are frequent and the wood carbon is redistributed by the prevailing winds. Tuberculosis is endemic in the Ivory Coast (3,000 new cases/yr in a population of 4 million), and tuberculosis preceded the lung cancer in 8/86 patients. (20 refs)

78-4742 **Development of Carcinomas in Billroth II-Resected Stomachs.** (Ger) Radomsky, J. (Chirurgische Klinik, Krankenhaus Nordwest, D-6000 Frankfurt am Main-Praunheim, W. Germany); Alken, P. *Therapiewoche* 28(8): 1609; 1978.

From 1964 to 1976, gastric resection was performed in 3,000 patients: for gastric ulcer in 1,807, for primary gastric tumors in 1,156, and for carcinoma of the gastric stump after Billroth II gastric resection in 37. The underlying disease that necessitated the Billroth II resection was gastric ulcer in most cases. The av latent period after the first surgery was 27.8 yr. The gastric stump carcinoma was localized to the anastomotic ring in 46% of all patients, it invaded the gastroenterostomy and the entire gastric stump in 35%, and it was distant from the anastomosis in 19%. These observations and literature data indicate that there is an increased risk of gastric stump carcinoma after Billroth II gastric resection. (no refs)

78-4743 **Epidemiology of Gastric Cancer. Part II.** (Ger) Wolff, G. (Regierungskrankenhaus, Scharnhorststrasse 37, DDR-104 Berlin, E. Germany); Lauter, J. *Arch Geschwulstforsch* 47(6): 531-540; 1977.

A comparison of 257 patients with gastric cancer and 766 patients with different stages of chronic gastritis or normal gastric mucosa revealed several common variables between the cancer and gastritis groups. However, the former could be distinguished from the latter by exposure to carcinogens, social class, genetic factors, and digestive diseases. It is suggested that these factors are important to the etiology of gastric cancer in atrophic gastritis patients. (13 refs.)

78-4744 **Cancer Incidence in the Province of Zaragoza from 1970 to 1974.** (Spa) Zubiri, A. (Junta Provincial, Asociacion Espanola Contra el Cancer, Joaquin Orus, Zaragoza 1, Spain). *Rev Esp Oncol* 24(2): 323-348; 1977.

The incidence of cancer was studied in the province of Zaragoza, Spain, during 1970-1974. The incidence per 100, 000 was found to rise progressively from 176.9 during the 1960-1964 to 195 during 1965-1969 and to 237.8 during 1970-1974. The mean annual incidence during 1970-1974 was 259.8 for men and 216.6 for women. The mean annual incidences were 9.58 (17.07 in men and 2.38 in women) for tumors of the buccal cavity and pharynx; 77.39 (84.61 in men and 70.44 in women) for tumors of the digestive and peritoneal organs; 29.67 (51.92 in men and 8.28 in women) for tumors of the respiratory tract; 45.28 (26.33 in men and 63.5 in women) for tumors of the bones, soft tissues, skin, and breast; 42.22 (48.63 in men and 36.05 in women) for tumors of the genitourinary organs; and 30.97 (27.46 in men and 34.34 in women) for tumors of other localizations. Tumors of the stomach were most frequent in men (43.41 in 1965-1969 and 43.73 in 1970-1974), followed by malignant neoplasms of the lungs, bronchus, and trachea (24.68 in 1965-1969 and 34.36 in 1970-1974). Among women, breast cancer was most frequent (46.41), followed by stomach cancer (29.93) during 1970-1974. The incidence of urinary bladder cancer increased considerably in men from 6.68 during 1965-1969 to 15.83 during 1970-1974. (6 refs)

78-4745 **Characteristic Features of Morbidity due to Rectal Carcinoma.** (Rus) Bul'bulian, M. A. (Cancer Res. Center, Moscow, USSR); Knysh, V. I. *Vestn Akad Nauk SSSR* (5): 86-90; 1978.

The incidence, distribution, and etiology of rectal carcinoma are discussed briefly, and the morbidity and mortality indices for this neoplasm in the Soviet Union are presented. The standardized mortality index increased from 2.2 in 1960 to 4.3 in 1972. The highest incidence of rectal carcinoma was observed in individuals > 70 yr-old, and it was 210 and 146 times greater than the respective incidences in men and women < 30-yr-old. (30 refs)

78-4746 **Environment and Cancer.** (Eng) Sahyun, M. R. (St. Paul, MN). *Chem Eng News* 56(12): 43-44; 1978.

The death rate from cancer in the US population for 1974 was estimated using the years 1900-1960 as a baseline for computation. The observed death rate was 13% higher than that expected. This 13% represents fatal neoplasia having a new and presumably environmental etiology. Extrapolation of present trends to the year 1995 indicated a death rate 21% higher than that tabulated from the historical baseline; ie, the excess morbidity due to new etiological factors is 21% of the total. Thus, this 13%-21% range in upper limit values is more representative of actual environmental hazard. (no refs)

78-4747 **Cancer Morbidity in 1975.** (Eng) Rahim, M. A. (Radiotherapy Unit, Medical Coll. Hosp., Dacca, Bangladesh); Choudury, M. H.; Sarma, S. K.; Ahmed, Q. A.; Hossian, M. A. *Bangladesh Cancer Rep* 1(1): 11-17; 1977.

The Bangladesh cancer incidence for 1975 is reported by site. The incidence of cancer of the larynx, lungs, and oropharynx was high in men, and the incidence of cancer of the cervix uteri, breast, and oral cavity was high in women. There was also a high incidence of male breast cancer, with the male:-female ratio being 1:10.8. (10 refs)

78-4748 **Lakes Area Tumor Service Registry: Report 1970-1975.** (Eng) Patterson, J. C. (Lakes Area Tumor Service Registry, Roswell Park Memorial Inst., 666 Elm St., Apartment 3G, Buffalo, NY, 14263). (Buffalo: Roswell Park Memorial Inst.): 85 pp.; 1975.

Survival and deomographic data are presented for 16,463 cases of malignant disease treated at 24 western New York and neighboring Pennsylvania hospitals between January 1970 and December 1975. The data, collected by the Lakes Area Tumor Service Registry, include cancer incidence by site, sex, age distribution, stage of disease at diagnosis, and treatment. (no refs)

78-4749 **Abnormal Cell Proliferation in Human Lithiasic Gallbladder Mucosa (Meeting Abstract).** (Eng) Putz, P. (Gastrointestinal Unit, Vrije Universiteit Brussels, Brussels, Belgium); Willems, G. *Digestion* 16(3): 269; 1977.

78-4750 **The Register on Effects for the Clean Air Program in the Cologne District.** (Ger) Zimmermeyer, G. (Essen, W. Germany). *Staub-Reinhalt Luft* 38(6): 222-226; 1978.

The legal and environmental bases for the second Clean Air Program for the Western Ruhr District are presented, together with the results of epidemiological studies and investigations on plants and various elements. The Clean Air Program contains information on the type and extent of observed and expected air pollutants and their environmental consequences, observations on the causes of air pollution, and recommendations for limiting the air pollution. The program's register on the effects of pollution on humans in the district contains information on carboxyhemoglobin, carcinoembryonic antigen, complement C-3, max lung capacity, blood lead concentrations, sputum immunoglobulin content, and the condition of tonsils and cervical and mandibular lymph nodes. In addition, observations were made on lung cancer, bronchitis, sinusitis, benzpyrene, and phenol. A register of effects on plant material covers the exposure of lichens, standardized grass samples, and various selected crops and feed material to the various air pollutants. There is no conclusive evidence that high air pollution levels in Duisburg have had a detrimental health effect on area residents. There is, however, evidence indicating that the disease incidence in clean air districts is similar to that in the polluted Duisburg area. (2 refs.)

78-4751 **The Benefits of Catastrophe: A Major Reduction in Tumor Prevalence Associated with Greatly Diminished Frog Populations (Meeting Abstract).** (Eng) McKinnell, R. G. (Univ. Minnesota, Saint Paul, MN, 55108); Gorham, E.; Martin, F. B.; Schaad, J. W. *Proc Am Assoc Cancer Res* 19: 212; 1978. (no refs)

78-4752 **Clinical Investigations of the Etiology of Childhood Cancers (Meeting Abstract).** (Eng) Jaffe, N. (Sidney Farber Cancer Inst., Boston, MA, 02115); Marchetto, D. J.; Meadows, A.; Winston, K. R.; Li, F.P.; Scher, C. D. *Proc Am Assoc Cancer Res* 19: 157; 1978. (no refs)

78-4753 **Quantitative Theories of Carcinogenesis.** (Eng) Whittemore, A. (Hunter Coll., City Univ. New York, New York, NY); Keller, J. B. *SIAM Rev* 20(1): 1-30; 1978.

Several mathematical theories of carcinogenesis are presented. These theories involve the transformation of a normal cell and the subsequent proliferation of the transformed cell and its descendants to form a tumor. They predict the rate of tumor occurrence as a function of time and of the concentrations of the agents that cause transformation or accelerate growth. The consequences of these theories are compared with data on human cancer incidence and with laboratory findings. A new theory is presented that includes features of previous theories but that also describes the results of initiation-promotion experiments. The present theories do not distinguish between benign and malignant tumors and they do not include the possibility of cell repair and the action of the immune system. (36 refs.)

78-4754 **Skin Tumours in White South Africans. Part IV. Influence of Occupation, Sun Sensitivity, Colouring and Associated Skin Tumours on the Incidence of Skin Tumours.** (Eng) Whiting, D. A. (Dermatology Section, Dept. Internal Medicine, Southwestern Medical Sch., Univ. Texas Health Science Center, 5323 Harry Hines Boulevard, Dallas, TX, 75235). *S Afr Med J* 53(5): 162-166; 1978.

The effect of occupation, sun sensitivity, coloring, and associated skin tumors on the incidence of skin tumors in white South Africans in the southern Transvaal was investigated. Outdoor and industrial occupations did not increase the incidence of any skin tumor except that of squamous cell carcinoma. However, they did accelerate the onset of solar keratoses and squamous and basal cell carcinomas. Fair and sun-sensitive skin, red hair, and blue eyes predisposed a person to the development of skin cancer. Significant associations included a large number of seborrheic keratoses with polyps, intradermal nevi with cavernous hemangiomas, and intradermal nevi with lentigines. A high proportion of solar keratoses was connected with squamous cell carcinoma, basal cell carcinomas with superficial basal cell carcinoma, and solar keratoses with Bowen's disease. Although 61% of the squamous cell carcinomas were associated with solar keratoses, only 2.5% of the patients with the latter had the former. Likewise, 64% of the patients with Bowen's disease had associated solar keratoses, but only 1% of the patients with the latter had the former. Only 1.6% of the patients with basal cell carcinomas had associated superficial basal cell carcinomas, and only 1/33 patients with Bowen's disease had an associated internal malignancy. (7 refs)

78-4755 **Density-dependent Limitation of Growth and the Regulation of Cell Replication by Changes in the Triggering Level of the Cell Cycle Switch.** (Eng) Gilbert, D. A. (Natl. Inst. Virology, P.O. Bag X4, Sandringham 2131, Transvaal, South Africa). *Biosystems* 9(4): 215-228; 1977.

The effects of altering the sensitivity threshold of a cell to initiation and suppression of proliferation were studied mathematically. Assuming the uptake of regulators by an equilibrium process, the various types of dependency of the max cell density on the initial level of the regulator (eg, serum) in the medium can be explained. Moreover, it may also be possible to distinguish whether an altered threshold or equilibrium constant is responsible for modified proliferation characteristics. The model suggests that some cells are more susceptible to control via changes in the relevant equilibrium constant and that others respond more markedly to threshold modulation. If control occurs through inhibition of replication, it is possible for the system to exhibit a critical mass; if this is exceeded, proliferation could continue indefinitely, provided adequate nutrients are available. An oscillator concept of the cell cycle accounts for the existence of a threshold, and it also permits a general understanding of its modulation by multiple agents. In cancer, a change in the regulator would only give rise to hyperplasia, a process that would continue until some factors became limiting. Competition between normal and cancer cells for the regulator would depend on the relative equilibrium constants: tumor cells would proliferate if the regulator is an inhibitor, but they would cease to replicate if it is a mitogen. Thus, tumors that fail to grow indefinitely would arise in tissue where control is exerted through mitogenic action. (28 refs.)

78-4756 **The Multistage Theory of Carcinogenesis and the Age Distribution of Cancer in Man.** (Eng) Moolgavkar, S. H. (Fox Chase Cancer Center, 7701 Burholme Ave., Philadelphia, PA, 19111). *J Natl Cancer Inst* 61(1): 49-52; 1978.

The multistage theory of carcinogenesis is discussed in relation to the age distribution of cancer in man. The incidence function predicted by the multistage theory of carcinogenesis can be expressed as an infinite (power) series that explicitly exhibits its dependence on transition rates and of which the Armitage-Doll approximation is the first nonzero term. When the transition rates are not small enough (about 10^{-4}/cell/yr), the second nonzero term in the series expansion makes an important contribution to the incidence function. In this case, the Armitage-Doll approximation is a poor one. This is illustrated by a hypothetical example that makes it possible to accomodate, within the multistage theory, the finding that many tumors show deviations below the incidence curve predicted by the Armitage-Doll approximation. Assumptions implicit in the theory have been ignored by some epidemiologists during their attempts to model age-specific incidence curves, and probabilities derived from population data have been confused with probabilities predicted by the model. The multistage theory as originally proposed is time-homogeneous, and no provision is made for a change in transition rates with changes in the environment. Hence, the theory can legitimately be applied only to cohort data for cancers that are not sensitive to environmental changes. (7 refs)

78-4757 **Diseases of Aging Untreated Virgin Female RFM and BALB/c Mice.** (Eng) Cosgrove, G. E. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Satterfield, L. C.; Bowles, N. D.; Klima, W. C. *J Gerontol* 33(2): 178-183; 1978.

Autopsies were performed on 278 RFM/Un and 331 BALB/c germfree-derived, specific-pathogen-free mice used as controls in a radiation experiment, and the incidence of various diseases was calculated. The incidence of neoplastic disease was 88% in the RF mice, 94% in the BALB/c mice. A total of 78% of the RF and 67% of the BALB/c mice developed leukemia or lymphoma. Most of these (67% and 61%, respectively) were reticulum cell sarcomas. However, RF mice had an 8% incidence of thymic lymphoma, compared with 1% in BALB/c mice. Lung tumors were found in 26% of the RF mice and 32% of the BALB/c mice; the most significant difference between the strains occurred with adrenal cortex neoplasms (< 1% for RF and 29% for BALB/c mice). A total of 98% RF mice and 74% BALB/c mice had glomerulosclerosis; high incidences of uterine hyperplasia, adrenal cortical atrophy, and edema were also noted. BALB/c mice tended to live approx 100 days longer than RF mice. The findings for RF mice are compared to literature data from previous experiments with this strain. (20 refs)

See also:
*(Rev.): 78-4206, 78-4213, 78-4214, 78-4215, 78-4216, 78-4217, 78-4218, 78-4219, 78-4221, 78-4222, 78-4223, 78-4225, 78-4226, 78-4227, 78-4232, 78-4234, 78-4246.
*(Chem.): 78-4257, 78-4294, 78-4295, 78-4296, 78-4327, 78-4346.
*(Phys.): 78-4358, 78-4370, 78-4374, 78-4380, 78-4382.
*(Viral): 78-4447, 78-4469, 78-4508.
*(Path.): 78-4643, 78-4651, 78-4654, 78-4661.

78-4758 **Organ Culture of Canine Prostate.** (Eng) Herwig, K. R. (Section Urology, Depts. Surgery, Anatomy, Univ. Michigan Medical Center, Ann Arbor, MI, 48109); Fischer, T. V.; Burkel, W. E.; Kahn, R. H. *Invest Urol* 15(4): 291-294; 1978.

Studies were conducted to develop a reliable culture preparation of dog prostate explants. All explants were interpreted in a double-blind fashion using a specific list of observable variables with defined limits for each. A multiple channel counter tabulated the information on epithelial states, and the data were stored in a computer system for statistical analysis. Explants of canine prostate were minimally maintained when cultivated in Trowell's T-8 medium supplemented with testosterone and insulin. Pretreatment of the dog with methylprednisolone (30 mg/kg im 2 hr before prostate removal) or addition of vitamin A (25 or 50 IU) to the culture medium markedly improved the maintenance of normal structure. Methylprednisolone pretreatment and the addition of vitamin A had a significant effect. Extrapolating any clinical significance of the effect of a glucocorticoid and vitamin A on the intact prostate is still speculative, but the findings suggest that it may be possible to develop a steroid of clinical usefulness for modifying the differentiation of the prostate. (10 refs)

78-4759 **Establishment of Human Myeloma Cells In Vitro (Meeting Abstract).** (Eng) Burk, K. H. (Dept. Lab. Medicine, M. D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); Trujillo, J. M.; Drewinko, B.; Ahearn, M. J. *Proc Am Assoc Cancer Res* 19: 211; 1978. (no refs)

78-4760 **Human Leukemic Cell Growth-Suppressing Factor Detected in Cell Culture (Meeting Abstract).** (Eng) Park, C. H. (Kansas Univ. Medical Center, Kansas City, KS, 66103); Savin, M. A.; Amare, M. *Proc Am Assoc Cancer Res* 19: 97; 1978. (1 ref)

78-4761 **Vitality of Tumor Fragments as a Function of Temperature. Correlation with Electrical Impedance.** (Fre) Santini, R. (Laboratoire de Physiologie pharmacodynamie, I.N.S.A., Batiment 406, 20, avenue Albert-Einstein, 69621 Villeurbanne Cedex, France); Lenoir, J.; Jenin, P.; Thomasset, A. *C R Acad Sci [D] (Paris)* 285(15): 1363-1365; 1977.

The ability of fragments of a Walker 256 rat carcinoma to develop an sc tumor in another rat was determined following heat treatment. The tumorigenicity of the fragments disappeared after they were treated for 150 min at 37 C, 80 min at 41 C, and 10 min at 44 C. Study of the electrical impedance of the tumor fragments showed a good correlation between regeneration after heat treatment and impedance only at 37 C. (9 refs.)

78-4762 **Spontaneous Transformation of Bovine Lens Epithelial Cells. Kinetic Analysis and Differentiation in Monolayers and in Nude Mice.** (Eng) Courtois, Y. (Unite de recherches querontologiques, INSERM U 118, 29, rue Wilhem, F-75016 Paris, France); Simonneau, L.; Tassin, J.; Laurent, M. V.; Malaise, E. *Differentiation* 10(1): 23-30; 1978.

Bovine lens epithelial cells are known to perform two determined functions in vivo. They synthesize the lens capsule and, in the germinal region, they differentiate in fiber cells with massive production of crystallin proteins, inactivation, and pyknosis of the nucleus. Adult bovine lens epithelial cells can be cultured, but no massive crystallin production has been demonstrated in vitro. In long-term cultures, they spontaneously acquired many characteristics of transformation: unlimited growth potential, abnormal karyotype, and multilayering. Viral particles were detected rarely. However, the cells retained their epithelioid character and the ability to synthesize lens capsule material. The kinetic characteristics of the cells were determined. When injected into nude mice, the cells proliferated and formed tumors in which synthesis of α-crystallin was demonstrated. These results show that in vitro transformation of lens epithelial cells does not affect their potential for terminal differentiation. (30 refs)

78-4763 **Androgen-Dependent Human Prostatic Carcinoma Tumor Line in Nude Mice (Meeting Abstract).** (Eng) Reid, L. (Albert Einstein Sch. Medicine, Bronx, NY, 10461); Leav, I.; Merk, F.; Albert, J.; Geller, J. *Proc Am Assoc Cancer Res* 19: 151; 1978. (no refs)

78-4764 **Accumulation of Spermidine and Spermine in Ehrlich Ascites Tumor Cells During Plateau**

Phase Growth. A Function of an Increased Fraction of G2 Phase Cells and Polyploid Cells. (Eng) Andersson, G. (Dept. Zoophysiology, Univ. Lund, Helgonavagen 3, S-223 62 Lund, Sweden); Osterberg, S.; Heby, O. *Int J Biochem* 9(4): 263-267; 1978.

The effect of plateau-phase growth on the accumulation of the polyamines spermidine and spermine in Ehrlich ascites tumor cells was determined. Transplantation of 7-day-old Ehrlich ascites tumor cells into new hosts resulted in a rapid induction of polyamine synthesis. During the first 2 hr, the activities of L-ornithine decarboxylase (ODC; putrescine synthesis) and S-adenosyl-L-methionine decarboxylase (AMD; spermidine and spermine synthesis) increased approx 9.5 and 7 times, respectively. ODC activity rose to a peak value 8 hr after transplantation, but AMD activity dropped between 2 and 6 hr before showing a second peak at 10-12 hr. Subsequently, both enzyme activities decreased continuously with tumor growth, and they exhibited high positive correlations with the rate of cell proliferation. Despite the decrease in polyamine synthesis, there was an accumulation of spermidine and spermine during plateau-phase growth. Further studies showed that the increase in spermidine and spermine was due to an accumulation of cells in the G2 phase of the diploid cell cycle and in the G1 phase of the tetraploid cell cycle. (22 refs)

78-4765 **Hormone-responsive Growth In Vivo of a Tissue Culture Cell Line Established from the MT-W9A Rat Mammary Tumor.** (Eng) Sirbasku, D. A. (Dept. Biochemistry and Molecular Biology, Univ. Texas Medical Sch., Houston, TX, 77025). *Cancer Res* 38(4): 1154-1165; 1978.

The in vivo growth properties of the MTW9/PL cell line, established in culture from a parent MT-W9A hormone-responsive rat mammary tumor, are reported. The MTW9/PL cells are epithelial and grow in culture medium supplemented with a wide variety of sera. When these cells were injected into W/Fu rats, tumor formation was noted in normal females, estrogen-treated males, estrogen-treated ovariectomized females, and estrogen-treated castrated males. Only 0%-30% tumor formation occurred in ovariectomized females or castrated males, and the tumors that formed in gonadectomized hosts were usually smaller than those formed in normal or estrogen-treated hosts. Both progesterone and testosterone were capable of promoting tumor formation in ovariectomized females; hydrocortisone inhibited estrogen-promoted growth. Supraphysiological levels of estrogen inhibited tumor formation in normal intact females. The MTW9/PL cells retained their dual requirement for physiological levels of estrogens and prolactin for optimal tumor formation in W/Fu females as well as a requirement for physiological levels of thyroid hormones. Chromosomal analysis revealed a modal chromosome number of 62. A comparison of the cloned line with the parent line revealed > 11 hormone-related properties shared by the two lines. After 2 yr in culture, the MTW9/PL cells still retained the hormone-responsive growth characteristics of carcinogen-induced rat mammary tumors and normal rat mammary tissue. (35 refs.)

78-4766 **Growth Hormone Modulation of Murine Erythroleukemia Cell Growth In Vitro.** (Eng) Golde, D. W. (Div. Hematology-Oncology, Dept. Medicine, Univ. California Los Angeles Sch. Medicine, Los Angeles, CA, 90024); Bersch, N.; Li, C. H. *Proc Natl Acad Sci USA* 75(7): 3437-3439; 1978.

Friend leukemia virus-infected erythroleukemia cells were found to be responsive to growth hormone (somatotropin) in vitro. In a serum-free clonogenic assay, as little as 0.1 nanogram (ng)/ml of human growth hormone caused a prominent stimulation of cell proliferation. The peak activity of human growth hormone occurred at 200 ng/ml, resulting in a twofold increase in cloning. Human chorionic somatomammotropin and the Cys(Cam)^{53}-hGH(1-134) fragment of human growth hormone were also active, but a biologically inert oxidized human growth hormone had no growth-promoting effect in vitro. In addition, cell proliferation was stimulated by insulin, with peak potentiation occurring at 1 ng/ml, and by prolactin, which had a demonstrable stimulatory effect between 50 and 100 ng/ml. These observations indicate that growth hormone and related polypeptides have a direct effect on the in vitro proliferation of erythroleukemia cells in the absence of serum. The results confirm that there is a direct action of growth hormone on mammalian cells, and they suggest that pituitary hormones may affect leukemic cell growth. (26 refs.)

78-4767 **Warfarin Enantiomers, Anticoagulation, and Experimental Tumour Metastasis (Letter to Editor).** (Eng) Poggi, A. (Lab. Haemostasis and Thrombosis Res., Istituto di Ricerche Farmacologiche "Mario Negri", 20157 Milan, Italy); Mussoni, L.; Kornblihtt, L.; Ballabio, E.; de Gaetano, G.; Donati, M. B. *Lancet* 1(8056): 163-164; 1978.

The effect of sodium warfarin on Lewis lung carcinoma (3LL) induction in mice was investigated. Mice were treated with racemic sodium warfarin (RS) or its R(+) or S(-) enantiomers starting 2 days before im implantation of 3LL. Treatment was continued until the animals were killed. The prothrombin-complex activity was < 5% in animals treated with RS or S(-) warfarin, preparations endowed with anticoagulant activity, but it ranged from 90% to 110% in R(+) warfarin-treated and control mice. RS and S(-) warfarin, but not R(+) warfarin, significantly reduced both primary tumor and spontaneous lung metastases. Iv injection of 3LL cells induced an acute drop in blood platelets and other signs of acute disseminated intravascular coagulation. The drop in platelet count was prevented completely by RS or S(-) warfarin; R(+) warfarin was ineffective. The characteristic effects of these compounds also applied to the development of lung colonies, with tumor formation in R(+) warfarin-treated mice being the same as that in controls. These findings suggest that the antimetastatic effect of oral anticoagulants is not related directly to these compounds but requires impairment of vitamin K availability. (13 refs)

78-4768 **Regional Differences in Tumor Growth (Meeting Abstract).** (Eng) Auerbach, R. (Univ. Wisconsin, Madison, WI, 53706); Morrissey, L. W.; Kubai, L.; Sidky, Y. A. *Proc Am Assoc Cancer Res* 19: 177; 1978. (no refs)

78-4769 **Etiology of Infantile-Type Laryngeal Papillomas.** (Pol) Zakrzewski, A. (Klinika Laryngologiczna, Przybyszewskiego 49, 60-355 Poznan, Poland); Pruszewicz, A.; Kulczynski, B.; Citowicki, W.; Konczewska, H.; Topilko, A.; Sobieszczyk, S. *Otolaryngol Pol* 31(6): 641-646; 1977.

Hormonal, biochemical, and ultrastructural studies were made of 12 children aged 5-16 yr with infantile-type laryngeal papillomas. Serum gonadotropin, growth hormone, and Fe, Cu, Na, K, and Mg levels were measured along with Fe absorption and total binding capacity. The results of the hormonal investigations were within normal limits. However, the finding of a statistically significant decrease in serum Cu and Fe levels and the simultaneous demonstration of adenovirus particles (crystalline forms in the nuclei and B- and C-type particles crossing the cell membrane) are new contributions to the etiology of papillomas. (20 refs.)

78-4770 **Human Brain Tumor Transplantation into Nude Mice (Meeting Abstract).** (Eng) Shapiro, W. (Memorial Sloan-Kettering Cancer Center, New York City, NY, 10021); Basler, G.; Horten, B.; Chernkik, N.; Posner, J. *Proc Am Assoc Cancer Res* 19: 153; 1978. (no refs)

78-4771 **The Role of the Lungs and Liver in the Kinetics of Transplanted Lymphoid Leukemia L1210 Cells Labelled with Radiochromium ^{51}Cr.** (Eng) Skierski, J. (Dept. Biophysics and Biomathematics, Medical Center Postgraduate Education, Warsaw, Poland); Doroszewski, J. *Leuk Res* 1(4): 309-313; 1977.

The fate of a 0.1-ml inoculum of 10^7 ^{51}Cr-labeled L1210 leukemia cells administered either iv or via spleen puncture was investigated in inbred DBA-2/wf and BALB/c x DBA-2/wf/F_1 mice. The mice were decapitated either 5 or 90 min later. Five minutes after iv injection, 15% of the dose was found in the blood, 50% in the lungs, 15% in the liver, 0.3% in the spleen, and 2.15% in the kidneys. By 90 min postinjection, the radioactivity in the blood fell to <4% and that in the lungs to 22%. However, the radioactivity in the liver increased to 30% and that in the spleen to 2.25%; the radioactivity in the kidneys did not change. Five minutes after intraportal injection, approx 2% of the dose was present in the lungs, 65% in the liver, <7% in the blood, and approx 1% in the kidneys. By 90 min, the radioactivity in the liver fell to 35% and that in the lungs to 1%. The radioactivity in the kidneys increased to 3%; no change was noted in the blood. It is suggested that many cells taken up by the liver or passing through it undergo functional changes or are destroyed; cells taken up by the lungs are partially released undamaged and/or in the form of insoluble fragments. (14 refs)

78-4772 **Morphogenesis of Embryonal Gastric Transplants in Syngeneic Recipients of Different Age.** (Rus) Leitina, B. I. (Cancer Res. Center, Moscow, USSR); Aruin, L. I.; Zinzar, S. N.; Svet-Moldavsky, G. Ya.; Turusov, V. S. *Vestn Akad Med Nauk SSSR* (2): 50-59; 1978.

The effect of recipient age on the differentiation of transplanted stomach tissue was studied in BALB/c, DBA/2, and CBA mice. Two series of experiments were carried out. In the first series, minced stomach tissue from 18- to 20-day embryos was transplanted sc into syngeneic mature mice. The mice were sacrificed at varying times between 2-30 days posttransplantation. In the second series, a segment of the stomach fundus from 18- to 20-day embryos was transplanted sc into mature mice, 11- to 12-day old mice, 5- to 6-day-old mice and newborn (1-day-old) mice. The mice were sacrificed on day 50 after transplantation. In the first series, the transplanted stomach tissue showed progressive differentiation. In the second series, two types of cysts (with nodes 2-5 mm in diameter) were detected in the transplanted stomach segment. The newborn mice had only the undifferentiated type, but the rest of the mice had both undifferentiated and differentiated cysts. (14 refs)

78-4773 **Molecular Biological Aspects of the Problem of Human Genetics.** (Jpn) Sekiguchi, M. (Dept. Biology, Faculty Science, Kyushu Univ., Fukuoka, Japan). *Jpn J Hum Genet* 22(2/3): 137-143; 1977.

The use of phage T4 repair endonuclease on xeroderma pigmentosum (XP) cells with the aid of Sendai virus resulted in restoration of the repair capabilities of Groups A to E XP cells. This suggests that the incision step of excision repair in human cells is controlled by at least five genes, but only one gene is involved in the T4 system. (3 refs)

78-4774 **The Relationship Between Neoplastic Transformation and the Cellular Genetic Apparatus (Meeting Abstract).** (Eng) Ts'o, P. O. (Div. Biophysics, Sch. Hygiene and Public Health, John Hopkins Univ., Baltimore, MD, 21205). *In Vitro* 14(4): 385; 1978. (no refs)

78-4775 **Transcriptional Control of α2u-Globulin Gene in Hepatoma (Meeting Abstract).** (Eng) Chan, K. M. (Inst. Cancer Res. and Dept. Biochemistry, Columbia Univ., New York, NY, 10032); Kurtz, D. T.; Feigelson, P. *Proc Am Assoc Cancer Res* 19: 24; 1978. (no refs)

78-4776 **Alterations in the Complexity of Nonreiterated Sequence Transcripts in the Rat Liver During Regeneration (Meeting Abstract).** (Eng) Garrett, C. T. (George Washington Univ. Medical Center, Washington, DC, 20037); Gonzalez, F. J.; Wiener, D.; Genta, V. M. *Proc Am Assoc Cancer Res* 19: 85; 1978. (no refs)

78-4777 **Histone Proteases and the Distribution of Retained Histones in Nuclei from Rat Ventral Prostate Cells Undergoing Hypertrophy or Hyperplasia.** (Eng) Kadohama, N. (Dept. Medicine, Div. C, Roswell Park Memorial Inst., Buffalo, NY); Shirley, M. A.; Anderson, K. M. *Int J Biochem* 8(8): 601-609; 1977.

The distribution of histones and histone proteases was studied in ventral prostate (VP) nuclei from normal rats, short-term (3-day, designated C_3), or long-term castrates (7-day, designated C_7), or castrates treated with testosterone propionate for 2 or 3 days (designated T_2 and T_3, respectively). C_3T_3 rats showed cellular hypertrophy (growth), C_7 marked prostate involution and cell death, and C_7T_2 cellular replication (hyperplasia). The distribution of histones in VP nuclei from normal rats, C_3, C_7, and C_3T_3 did not differ. VP nuclei from C_7T_2 contained less histone F2a2 than those of controls. The distribution of partially degraded histones in nuclear extracts from growing or dividing VP cells differed from each other and from their castrated controls, suggesting a differing distribution of histone proteases and/or modifications of F2a2. A functional role for histone proteases exhibiting apparent cell-cycle substrate specificity and/or modification of F2a2 was suggested. Limited selective proteolysis of histones in nuclei from regressing, hypertrophic, or hyperplastic prostate cells may be necessary to prepare chromatin for the subsequent molecular events (eg replication and reprogramming) required for these processes. (51 refs.)

78-4778 **Increased Frequency of Pi Z Phenotypes of Alpha-1-Protease Inhibitor (Alpha-1-Antitrypsin) in Patients with Lung Cancer (Meeting Abstract).** (Eng) Arnaud, P. (Dept. Immunology and Microbiology, Medical Univ. South Carolina, Charleston, SC); Chapuis-Cellier, C.; Bata, J.; Fudenberg, H. H. *Clin Res* 26(3): 431A; 1978. (no refs)

78-4779 **Colchicine and Cytochalasin B (CB) Effects on Random Movement, Spreading and Adhesion of Mouse Macrophages.** (Eng) Cheung, H. T. (Immunobiology Res. Center, Univ. Wisconsin, Madison, WI, 53706); Cantarow, W. D.; Sundharadas, G. *Exp Cell Res* 111(1): 95-103; 1978.

The role of microtubules and microfilaments in the control of random movement, spreading, and adhesion of peritoneal macrophages was investigated in C57BL/6 mouse peritoneal exudate cells (PEC) treated with colchicine and cytochalasin B (CB). Movement of complete Freund's adjuvant-induced PEC was substantially enhanced at colchicine concentrations $> 10^{-8}$ M. Max movement was observed in the concentration range 10^{-5}-10^{-4} M. The affected cells were mostly macrophages; it is not known why polymorphonuclear WBC did not also migrate. An increase in random movement of macrophages was also obtained with thioglycollate-induced PEC. Colchicine inhibited cell spreading down to a concentration of 10^{-10} M. At concentrations $> 10^{-8}$ M, there was a significant inhibition of adhesion that increased with increasing colchicine concentrations. CB inhibited the movement of peritoneal macrophages significantly at concentrations 10^{-7} M. At concentrations $> 10^{-6}$ M, CB caused a dramatic inhibition of spreading. There was a greater enhancement of migration in the presence of 10^{-8} M CB and $> 10^{-8}$ M colchicine than in the presence of colchicine alone. Furthermore, in the presence of 10^{-8} M CB, colchicine enhanced movement at concentrations as low as 10^{-12} M. These findings suggest a cooperative interaction between cytoplasmic microfilaments and microtubules in controlling cell movement, adhesion, and spreading. (23 refs)

78-4780 **Models for the Specific Adhesion of Cells to Cells.** (Eng) Bell, G. I. (Theoretical Biology and Biophysics Group, Theoretical Div., Univ. California's Los Alamos Scientific Lab., Los Alamos, NM, 87545). *Science* 200(4342): 618-627; 1978.

A theoretical framework for quantitatively assessing the role that bonds between cells or between cells and surfaces play in adhesion is proposed. The framework is applicable to adhesion mediated by reversible bonds between specific molecules such as antigen and antibody, lectin and carbohydrate, or enzyme and substrate. Based on a knowledge of the reaction rates for reactants in soln and of their diffusion constants both in soln and on membranes, it is possible to estimate reaction rates for membrane-bound reactants. Two models are developed for predicting the rate of bond formation between cells, and the predictions are compared with experimental findings. The force required to separate two cells is shown to be greater than the expected electrical forces between cells and of the same order of magnitude as the forces required to pull gangliosides out of the cell membrane. (71 refs)

78-4781 **Phosphate Metabolites in Lymphoid, Friend Erythroleukemia, and HeLa Cells Observed by High-Resolution ^{31}P Nuclear Magnetic Resonance.** (Eng) Navon, G. (Dept. Chemistry, Tel-Aviv Univ., Ramat-Aviv, Tel-Aviv, Israel); Navon, R.; Shulman, R. G.; Yamane, T. *Proc Natl Acad Sci USA* 75(2): 891-895; 1978.

High-resolution phosphorus-31 (^{31}P) nuclear magnetic resonance (NMR) spectra were observed for lymphoid, Friend erythroleukemia, and HeLa cells. Some phosphate metabolites [glycero(3)phosphocholine, phosphoethanolamine, phosphocholine, glycero(3)phosphoethanolamine, and creatine phosphate] were identified, and their concentrations were found to vary among the different cell lines. ATP/ADP ratios showed large variations among the cell lines. Although this ratio approached 1 in Friend and HeLa cells, high ATP concentrations were maintained in the lymphoid line cells. Phosphorylcholine and phosphorylethanolamine and their glyceryl esters were present in relatively high concentrations in the cells, but their ratios varied considerably. A high concentration of an unidentified diphosphodiester compound was observed in the Friend erythroleukemia and HeLa cells. No appreciable pH gradient across the cell membrane was observed in any of the cell lines, in contrast to the large pH gradient detected in microorganisms. It is concluded that the ^{31}P NMR spectra will make it possible to detect unexpected differences in the level of metabolites among cells from different sources as well as such differences between normal and transformed cells. (14 refs)

78-4782 **Lipid-Chemical Features of Human Gastric Cancerous, Polypous and Ulcerative Tissues.** (Eng) Nakazawa, I. (Third Dept. Internal Medicine, Tohoku Univ. Sch. Medicine, Sendai 980, Japan); Asaki, S.; Ito, S. *Tohoku J Exp Med* 123(4): 365-370; 1977.

The lipid levels of 14 cases of gastric cancer, 9 cases of gastric polyp, and 4 cases of gastric ulcer were studied to examine the biochemical differences between malignant and benign gastric tissue. Specimens of each lesion and the surrounding normal tissue were collected surgically. Part of each specimen was examined histologically, and the other part was subjected to lipid analyses by thin-layer and gas-liquid chromatography. The total lipid content was decreased in 71% of the gastric cancer cases and in 75% of the polyp and ulcer cases. The phospholipid fatty acid content was 83% lower in the cancerous lesions and 50% lower in the polypous lesions than in the surrounding normal tissue. Comparison of the phospholipid fatty acid composition in cancerous and polypous tissues indicated that the percentage of $C_{18:2}$ was decreased and that of $C_{20:4}$ was increased in the former as compared to the latter. The increase of $C_{20:4}$ in cancerous tissues could result in an increased fluidity of the cell membrane, and it might constitute at least one of the mechanisms involved in the malignant growth of cancerous gastric tissues. (10 refs)

78-4783 **Lipid Growth Factors in Serum (Meeting Abstract).** (Eng) Kaplan, A. E. (Carcinogen Metabolism and Toxicology Branch, NCI, Bethesda, MD, 20014); Bartholomew, J. C. *Proc Am Assoc Cancer Res* 19: 201; 1978. (no refs)

78-4784 **Membrane Lipid Dynamics and Density Dependent Growth Control in Normal and Transformed Avian Cells.** (Eng) Nicolau, C. (Institut fur Strahlenchemie im Max-Planck-Institut fur Kohlenforschung, D4330 Mulheim a. d. Ruhr, W. Germany); Hildenbrand, K.; Reimann, A.; Johnson, S. M.; Vaheri, A.; Friis, R. R. *Exp Cell Res* 113(1): 63-73; 1978.

The membrane fluidity of normal chick embryo fibroblasts and normal Japanese quail fibroblasts and their Rous sarcoma virus (RSV)- and methylcholanthrene (MC) transformed counterparts was investigated by fluorescence polarization. Normal cells and their isolated plasma membranes showed significant changes in fluidity as a function of population density, but neither RSV- nor MC-transformed cells or their isolated plasma membranes showed this effect. Stimulation of growth by addition of calf serum to cultures of quiescent, density-inhibited normal cells was accompanied by increased membrane lipid fluidity that occurred <2 hr after stimulation. Neither sparse normal cells nor sparse or dense transformed cells showed any significant fluidity change in their membrane lipids following addition of serum. Enzyme and electron microscopic analysis of the ratios of different membrane types (plasma membrane:intracellular membrane) in each cell type showed that this ratio did not vary with respect to cell population density but that it was different between transformed and normal cells. It is concluded that the observed fluidity changes reflect organizational differences occurring as a function of population density in cell cultures that retain density-dependent growth control. (31 refs)

78-4785 **Role of Polyamines in the Regulation of Nuclear Division in Normal and Transformed Mammalian Cells (Meeting Abstract).** (Eng) Sunkara, P. S. (Dept. Developmental Therapeutics, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX, 77030); Seman, G.; Rao, P. N. *Proc Am Assoc Cancer Res* 19: 16; 1978. (no refs)

78-4786 **Transglutaminase and Isopeptides in Normal and Transformed Cells (Meeting Abstract).** (Eng) Birckbichler, P. J. (Biomedical Div., Samuel Roberts Noble Foundation, Ardmore, OK, 73401); Orr, G. R.; Carter, H. A.; Conway, E.; Maxwell, M. D.; Patterson, M. K. *Proc Am Assoc Cancer Res* 19: 67; 1978. (no refs)

78-4787 **γ-Glutamyltranspeptidase Activity in Normal, Regenerating and Malignant Hepatocytes.** (Eng) Cheng, S. (Dept. Biochemistry, Univ. Southern California Sch. Medicine, Los Angeles, CA, 90033); Nassar, K.; Levy, D. *FEBS Lett* 85(2): 310-312; 1978.

To define factors affecting the activity of γ-glutamyltranspeptidase (GTP), the levels of this enzyme were investigated in quiescent hepatocytes, regenerating liver, Morris hepatoma 7777, hepatocytes in monolayer culture, and hepatoma tissue culture (HTC) cells in monolayer and spinner culture. Isolated normal hepatocytes from male Sprague-Dawley rats were found to possess very low enzyme activity. A comparison of the activity observed in whole cells with that in the corresponding cell homogenate indicated that 42% of the enzyme was associated with the cell surface. HTC cells exhibited a 150-fold increase in enzyme activity, with 52% being associated with the cell surface. Morris hepatoma 7777 also showed a large increase in enzyme activity. Normal (RL-34) and 4-nitroquinoline 1-oxide-treated (RL-34-HII) cells were not and only slightly tumorigenic, respectively. These cells exhibited a six- to sevenfold increase in enzyme activity and a cellular distribution, indicating a 70% and 50% surface association, respectively. The spontaneously transformed cell line, RLC C-1, expressed highly elevated levels, comparable to those of the HTC cell system, with 70% of the activity being associated with the cell surface. These results suggest that increased GTP activity correlates with the degree of tumorigenicity expressed by the cell line or tissue. Regenerating liver expressed low activity levels. A 2.5-fold increase in activity was observed after 12 hr, but it returned rapidly to a slightly elevated plateau. The comparatively low GTP activity in the regenerating liver suggests that the marked increases in its activity are characteristic of hepatocyte transformation and not of rapid cell proliferation. (17 refs)

78-4788 **Distribution of Glycoconjugates in Mouse Fibroblasts with Varying Degrees of Tumorigenicity.** (Eng) Winterbourne, D. J. (Macromolecular Biology Section, NCI, NIH, Bethesda, MD, 20014); Mora, P. T. *J Supramol Struct* 7(1): 91-100; 1977.

Glucosamine labeled glycoconjugates were compared in two clones and the parent embryonic mouse cell line. The two clones were at least 100-fold less tumorigenic than the parent cell line. Hyaluronic acid, heparan sulfate, and chondroitin sulfate as well as a complex mixture of glycopeptides were found in the medium, the trypsinate, and the trypsinized cells. The three cell lines had very similar in vitro growth properties, including their plating efficiency in viscous medium, and similar distributions of glycoconjugates throughout the culture. The only difference that correlated with higher tumorigenicity was a slightly higher labeling of hyaluronic acid relative to the other glycoconjugates in the parent cell line. The mechanism by which increased secretion of hyaluronic acid could benefit tumor growth is not clear, but it is possible that it could mask antigens, provide an appropriate extracellular matrix in which the cells could proliferate, or block lymphocyte proliferation. (38 refs)

78-4789 **Characterization of the L-Arabinose Sensitive Strain SV3 of *Salmonella typhimurium* and of L-Arabinose Resistant Mutants Selected in this Strain (Meeting Abstract).** (Eng) Pueyo, C. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC); Lopez-Barea, J. *Genetics* 88(4, part 2, Suppl): s78-s79; 1978. (no refs)

78-4790 **Serum Antigens and Antibodies in the Diagnosis of Thyroid Cancer.** (Eng) DeGroot, L. J. (Thyroid Study Unit, Dept. Medicine, Univ. Chicago, Chicago, IL); Hoye, K.; Refetoff, S.; Van Herle, A. J.; Asteris, G. T.; Rochman, H. *J Clin Endocrinol Metab* 45(6): 1220-1223; 1977.

The sera of 80 patients with thyroid cancer and 275 patients with a history of radiation exposure were analyzed for carcinoembryonic antigen (CEA) and thyroglobulin (TG) by radioimmunoassay and for antithyroglobulin (TGHA) and antimicrosomal antibodies (MCHA) by RBC hemagglutination. Control values were obtained from a population of blood bank volunteers with an approx equal distribution of sex and age (19-35 yr). In the 275 irradiated patients, the number of positive results for each test was increased above that of controls, with the increase for TGHA and TG being significant ($p < 0.05$). However, patients with benign or malignant thyroid lesions in the irradiated group could not be screened on the basis of these test results. Thyroid cancer patients that lacked a history of x-ray exposure had more frequent positive responses for TGHA, MCHA, and CEA than cancer patients with such history. Among all tests and categories of patients, only the TG assay was distinctive in its association with thyroid cancer. Elevations of TG $>$ 300 nanograms/ml were found only in 4/4 Stage IV thyroid cancer patients, but in these patients the diagnosis was usually clinically obvious. In addition, 24/117 patients without cancer also had positive TG levels and, overall, 72% of the cancer patients had negative levels. (19 refs.)

78-4791 **Intracellular Magnesium Concentrations Found in Normal and Leukemic Human Leukocytes (Meeting Abstract).** (Eng) Brown, E. R. (Univ. Chicago Health Sciences, Chicago Medical Sch., Chicago, IL, 60612); Hazdra, J. J.; Callaghan, O. H. *Proc Am Assoc Cancer Res* 19: 172; 1978. (2 refs)

78-4792 **A Novel Quantitative Separation of Cr(III) from Numerous Metal Ions on β-Stannic Arsenate Thin Layers in DMSO-HCl Systems.** (Eng) Qureshi, M. (Chemistry Section, Z. H. Coll. Engineering and Technology, Aligarh Muslim Univ., Aligarh, India); Varshney, K. G.; Fatima, N. *Sep Sci Technol* 13(4): 321-333; 1978.

The use of β-stannic arsenate thin-layer chromatography and a dimethyl sulfoxide (DMSO) solvent system for the separation of Cr(III) from metal ions is described. The method is precise and accurate, and it can be applied for the quantitative separation of Cr(III) from mixtures containing Cu, Nb, Ta, Ce, Ni, Co, Al, Zn, Mn, Th, La, Cd, and Pt. The selective separation is a consequence of the kinetic inertness of Cr(III), the unique properties of DMSO, and the selectivity of the β-stannic arsenate layers. (19 refs)

78-4793 **Globin Messenger RNA Content in Hepatomas: A Test of Retrogenesis.** (Eng) Longenecker, J. P. (Dept. Biochemistry, Sch. General Studies, Australian Natl. Univ., P.O. Box 4, Canberra ACT 2600, Australia); Williams, J. F. *Cancer Lett* 4(1): 45-49; 1978.

The globin messenger RNA (mRNA) content of fetal, neonatal, and normal adult liver, of the transplantable Morris hepatoma 5123C, and of the host liver of animals carrying the tumor was measured in an attempt to identify an early developmental gene product under pretranslational control that could be used in clinical diagnosis. Hybridization experiments indicated that 90% of the fetal globin mRNA was lost 2 days after birth. Tumor and host liver extracts contained 4.13 and 2.38 times as much globin mRNA, respectively, as extracts of normal adult liver; however, the max hybridization was only 35% in all three tissues. This indicates that the reactive mRNA species from normal, host, and tumor extracts were the same and that they differed from the fetal and neonatal mRNA species. These results show that the gene for fetal globin mRNA is not reexpressed in the hepatoma and that it is not induced in the host liver. (15 refs)

78-4794 **Effect of Patulin on In Vitro Syntheses of RNA and Proteins.** (Fre) Hatey, F. (Institut de Recherches Scientifiques sur le Cancer, 94800 Villejuif, France); Moule, Y. *Ann Nutr Aliment* 31(4/5/6): 867-870; 1977.

The effects of patulin, a mycotoxin isolated from *Byssochlamis nivea*, on RNA transcription and translation was studied in vitro. Patulin inhibited RNA synthesis in isolated rat liver nuclei by 50-100% at a concentration of about 200 μg/ml. In transcriptional studies using RNA polymerases isolated from *Escherichia coli*, the mycotoxin apparently acted on RNA polymerase, and blocking occurred at the start of transcription. (11 refs)

78-4795 **Biochemical Analysis of Damage Induced in Yeast by Formaldehyde. II. Induction of Cross-Links Between DNA and Protein.** (Eng) Magana-Schwencke, N. (Fondation Curie-Institut du Radium, Biologie, 15, rue Georges Clemenceau, Batiment 110, 91405 - Orsay, France); Ekert, B. *Mutat Res* 51(1): 11-19; 1978.

The nature of the molecular damage caused by formaldehyde (FA) in the eucaryotic organism *Saccharomyces cerevisiae* was studied. A haploid strain of *S. cerevisiae*, N123 (a his$_1$), was used. ^{3}H-labeled cells were treated with 66 mM FA for 30 min in phosphate buffer, filtered, washed, and applied to neutral sucrose gradients. In the FA-treated cells, the DNA was strongly linked to a fast-sedimenting structure. Protoplasts were then incubated with 16.5-298 mM FA. A dose-effect relation was found for the induction of DNA-protein cross-links after FA treatment; no evidence for a threshold dose of FA was found. DNA extractability was measured after UV irradiation of protoplasts, and a dose-effect relation was found for the induction of cross-links. The equilibrium density-gradient centrifugation profile of DNA from FA-treated cells showed that a main nuclear peak at a density of 1,700 g/cm^3, which is found with untreated cells, had been displaced toward the lighter region of the gradient. This was due to the binding of DNA with a protein-containing material. (25 refs.)

78-4796 **Reverse Transcription of tRNA.** (Eng) Wittig, B. (Institut fur Molekularbiologie und Biochemie, Freie Universitat Berlin, Arnimallee 22, D-1000 Berlin

33, W. Germany); Wittig, S. *Nucleic Acids Res* 5(4): 1165-1178; 1978.

The 3′ terminus of transfer RNA (tRNA) was elongated enzymatically by an oligo(A) tail. A fragment of DNA polymerase I *Escherichia coli* was used in the presence of manganese to phase and synthesize a cleavable primer at the oligo-(A)-tRNA template. When the three-dimensional structure of oligo(A)-tRNA was unfolded under conditions in which the primer still hybridized at the oligo(A) tail, the DNA polymerase I fragment transcribed oligo(A)-tRNA into DNA. Reverse transcription was slowed and its fidelity was suspended by the 1-methyladenine in oligo(A)-tRNA-Phe (yeast). The reaction was stopped by the highly modified Y base present in this template. Approx full-length transcripts can be obtained from oligo(A)-tRNA$_3$-Gly (*E. coli*). The transcription products were characterized by sequence analysis. (28 refs)

78-4797 **Human Uracil DNA N-Glycosidase: Studies in Normal and Repair Defective Cultured Fibroblasts.** (Eng) Kuhnlein, U. (Dept. Biochemistry, Univ. California, Berkeley, CA, 94720); Lee, B.; Linn, S. *Nucleic Acids Res* 5(1): 117-125; 1978.

Uracil DNA N-glycosidase activity was analyzed in extracts of human fibroblasts from normal subjects, those with xeroderma pigmentosum (XP), and a subject with ataxia telangiectasia (AT). This enzyme is involved in the excision of uracil from DNA. Cell lines representative of complementation Groups A and D of XP and AT had almost the same level of activity as the normal cells. In contrast, cells from two XP variants (XP4BE and XP13BE) had roughly half the normal level of activity, and cells from the heterozygous mother of XP4BE had an intermediate level of activity. In spite of these quantitative differences, there were no systematic alterations in reaction characteristics, apparent Km for the substrate, or purification characteristics for the enzyme for any of the lines. A causal relationship, if any, between uracil DNA N-glycosidase levels and repair deficiency diseases is concluded to be equivocal. (25 refs)

78-4798 **Extracellular DNA Polymerase from Breast Cyst Fluids and Derived Cell Cultures (Meeting Abstract).** (Eng) Witkin, S. S. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Borenfreund, E.; Bendich, A.; Fleisher, M.; Schwartz, M. K. *Proc Am Assoc Cancer Res* 19: 175; 1978. (no refs)

78-4799 **Invasive Locomotory Behavior Between Malignant Human Melanoma Cells and Normal Fibroblasts Filmed in Vitro.** (Eng) Stephenson, E. M. (Zoology Building, A08, Univ. Sydney, Sydney 2006, Australia); Stephenson, N. G. *J Cell Sci* 32: 389-418; 1978.

The relative invasive capacities of human malignant melanoma (MM96) cells and normal adult human skin fibroblasts (HSF) were compared with those of a standard population of chick heart fibroblasts (CHF). The different cell types were confronted in pairs and filmed as they moved outward and eventually met in a situation in which each could potentially invade the other. Replicate cultures were fixed and stained 24 to 40 hr after junction. MM96 cells invaded the CHF population but eventually encountered moderate obstruction. Invasion was mainly by oriented movement between two successive pairs of chick cells. Contact paralysis of ruffling of MM96 was not seen. HSF did not invade the CHF; they were contact-inhibited by the latter and typically reversed their direction of movement. MM96 cells invaded the HSF without obstruction, using several different pathways for locomotory invasion, including overlapping. CHF and HSF were contact-inhibited by leading-edge collisions with MM96 cells. Fibroblast invasion of MM96 was by underlapping of orthogonally oriented, bipolar melanocytes or dendritic processes. HSF were less obstructed than CHF during invasion of MM96. Initiation of invasion of HSF by CHF was apparently orientation-dependent and was assisted by the alignment, morphology, and contact-mediated withdrawal of the HSF. Heterologous contact inhibition did not appear defective for either cell type. In all confrontations involving both malignant and nonmalignant cells, the extent and pattern of invasion could be related to apparent deficiencies in heterologous contact inhibition of locomotion. (21 refs.)

78-4800 **Personality Structures of Breast Cancer Patients (IV). Depression and Cancer: A Comparison.** (Spa) Lerer, S. B. (Departmento de Psiquiatria, Facultad de Medicina, Univ. Javeriana, Bogota, Colombia); Silva, J. L.; Garcia, D.; Gomez, R.; Mendez, J. L. *Rev Colomb Psiquiatria* 6(3): 255-273; 1977.

Psychological studies were performed on 20 women with adenocarcinoma of the breast and 20 women with neurotic depression and hyperchondria. Compared with the neurotic depressives, the cancer patients were significantly more repressed and less assertive. They also had less capacity for play, socializatioin, and interpersonal relationships. Although personal losses triggered psychosomatic symptoms in both groups, the cancer patients derived less secondary gain from their symptoms. The possibility that a submissive attitude can adversely effect a cancer patient's prognosis is discussed. (21 refs)

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